An Investigation of Social and Pharmacological Exposure to Secondhand Tobacco Smoke as Possible Predictors of Perceived Nicotine Dependence, Smoking Susceptibility, and Smoking Expectancies Among Never-Smoking Youth

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Abstract

Introduction—Recent studies evidenced that adolescent never-smokers exposed to secondhand tobacco smoke (SHS) endorsed nicotine dependence symptoms. Other studies showed that SHS exposure measured with biomarkers among never-smokers independently predicted withdrawal sensations and prospective smoking initiation. The aim of the present study was to replicate and extend these findings by investigating whether social and pharmacological measures of SHS exposure predicted precursors to smoking among never-smoking adolescents.

Methods—Participants included 327 never-smokers aged 11–15 years attending sixth or seventh grade in French language schools in Montréal, Canada. They completed self-report questionnaires measuring their smoking status, social smoke exposure (number of smokers in their environment and number of situations where SHS exposure occurs), and precursors to smoking initiation (smoking expectancies, perceived nicotine dependence, and smoking susceptibility). Each participant provided a saliva sample from which cotinine biomarkers were derived to measure pharmacological exposure to SHS.

Results—When predictors were modeled individually, number of smokers predicted perceived nicotine dependence ($p = .05$), smoking susceptibility ($p = .001$), and expected benefits ($p = .05$), whereas number of situations predicted smoking susceptibility ($p = .01$). When predictors were

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Declaration of Interests

None declared.

modeled simultaneously, number of smokers predicted perceived nicotine dependence ($p = .01$), smoking susceptibility ($p = .01$), and expected benefits ($p = .05$).

Conclusions—Social smoke exposure was a predictor for smoking precursors. Pharmacological exposure to SHS did not predict smoking precursors, which may be partly attributable to the low cotinine values observed in our sample. Suggestions for improved pharmacological measurement of SHS and implications for public health are discussed.

Introduction

Tobacco smoking usually emerges during adolescence and remains the most preventable cause of disease, disability, and death (Chassin, Presson, Pitts, and Sherman, 2000; U.S. Department of Health and Human Services [USDHHS], 2004). Of increasing importance is the percentage of youth exposed to secondhand tobacco smoke (SHS). North American estimates show that 60% of children are exposed to SHS, with 25% of children exposed at home (USDHHS, 2006). In youth, SHS has been linked to serious diseases (USDHHS, 2006), but little is known about the possible behavioral repercussions of SHS exposure.

SHS Exposure and Smoking Behavior

Becklake, Ghezzo, and Ernst (2005) reported that SHS exposure in nonsmoking children, measured with salivary cotinine, independently predicted smoking initiation 4 years later. This longitudinal study was the first to demonstrate that objectively measured SHS in nonsmoking children is a risk factor for smoking initiation during adolescence, after controlling for the number of smokers in the household. SHS exposure has been associated with never-smokers endorsing nicotine dependence symptoms. Okoli, Rayens, and Hahn (2007) examined the effects of SHS on hospitality employees. Nonsmokers with high levels of hair nicotine were more likely to rate *DSM-IV* nicotine withdrawal symptoms than nonsmokers with low levels of hair nicotine. Similarly, Bélanger et al. (2008) reported that 5% of never-smoking fifth graders endorsed at least one symptom of nicotine dependence measured in self-reports. Symptoms were more likely to be endorsed with greater exposure to SHS in a car, even after controlling for sibling and peer smoking, and smoking susceptibility. Most recently, experimental data showed that pharmacological exposure to nicotine in SHS contributed to nicotine dependence in laboratory rats (Small et al., 2010).

Overall, these studies suggest that SHS exposure may be a risk factor for smoking behavior, whereby exposure to nicotine in SHS could have independent effects from social modeling on smoking initiation. However, no study evaluated the differential behavioral effects of social modeling and nicotine exposure among never-smoking adolescents.

Nicotine Exposure from SHS

Nonsmokers exposed to high levels of SHS may absorb amounts of nicotine similar to smokers. Al-Delaimy, Fraser, and Woodward (2001) demonstrated that the hair nicotine levels of nonsmokers working in venues where smoking was permitted had hair nicotine levels comparable to those of smokers (6.7 vs. 7.9 ng/mg). Similar results showed that nonsmokers highly exposed to SHS at work had hair nicotine values that resembled those of smokers (1.0 vs. 1.2 ng/mg; Dimich-Ward, Gee, Brauer, and Leung, 1997). Moreover,

nicotine absorbed from SHS can be in sufficient quantities known to have psychoactive effects in smokers (Pacifici et al., 1995; Perkins, Fonte, Sanders, Meeker, and Wilson, 2001). In fact, the blood levels of nicotine from SHS in nonsmokers in another study (Pacifici et al.) were similar to the levels that function as discriminative cues in smokers in another study (Perkins et al.). Given that nicotine is a psychoactive substance associated with addiction among smokers (USDHHS, 1988) and sensitization in animals (Vezina, McGehee, and Green, 2007), these findings raise the question of whether pharmacological exposure to nicotine in nonsmokers contributes to neuroadaptations that could alter subsequent responses to nicotine.

Children are particularly vulnerable to the effects of nicotine exposure through SHS, partly because the pharmacokinetics of nicotine differs in children and adults. In an experimental study, nonsmoking children and adults were exposed to controlled levels of SHS on an enclosed bus (Willers, Skarping, Dalene, and Skerfving, 1995). Compared with adults, children had higher peak levels of urinary cotinine (22 vs. 11 mg/L) and higher doses of nicotine (2.3 vs. 1.7 mg/kg) during and shortly after exposure. The nicotine dose among children was also higher throughout the 7 days postexposure (27% vs. 16%). Children exposed to the same amounts of SHS as adults absorb higher doses of nicotine, suggesting that SHS exposure could possibly lead to greater neural effects in children than adults.

Social Exposure to Smoking

Social smoke exposure is a known risk factor for smoking initiation and refers to observing smokers in one's social environment. Longitudinal data have shown that smoking by parents, siblings, friends, and school personnel are risk factors for adolescent smoking initiation (O'Loughlin, Karp, Koulis, Paradis, and DiFranza, 2009). The effects attributed to social smoke exposure may be the result of role modeling and possibly nicotine exposure through SHS. However, few have considered that greater exposure to smokers is largely associated with greater exposure to nicotine. It is thus important to measure both social and pharmacological exposure to SHS to consider the unique contribution of each exposure type on smoking initiation.

The Present Study

The present study evaluated the effects of SHS exposure on smoking precursors (i.e., perceived nicotine dependence, smoking susceptibility, and smoking expectancies) among never-smoking youth. Specifically, we sought to replicate the findings of Bélanger et al. (2008) whereby SHS exposure contributed to self-reports of nicotine dependence among childhood never-smokers. Next, we sought to extend these findings by evaluating the effects of the number of situations of SHS exposure on smoking precursors. Then, we evaluated the effects of exposure to nicotine from SHS on smoking precursors with the use of biochemical measures, which were not included in the study by Bélanger et al.

It was first hypothesized that a greater number of smokers would predict smoking precursors. Second, it was hypothesized that a greater number of situations of SHS exposure would predict smoking precursors. Third, it was hypothesized that cotinine would predict

smoking precursors. Fourth, it was hypothesized that number of smokers, number of situations, and cotinine would independently predict smoking precursors.

Methods

Participants

A total of 377 students (54.3% females; $M_{\text{age}} = 12.7$, $SD = 0.7$) attending sixth or seventh grade (86.2% in seventh grade) participated in the study. This study was conducted in five French language public schools in the greater Montréal area. In Canada, French-speaking youth are more likely to be exposed to smokers at home than English-speaking youth (15% vs. 8%; Health Canada, 2008). Students in sixth and seventh grades were selected purposefully because there is a higher probability they never tried smoking compared with older students (Health Canada, 2007). The present study has been approved by the ethical review committee of Concordia University (UH2006-063-2).

Procedure

School principals and teachers from targeted school boards were contacted to obtain permission to collect data in their classrooms during class time. Consent forms were sent home to parents with the schoolchildren. On the consent form, parents were told that the primary objective of the study was to evaluate the behavioral effects of SHS exposure. They were also told that their child would answer questions about smoking and provide a saliva sample. It was emphasized that the saliva samples would be destroyed upon completion of the cotinine assays. Sixty-three percent of schoolchildren ($n = 395$) received permission from their parents to participate; 5% of these children ($n = 18$) were absent at the time of data collection. In the classroom, participants were asked to complete self-report questionnaires and trained research assistants collected saliva samples. To maximize honest responding, research assistants reminded participants that their responses would be kept confidential. Further, they instructed participants to answer questions individually and in silence.

Measures

Smoking Status—Smoking status was first determined with the item: “Have you smoked a whole cigarette in the last 6 months?” Then, to verify that they were never-smokers, the smoking status of participants who answered *no* to this question ($n = 338$) was cross-validated with three additional questions that included the response option “I have never smoked.” Participants who consistently reported they had never smoked on all three questions were classified as never-smokers and retained for analyses ($n = 327$).

Social Smoke Exposure—Social smoke exposure was measured with two separate variables: number of current smokers in the social environment and number of situations where SHS exposure currently occurs. Number of smokers was calculated by summing the number of current smokers among parents, siblings, and closest friends (range 0–9 smokers). Number of situations of SHS exposure was calculated by summing the number of situations in which participants observed their parents (30 situations; e.g., drinking coffee, after work, with guests), siblings (30 situations; e.g., back from school, when bored), and peers smoke

(27 situations; e.g., before school, during recess, at lunch). The number of situations of SHS exposure ranged from 0 to 87 situations. Participants endorsed all the situations in which they observed their parents, siblings, and friends smoke from a checklist comprising the 87 situations (Racicot and McGrath, 2009).

Pharmacological Exposure—Pharmacological exposure to nicotine was measured with salivary cotinine. The samples were sent to Salimetrics (State College, PA) for cotinine assays completed in duplicate. All samples were tested with a high sensitivity enzyme immunoassay (Salimetrics, 2007b), which has a detection limit of 0.05 ng/ml; 20 µl of saliva sample were used for each determination. Range of standard curve was 0.8–200 ng/ml, mean intra-assay coefficient of variation was 4.1%, and mean inter-assay coefficient of variation was 6.6%. Accuracy averaged 95%, and linearity of dilution averaged 105%. The average of the two assays was used for analyses. Using the same strategy as Haw and Gruer (2007), values below the detection limit were assigned half the value of the detection limit (0.025 ng/ml). Salimetrics (2007a) indicates that, for nonsmokers, values below 15 ng/ml suggest one is not exposed to SHS, whereas values between 15 and 50 ng/ml suggest one is exposed to SHS. These values, however, should be used as general guidelines and not strict cutoff points for categorizing individuals according to their levels of SHS exposure. Differences in respiratory rate, nicotine metabolism, and intensity of SHS exposure could lead to misclassifications of SHS exposure levels (Salimetrics, 2007a).

Perceived Nicotine Dependence—Perceived nicotine dependence was measured with the Nicotine Dependence Scale for Adolescents (NDSA; Nonnemaker et al., 2004). A composite score, created by summing the six items (e.g., “I sometimes have strong cravings where it feels like I am in the grip of a force that I cannot control: 0 = Not at all true, 3 = Very true”), ranges from 0 (no perception of nicotine dependence) to 17 (highest perception). The NDSA shows good internal consistency (Cronbach’s $\alpha = 0.81$).

Smoking Susceptibility—Smoking susceptibility was defined as a lack of a firm commitment to not smoke among never-smokers (Leatherdale, Brown, Cameron, and McDonald, 2005; Leatherdale, Cameron, Brown, Jolin, and Kroeker, 2006; Pierce, Choi, Gilpin, Farkas, and Merritt, 1996). Smoking susceptibility was measured using two items (e.g., “Have you ever been curious about smoking a cigarette: 0 = No, 1 = Yes”) from the pan-Canadian Youth Smoking Survey (Health Canada, 2007) and three items commonly used in the literature (e.g., “If one of your best friends were to offer you a cigarette, would you smoke it: 0 = Definitely no, 3 = Definitely yes”; Leatherdale et al.; Pierce et al.). A composite score was created by summing the five items, and the total score ranged from 0 (no susceptibility) to 11 (highest susceptibility). Previous cross-sectional data showed that smoking by friends, schoolmates, and parents contributed to smoking susceptibility among never-smoking youth (Leatherdale, McDonald, Cameron, Jolin, and Brown, 2006), while longitudinal data evidenced that smoking susceptibility among never-smokers at baseline predicted smoking initiation at 4-year follow-up (Pierce et al.).

Smoking Expectancies—Using the French–Canadian version (Racicot, McGrath, Hine, O’Loughlin, and Guyon, 2008) of the Smoking Expectancy Scale for Adolescents (Hine,

Honan, Marks, and Brettschneider, 2007), items were categorized based on expected benefits and expected costs (second-order model; Hine et al.). Using a 10-point Likert scale (0 = completely unlikely and 9 = completely likely), participants rated the likelihood that each consequence would happen to them if they smoked cigarettes. Scores for expected costs and expected benefits were calculated as the sum of the items for each category. This scale shows good internal consistency (Cronbach's α : expected costs = 0.94; expected benefits = 0.92; Racicot et al.). Previous cross-sectional data demonstrated that smoking expectancies are associated with smoking by parents and peers (Hine et al.), while longitudinal data showed that they mediate the relationship between exposure to smokers and prospective smoking (Hine, McKenzie-Richer, Lewko, Tilleczek, and Perreault, 2002).

Data Analyses

Multiple linear regression was conducted to test hypotheses. Prior to testing correlations and regression models, cotinine, number of smokers, and number of situations were log-transformed to correct for positive skewness and respect assumptions of normality. Predictors (cotinine, number of smokers, and number of situations) were first modeled individually for each outcome variable (perceived nicotine dependence, smoking susceptibility, expected costs, and expected benefits). All three predictors were then modeled simultaneously for each outcome variable. All models were age adjusted (see Table 1 for correlations). All models were also tested including both age and gender, but results did not differ and gender did not significantly contribute to the models (results not depicted).

Results

Descriptive Statistics

The majority of participants reported that neither parent smoked (68.6%), compared with one parent who smoked (20.3%) or both parents (11.1%). The average number of situations in which parents smoked was 2.96 ($SD = 5.86$). Similarly, the majority reported having no siblings who smoked (91.7%), compared with having at least one sister or brother who smoked (7.1%) or at least two siblings who smoked (1.2%). The average number of situations in which siblings smoked was 0.47 ($SD = 2.25$). Likewise, the majority reported none of their friends smoked (78.8%), compared with one friend who smoked (9.8%), two (6.2%), three (2.2%), four (0.9%), or five or more (2.2%). The average number of situations in which peers smoked was 0.47 ($SD = 1.28$).

Salivary cotinine values were below the detectable limit (0.05 ng/ml) for 61 participants, and two participants did not provide enough saliva to conduct the assay. Half the value of the detection limit (0.025 ng/ml) was assigned for these participants (Haw and Gruer, 2007). (An alternative method for handling cotinine data described by Pickett, Schober, Brody, Curtin, and Giovino (2006) was also conducted. The results did not differ and are not presented.) Analyses were also conducted with and without the participants who did not provide a large enough sample size. Excluding these two participants did not change the results, so they were retained in the final analyses. Mean value for cotinine was 0.71 ng/ml ($SD = 1.90$).

Mean score ($M = 0.25$, $SD = 0.95$) on the NDSA was lower in our sample, compared with the mean in the validation study which only included current smokers ($M = 6.88$, $SD = 4.55$; Nonnemaker et al., 2004). Mean score for smoking susceptibility was low ($M = 1.26$, $SD = 1.90$), but 46.5% of participants endorsed at least one item, which is comparable to the percentage (41.0%) found by Bélanger et al. (2008). Mean score for expected costs was 6.15 ($SD = 2.02$) and 2.18 ($SD = 1.45$) for expected benefits. Hine et al. (2007) found similar results (expected costs: $M = 6.20$, $SD = 1.66$; expected benefits: $M = 2.66$, $SD = 1.80$). Tables 2 and 3 present descriptive statistics for cotinine and the outcome variables based on different levels of social smoke exposure.

Social and Pharmacological Exposure as Predictors of Perceived Nicotine Dependence

Four models were tested to predict perceived nicotine dependence (see Table 4). When predictors were modeled individually, number of smokers predicted perceived nicotine dependence ($F(2, 326) = 6.47$, $p = .05$) and explained 2% of the variance. Cotinine ($F(2, 326) = 0.28$, $p = .60$) and number of situations ($F(2, 326) = 0.93$, $p = .34$) did not predict perceived nicotine dependence. When modeled simultaneously, number of smokers independently predicted perceived nicotine dependence ($F(4, 326) = 8.07$, $p = .01$) and explained 2.4% of the variance. Cotinine ($F(4, 326) = 1.73$, $p = .19$) and number of situations ($F(4, 326) = 0.81$, $p = .37$) did not independently predict perceived nicotine dependence.

Social and Pharmacological Exposure as Predictors of Smoking Susceptibility

Four models were tested to predict smoking susceptibility. When modeled individually, number of smokers ($F(2, 326) = 18.80$, $p = .001$) and number of situations ($F(2, 326) = 8.80$, $p = .01$) predicted smoking susceptibility and explained 5.5% and 2.6% of the variance, respectively. Cotinine ($F(2, 326) = 0.20$, $p = .65$) did not predict smoking susceptibility. When modeled simultaneously, only number of smokers independently predicted smoking susceptibility ($F(4, 326) = 9.76$, $p = .01$) and explained 2.9% of the variance. Cotinine ($F(4, 326) = 2.63$, $p = .11$) and number of situations ($F(4, 326) = 0.02$, $p = .88$) did not independently predict smoking susceptibility.

Social and Pharmacological Exposure as Predictors of Expected Costs of Smoking

Four models were tested to predict expected costs. When modeled individually, linear regression yielded that cotinine ($F(2, 326) = 0.00$, $p = .95$), number of smokers ($F(2, 326) = 0.03$, $p = .87$), and number of situations ($F(2, 326) = 0.43$, $p = .51$) did not predict expected costs. When modeled simultaneously, linear regression showed that cotinine ($F(4, 326) = 0.38$, $p = .54$), number of smokers ($F(4, 326) = 0.37$, $p = .55$), and number of situations ($F(4, 326) = 1.09$, $p = .30$) did not predict expected costs.

Social and Pharmacological Exposure as Predictors of Expected Benefits of Smoking

Four models were tested to predict expected benefits. When modeled individually, number of smokers predicted expected benefits ($F(2, 326) = 4.22$, $p = .05$) and explained 1.3% of the variance. Cotinine ($F(2, 326) = 0.48$, $p = .49$) and number of situations ($F(2, 326) = 0.63$, $p = .43$) did not predict expected benefits. When modeled simultaneously, linear regression

showed that number of smokers ($F(4, 326) = 5.44, p = .05$) independently predicted expected benefits and explained 1.7% of the variance. Cotinine ($F(4, 326) = 0.13, p = .72$) and number of situations ($F(4, 326) = 1.84, p = .18$) did not independently predict expected benefits.

Conclusions

The present study evaluated whether social and pharmacological exposure to SHS predicted smoking precursors among never-smoking youth. Unlike previously reported research among young never-smokers, this study simultaneously evaluated both social and pharmacological exposure to SHS as possible predictors of precursors to smoking.

Results corroborated and extended those of Bélanger et al. (2008) whereby number of smokers predicted greater perceived nicotine dependence and smoking susceptibility among never-smoking adolescents, which supported our first hypothesis. Consistent with the findings by Hine et al. (2007), number of smokers also predicted greater expected benefits. Moreover, number of situations of SHS exposure significantly predicted smoking susceptibility, which supported our second hypothesis. To our knowledge, this is the first study to show that the number of situations of SHS exposure places never-smoking youth at a higher risk for smoking. This finding implies that future studies should not only ask participants about the number of smokers they are exposed to but also about the number of situations in which these smokers consume cigarettes. Although number of situations was no longer significant when it was modeled with number of smokers, the strong correlation among these two variables ($r = 0.79, p = .001$) likely explains this result. Developing and validating a scale measuring situations of SHS exposure will be necessary to further explore this question. Taken together, these findings show that young never-smokers exposed to smokers are more likely to perceive they are dependent on smoking, to be susceptible to smoking, and to hold positive beliefs about the consequences of smoking. Such adolescents are more likely to initiate smoking.

Unlike social exposure to SHS, pharmacological exposure did not predict smoking precursors. This finding did not support our third or fourth hypothesis and was inconsistent with previously reported research (Becklake et al., 2005; Okoli et al., 2007). Characteristics of our sample likely explain this finding. Cotinine values were low ($M = 0.71, SD = 1.90$), suggesting no or little recent pharmacological exposure to nicotine. The majority of participants (53.5%) self-reported they were exposed to no smokers. Having too few participants with moderate to high SHS exposure created power limitations when testing our hypotheses. A more heterogeneous sample with no, low, moderate, and high exposure to SHS should be recruited in future research to better test this question. Another explanation could be that only one saliva sample was collected. While this biomarker is a good indicator of SHS exposure, cotinine has a half-life of 32–82 hr in children (Jaakkola and Jaakkola, 1997) and thus captures SHS exposure over the previous 1–3 days.

Sample characteristics and methodological differences between the present study and previous research may explain why cotinine did not predict smoking precursors. Becklake et al. (2005) measured smoking initiation, but not smoking risk among never-smokers. Okoli et al. (2007) used hair nicotine, which reflects SHS exposure over the past month (Al-Delaimy,

2002). Furthermore, adults exposed to high levels of SHS in bars and restaurants (Okoli et al.) were more likely to absorb greater amounts of nicotine than youth in our study.

Strengths

Despite the limitations noted above, this study has a number of important strengths. First, self-report measures were largely derived from the Youth Smoking Survey (Health Canada, 2007), a national epidemiological survey for which normative data are available. Second, our study evaluated the effects of SHS exposure on four known precursors to smoking initiation. Third, pharmacological exposure to nicotine was measured with cotinine assays.

Recommendations for Future Research

Salivary cotinine values in this study represent short-term exposure to SHS (Jaakkola and Jaakkola, 1997). Future studies should also use hair nicotine biomarkers to evaluate cumulative exposure to SHS over longer time intervals, given that hair nicotine represents an exposure to SHS of about one month (Al-Delaimy, 2002). Further, future studies should measure nicotine with passive nicotine dosimeters that sample ambient air at home or at school (Eisner, Katz, Yelin, Hammond, and Blanc, 2001; Hammond and Leaderer, 1987). Such monitors provide a more accurate measure of the total amount of SHS over the course of a week and provide estimates that are not biased by nicotine metabolism.

Recent work on nicotine metabolism has shown that smokers who are genetically predisposed to metabolizing nicotine slowly smoke a lower number of cigarettes (Malaiyandi, Sellers, and Tyndale, 2005) and progress to nicotine dependence more rapidly (O'Loughlin et al., 2004). Genetic differences in metabolic rates are thus pertinent to the interpretation of biomarker values. As such, individuals who metabolize nicotine rapidly may have lower cotinine levels than individuals with similar levels of SHS exposure and slower metabolic rates. Future studies should collect DNA samples to facilitate the interpretation of cotinine values.

Final Remarks

Consistent with previous studies, exposure to smokers contributed to smoking precursors, including reports of perceived nicotine dependence by never-smokers. The number of SHS exposure situations contributed to smoking susceptibility but only when entered individually into the regression model. Results indicate that future studies should better evaluate social smoke exposure by measuring both the number of smokers and situations of SHS exposure. Such an approach would permit the identification of different exposure patterns (e.g., only one parent smokes in very few situations but smokes very heavily vs. both parents are smokers in a variety of settings but smoke very infrequently). Implications for such findings include that prevention efforts and media campaigns should not only stress who is smoking in youth's environment but also where and when. Given that children who are highly exposed to SHS are at an increased risk for smoking, prevention efforts should be tailored to the needs of this subgroup. Prevention programs should also educate the public about the negative repercussions of modeling tobacco use on youth.

Due to the low cotinine values observed in our sample, cotinine was not associated with smoking precursors. To better evaluate pharmacological exposure, future studies should include a more heterogeneous sample of participants with a broader range of SHS exposure. It is further recommended that future studies use longitudinal data, self-report questionnaires and multiple informants, objective measures of SHS exposure, and DNA analyses to account for genetic differences in nicotine metabolism. Importantly, this line of research may provide stronger arguments to ban smoking in public places, cars, and multi-unit dwellings. This could not only improve youth's health but also reduce the likelihood that they initiate smoking.

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Table 1

Zero-Order Correlations of Predictor and Outcome Variables

Variables	1	2	3	4	5	6	7	8
1. Age	–	.14 *	.14 **	.18 **	–.05	–.01	.16 **	.14 *
2. Cotinine		–	.47 **	.62 **	–.00	.04	–.01	.04
3. Number smokers			–	.79 **	–.02	.11 *	.16 **	.25 **
4. Number situations				–	–.05	.04	.08	.18 **
5. Expected costs					–	.09	–.12 *	–.17 **
6. Expected benefits						–	.10	.15 **
7. Perceived nicotine dependence							–	.31 **
8. Smoking susceptibility								–

Note.

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

Table 2

Means and *SD* for Salivary Cotinine and Smoking Precursors Based on the Total Number of Smokers of Whom Participants Were Exposed

Number of smokers	<i>n</i> (%)	Cotinine	Perceived nicotine dependence	Smoking susceptibility	Expected costs	Expected benefits
		<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
0	177 (54.1)	0.20 (0.53)	0.19 (0.79)	0.88 (1.59)	6.19 (2.03)	2.06 (1.35)
1	63 (19.3)	0.72 (1.11)	0.14 (0.59)	1.54 (2.21)	6.01 (2.26)	2.31 (1.55)
2	54 (16.5)	1.41 (3.00)	0.31 (1.08)	1.50 (1.78)	6.13 (1.95)	2.14 (1.55)
3–9	33 (10.1)	2.25 (3.66)	0.71 (1.71)	2.36 (2.42)	6.22 (1.66)	2.66 (1.56)

Note. Using frequency distributions, participants were categorized into four groups based on a composite score of parental, sibling, and peer smoking. The groups were aggregated based on exposure level ranging from no exposure to highest exposure (3–9 smokers).

Table 3

Means and *SD* for Salivary Cotinine and Smoking Precursors Based on the Total Number of Situations in Which Participants Were Exposed to SHS

Number of situations	<i>n</i> (%)	Cotinine	Perceived nicotine dependence	Smoking susceptibility	Expected costs	Expected benefits
		<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
0	172 (52.6)	0.16 (0.19)	0.20 (0.82)	0.94 (1.75)	6.24 (2.05)	2.08 (1.34)
1–3	59 (18.0)	0.38 (0.88)	0.22 (0.95)	1.51 (1.92)	6.22 (1.77)	2.44 (1.55)
4–9	46 (14.1)	1.17 (2.49)	0.29 (1.08)	1.70 (2.05)	5.79 (2.11)	2.30 (1.71)
10–87	50 (15.3)	2.57 (3.53)	0.42 (1.25)	1.66 (2.07)	6.09 (2.15)	2.13 (1.43)

Note. Using frequency distributions, participants were categorized into four groups based on the total number of situations in which they were exposed to parental, sibling, and peer smoking.

Table 4

Multiple Linear Regression Estimates for Perceived Nicotine Dependence, Susceptibility, and Smoking Expectancies

Predictor variable	Perceived nicotine dependence			Smoking susceptibility			Expected costs			Expected benefits		
	β	t	η^2	β	t	η^2	β	t	η^2	β	t	η^2
Model 1												
Age	0.16	2.96**	.03	0.13	2.37*	.02	-0.05	-0.97	.00	-0.02	-0.29	.00
Cotinine	-0.03	-0.53	.00	0.03	0.45	.00	0.00	0.06	.00	0.04	0.69	.00
Model 2												
Age	0.14	2.54*	.02	0.10	1.88	.01	-0.05	-0.94	.00	-0.03	-0.48	.00
Smokers	0.14	2.54*	.02	0.23	4.34**	.06	-0.01	-0.16	.00	0.12	2.06*	.01
Model 3												
Age	0.15	2.69**	.02	0.11	1.91	.01	-0.05	-0.84	.00	-0.02	-0.33	.00
Situations	0.05	0.96	.00	0.16	2.97**	.03	-0.04	-0.66	.00	0.05	0.79	.00
Model 4												
Age	0.15	2.77**	.02	0.11	2.00*	.01	-0.05	-0.86	.00	-0.02	-0.35	.00
Cotinine	-0.09	-1.32	.01	-0.11	-1.62	.01	0.04	0.61	.00	0.03	0.36	.00
Smokers	0.25	2.84**	.02	0.27	3.12**	.03	0.06	0.60	.00	0.21	2.33*	.02
Situations	-0.09	-0.90	.00	0.02	0.15	.00	-0.11	-1.05	.00	-0.14	-1.35	.01

Note. All models were age adjusted. Models 1–3 were tested with each predictor variable singularly. Model 4 was tested with all three predictor variables entered simultaneously. β = standardized beta coefficient; η^2 = eta squared, estimate of effect size.

*
 $p < .05$;

**
 $p < .01$.