Pharmacological and Social Smoke Exposure as Differential Predictors of Smoking Risk in Never-Smoking Youth

Simon Racicot

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ABSTRACT

Pharmacological and Social Smoke Exposure as Differential Predictors of Smoking Risk in Never-Smoking Youth

Simon Racicot

Studies investigating smoking risk among children and adolescents have typically focused on social smoke exposure via parents, siblings, and peers. Recently, researchers found that second-hand smoke exposure measured with biomarkers among never-smokers uniquely predicted smoking initiation and greater endorsement of withdrawal sensations. Based on these findings, researchers posited a physiological pathway between second-hand smoke exposure and smoking behaviour may exist. The aim of the present study was to simultaneously investigate whether social smoke exposure and pharmacological exposure to nicotine both uniquely contribute to greater smoking risk among never-smoking youth.

Participants included 338 never-smoking youth (53.5% females) aged 11-13 years (M = 12.68, SD = 0.67) attending 6th or 7th grade in French-speaking schools. Participants completed self-report questionnaires measuring their own smoking behaviours, social smoke exposure (parents, siblings, peers, school), and known risk factors for eventual smoking (smoking expectancies, smoking susceptibility, perceived nicotine dependence). Each participant also provided a saliva sample and an expired breath sample, from which cotinine and carbon monoxide biomarkers were derived, to objectively measure second-hand exposure.

Structural equation modeling was used to test the research hypotheses. Pharmacological exposure was not associated with smoking risk. Social smoke exposure of parental and peer smoking were significantly associated with smoking risk. When considered simultaneously, despite having models with acceptable to

good fit, pharmacological and social smoke exposure together largely explained only a small proportion of the variance in smoking risk (1.4-4.7%), with the exception of peer smoking which explained considerable variance (58%). These findings do not suggest that pharmacological and social smoke exposure are differential predictors of smoking risk. Further, they do not support the possibility of a physiological pathway from second-hand exposure to smoking behaviour. Rather, the results suggest biomarkers may actually be a good proxy for social smoke exposure. To better evaluate the possibility of a physiological pathway, future studies should aim to recruit participants with a wider range of smoke exposure (i.e., low, moderate, or high exposure) and to more precisely measure longer-term exposure to second-hand smoke (e.g., hair nicotine, DNA encoding for CYP2A6, CYP2B6, and CYP2E1 enzymes which metabolize nicotine).

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Predictors of Smoking Risk in Never-Smoking Youth

Tobacco smoking is an important public health issue that usually begins during adolescence (Chassin, Presson, Pitts, & Sherman, 2000; Gilpin, Choi, Berry, & Pierce, 1999). Recent Canadian statistics provide evidence that a considerable number of youth still engage in smoking-related behaviours. According to the pan-Canadian 2006-07 Youth Smoking Survey (YSS; Health Canada, 2008), 2.4% of youth in 5-9th grade and 11.4% of youth in 10-12th grade have smoked 100 or more cigarettes in their life. The percentage of youth who inhaled a few puffs of smoke, but did not smoke one whole cigarette was 9.2% among youth in 5-9th grade and 17.4% among youth in 10-12th grade. While 81.5% of youth in 5-9th grade and 51.8% of youth in 10-12th grade have never smoked one whole cigarette, nor have they inhaled even a few puffs of smoke. Among youth in 5-9th grade, 20.8% have tried at least one type of tobacco product; this percentage rises to 54.9% among youth in 10-12th grade. Cigarettes were the most prevalent product used, with 18.5% and 48.2% of youth having tried cigarette smoking in 5-9th grade and 10-12th grade, respectively. Québec was the province with the highest prevalence rate for ever trying cigarette smoking (29.0%) among youth in 5-9th grade. These statistics indicate that high percentages of youth continue to engage in smoking-related behaviours despite wide-spread public health prevention efforts.

Of additional interest is the percentage of young Canadians who are exposed to second-hand tobacco smoke. Specifically, the 2004-05 YSS reported that 23.0% of youth lived with at least one daily smoker who smoked inside of the household (Health Canada, 2007b). Similarly, the 2006 Canadian Tobacco Use Monitoring Survey (CTUMS; Health Canada, 2007a) reported that 9.2% of children aged 0-11

years and 14.2% of adolescents aged 12-17 years were exposed to second-hand smoke. Québec is the province where children and adolescents are most exposed to second-hand smoke, with rates of exposure of 18.4% for children 0-11 years old and 25.8% for adolescents 12-17 years old. Importantly, second-hand smoke exposure has been linked to serious diseases of the respiratory system in youth, including bronchitis (Ugnat, Mao, Miller, & Wigle, 1990) and asthma (Vork, Broadwin, & Blaisdell, 2007; Wilson, 2001). Second-hand smoke exposure is considered deleterious to the health of youth (United States Department of Health and Human Services [USDHHS], 2006). With the exception of health consequences, research has shed little light on other sequelae of second-hand smoke exposure, especially with respect to smoking-related behaviours.

Smoking Terminology with Youth

Many terms and expressions have been used in the smoking literature to define specific smoking behaviours. Based on the 2006-07 YSS (Health Canada, 2008) and the 2006 *Enquête québécoise sur le tabac, l'alcool, la drogue et le jeu chez les élèves du secondaire* conducted by the *Institut de la statistique du Québec* (Dubé & Camirand, 2007), smoking status in youth is largely determined by cigarette use over the last 30 days. *Current smokers* have smoked 100 or more cigarettes in their life. Among current smokers, *daily smokers* have smoked cigarettes everyday in the last month, whereas *non-daily* or *occasional smokers* have smoked cigarettes in the past month, but not everyday. *Never-smokers* have never smoked one whole cigarette and have never inhaled a few puffs of tobacco smoke (Health Canada, 2008). Never-smokers have also been defined as persons who have smoked less than one whole cigarette (Dubé & Camirand, 2007). *Ever-smokers* have smoked at least one whole cigarette or have inhaled a few puffs of smoke (Health Canada, 2008). *Puffers* have

inhaled a few puffs of tobacco smoke, but have never smoked one whole cigarette. *Experimental smokers* have smoked at least one whole cigarette but fewer than 100 cigarettes in the past month, whereas *former experimental smokers* did not smoke in the past month. *Former smokers* have smoked 100 or more cigarettes but did not smoke in the past month. *Non-smokers* comprise both former smokers and never-smokers (Dubé & Camirand, 2007).

Smoking initiation, *smoking uptake*, or *smoking onset* are three expressions in the smoking literature that largely signify transitioning from never smoking to ever smoking (e.g., Becklake, Ghezzo, & Ernst, 2005). *Continued smoking* or *maintenance* usually refers to ever-smoking at baseline and current smoking at a later time point (e.g., O'Loughlin, Paradis, Renaud, & Gomez, 1998). *Smoking susceptibility*, which comprises smoking intentions and self-efficacy, is defined as the absence of a strong commitment to not smoke on the part of never-smokers (Pierce, Choi, Gilpin, Farkas, & Merritt, 1996). *Smoking behaviour* is a widely used expression that broadly refers to all the definitions presented above.

Nicotine dependence is officially defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) as a "maladaptive use" of tobacco products that lasts at least one year (American Psychiatric Association [APA], 1994). DSM-IV criteria used to diagnose nicotine dependence in smokers include symptoms of tolerance and withdrawal. Specifically, *tolerance* refers to a need for "greater doses of nicotine to reach any desired effect". *Tolerance* is also observed when the "same doses of nicotine contribute to reduced effects". Nicotine withdrawal occurs within a day after reducing or ceasing to consume nicotine. *Withdrawal* is characterized by "depressed mood, insomnia, irritability, restlessness, anxiety, or concentration problems". Other DSM-IV criteria for nicotine dependence include the intake of

larger doses of nicotine over a longer period than what was originally planned, and a marked difficulty to reduce nicotine use. By definition, nicotine dependence is applicable to smokers who have been consuming tobacco products for 12 months or more. Nicotine dependence is typically diagnosed by healthcare professionals using clinical interviews combined with clinical judgment; in comparison, questionnaires are used to identify nicotine dependence in research studies.

Theories of Smoking Behaviour

Tobacco smoking in youth is a complex and multifaceted phenomenon. Social scientists have developed theories to try to explain why certain children and adolescents begin to smoke tobacco products, while others do not. In a comprehensive literature review article, Petraitis, Flay, and Miller (1995) reviewed 14 theories that attempt to explain the factors that contribute to substance use. Particularly, four theories which pertain specifically to tobacco smoking in youth have been discussed by Collins and Ellickson (2004). These four theories are: Theory of Planned Behaviour, Social Learning Theory, Social Attachment Theory, and Problem Behaviour Theory.

Theory of Reasoned Action (Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975) is a theory which places emphasis on cognitive processes. This theory posits the intentions of adolescents to smoke tobacco or not will be determined by their beliefs about the costs and benefits of using tobacco. Thus, this theory predicts that adolescents who believe that smoking tobacco will provide them with more benefits than costs are more likely to smoke. Moreover, this theory posits that adolescents who perceive that their parents, siblings, or peers encourage them to smoke are more likely to consume tobacco. Similarly, the *Theory of Planned Behaviour* (Ajzen, 1985, 1988), which is a revision of the Theory of Reasoned Action, posits that adolescents

who are more likely to smoke perceive more benefits than costs, believe other people expect them to smoke, and believe they possess a sense of personal control to accomplish their intention to smoke or perceive they would be unable to refuse to smoke if they were offered cigarettes. Hill, Boudreau, Amyot, Dery, and Godin (1997) found that applying the Theory of Planned Behaviour increased understanding of the stages of smoking acquisition in youth.

Social Learning Theory (Akers, 1977) proposes that adolescents will be more likely to smoke tobacco if they pay attention to the attitudes of, or observe and imitate the behaviour of role models who smoke, such as parents, siblings, or peers. This observation may contribute to the development of expectancies towards smoking. For example, youth who observe their father smoking when he is anxious and relaxed after smoking may come to believe smoking is a good coping skill for anxiety as well as a good technique to relax. Additionally, youth who imitate the behaviour of their role model may expect to be socially reinforced the same way the role model is socially reinforced. For example, if children perceive that smokers are more popular, they may come to believe they will increase their level of popularity if they smoke. Another notion central to the Social Learning Theory is the concept of self-efficacy (Bandura, 1986). Similar to the idea of personal control in the Theory of Planned Behaviour, self-efficacy refers to the idea that youth believe they have the ability to perform a behaviour (i.e., they believe they can smoke) or that they are incapable of refusing any cigarette offers. Support for the Social Learning Theory to better explain smoking acquisition in youth has been reported by Leatherdale, Brown, Cameron, and McDonald (2005).

Social Attachment Theory (Elliott, Huizinga, Ageton, 1985; Hirschi, 1969) proposes that adolescents with low attachment to conventional institutions that

discourage deviancy are more likely to smoke. For example, an adolescent who has very poor ties with his family, his school, or his church is more likely to have deviant friends, to possess unconventional values, and to adopt deviant behaviours, such as tobacco smoking. Bauman, Fisher, Bryan, and Chenoweth (1984) provided support for Social Attachment Theory in explaining smoking behaviour in children and adolescents.

Problem Behaviour Theory (Jessor & Jessor, 1977) posits that adolescents who are involved in other problematic activities, such as alcohol drinking, marijuana smoking, robbery, vandalism, or school absenteeism are more likely to smoke tobacco. This theory views tobacco smoking as part of an array of other problematic behaviours that are considered socially unacceptable for youth. Support for the Problem Behaviour Theory has been reported in Conrad, Flay, and Hill (1992).

Although the four theories described above contain distinct features, they all describe smoking risk factors that are of a psychosocial nature, such as perceived costs and benefits of smoking, smoke exposure through role models, or friendship with deviant peers. Theories like the Social Learning Theory place much emphasis on the idea that social exposure to smoking may be associated with the development of pro-smoking attitudes or expectancies and the imitation of this behaviour. Many researchers rely on this theoretical framework to study smoking initiation in youth. The Theory of Planned Behaviour contends that adolescents who have pro-smoking attitudes are more likely to intend to smoke. Unlike the Social Learning Theory, which proposes that social smoke exposure contributes to the development of smoking attitudes, the Theory of Planned Behaviour does not specify the mechanisms whereby attitudes develop. Recently, researchers have started to examine more

closely smoking expectancies and smoking susceptibility as these two constructs have been shown to be early precursors, or risk factors, for smoking behaviour.

Smoking Expectancies

Consistent with *Social Learning Theory* (Akers, 1977; Bandura, 1986), smoke exposure has been associated with the development of smoking expectancies. Substance-related expectancies are cognitive factors that have been predominantly described in the alcohol literature and have been linked to substance use (Christiansen, Smith, Roehling, & Goldman, 1989; Goldman, Del Boca, & Drakes, 1999). Broadly, expectancies can be defined as the expected positive and negative consequences associated with the consumption of a psychotropic substance (Wahl, Turner, Mermelstein, & Flay, 2005). Expectancies also have been identified as an important cognitive factor in the smoking literature (Hine, Summers, Tilleczek, & Lewko, 1997). Smoking expectancies, sometimes also referred to as "smoking consequences" or "smoking outcome expectancies" (Copeland & Brandon, 2002), have been studied in different populations, including university-level students (Brandon & Baker, 1991), adult smokers (Copeland, Brandon, & Quinn, 1995), adolescents (Hine, Honan, Marks, & Brettschneider, 2007), and children (Copeland et al., 2007). Specific measures have been developed for assessing smoking expectancies in adolescents.

One of the strategies used to measure smoking expectancies consists of asking participants to write down as many positive and negative expectancies they can generate in a period of 60 seconds (Anderson, Pollak, & Wetter, 2002; Vidrine, Anderson, Pollak, & Wetter, 2006). All expectancies are then classified into positive expectancies (e.g., "reduce tension") or negative expectancies (e.g., "addiction"). Although this strategy allows the measurement of expectancies that have been directly

generated by the participants, the number of expectancies generated may be limited due to the short duration (60 seconds) the participants are given to generate expectancies. Another concern with this strategy pertains to possible disagreements among researchers with regard to classification of expectancies.

Smoking expectancies have also been measured with scales where participants are asked to rate sets of items. Dalton, Sargent, Beach, Bernhardt, and Stevens (1999) used the Positive and Negative Outcome Expectations of Smoking Scale which comprised seven positive expectancies (e.g., "I think smoking would help me deal with problems or stress") and five negative expectancies (e.g., "If I started smoking regularly, I think it would be very hard for me to stop"). Participants were asked to rate their level of agreement with each expectancy using a Likert scale (i.e., strongly agree to strongly disagree). The advantage of using a set of items over self-generated responses is that all participants have to rate the same items, which represents a stronger quantitative approach than categorizing various responses generated by different participants.

Myers, McCarthy, MacPherson, and Brown (2003) used the Smoking Consequences Questionnaire (SCQ; Brandon & Baker, 1991) and created a short version for adolescents (S-SCQ). The original SCQ is a 50-item questionnaire that measures smoking expectancies in university students. The short version (S-SCQ) contains 21 items which were classified into four categories of expectancies: negative consequences (e.g., "smoking is hazardous to my health"), positive reinforcement (e.g., "cigarettes taste good"), negative reinforcement (e.g., "cigarettes help me deal with anger"), and appetite/weight control (e.g., "smoking controls my appetite"). Respondents rate the likelihood (i.e., the probability of occurrence) on a ten-point

scale (i.e., 0 = completely unlikely, 9 = completely likely). The internal consistency is high with Cronbach alphas ranging from .84 to .95.

Wahl and colleagues (2005) also created an abridged version of the original SCQ for adolescents. Their questionnaire included 13 items that were classified into four categories: taste (e.g., "I enjoy the taste sensations while smoking"), weight control (e.g., "Cigs keep me from eating more than I should"), boredom relief (e.g., "When I'm alone, a cigarette can help me pass the time"), and negative affective management (e.g., "Smoking calms me down when I feel nervous"). Participants rate their level of agreement with each item on a four-point Likert scale (i.e., 1 = disagree, 4 = agree). The internal consistency is high with Cronbach alphas ranging from .86 to .90.

Lewis-Esquerre, Rodrigue, and Kahler (2005) modified the SCQ to create the Adolescent Smoking Consequences Questionnaire (ASCQ). The ASCQ comprises 38 items divided into seven categories: negative affective reduction (e.g., "Smoking helps calm an angry person down"), taste or sensorimotor manipulation (e.g., "Cigarettes taste good"), social facilitation (e.g., "Most popular people smoke cigarettes"), weight control (e.g., "Smoking makes a person less hungry"), negative physical feelings (e.g., "Smoking burns a person's throat"), boredom reduction (e.g., "Smoking gives a person something to do with his or her hands"), and negative social impressions (e.g., "Smoking makes a person seem less attractive"). Respondents rate the likelihood of each item on a five-point Likert scale (i.e., never to always). The internal consistency of the ASCQ is satisfactory with Cronbach alphas ranging from .56 to .88, but is lower than that of other scales (cf. Myers et al., 2003; Whal et al., 2005).

Recently, Hine and colleagues (2007) developed the Smoking Expectancy Scale for Adolescents (SESA) using Australian students in 7^{th}-12^{th} grades. This scale included 43 items that made up eight factors: affect control (e.g., "Feel calm"), appearance costs (e.g., "Smell bad"), social costs (e.g., "Lose respect of your friends"), health costs (e.g., "Hurt your lungs"), addiction (e.g., "Become dependent on nicotine"), social benefits (e.g., "Look more attractive"), boredom reduction (e.g., "Help kill time"), and weight control (e.g., "Control your appetite"). These eight factors were also classified into two higher-order factors: expected costs (e.g., appearance costs, social costs, health costs, and addiction) and expected benefits (e.g., affect control, social benefits, boredom reduction, and weight control). Participants rate the likelihood of each item on a ten-point Likert Scale (0 = completely unlikely, 9 = completely likely). The SESA has high internal consistency with Cronbach alphas ranging from .73 to .87 for the eight-factor model and .86 to .89 for the two higher-order factor model. The authors assert the SESA possesses advantages over the other smoking expectancy scales described earlier because it was designed primarily to assess expectancies in adolescents and was not derived from the scale (i.e., the SCQ) that was originally created to measure expectancies in young adults. Consequently, the SESA measures a reasonable number of types of expectancies (i.e., 8 categories) and contains a satisfactory number of items (i.e., 48). Furthermore, the authors explain the SESA provides researchers with greater flexibility, as it can be used with its eight-factor or two higher-factor model. Importantly, the wording of the items on the SESA is in the second person (e.g., "Lose respect of your friends"), which may result in having participants feel personally concerned about the statement. In addition, the wording does not appear to presume respondents have already smoked

and thus appears to be suitable for never-smokers. The SESA therefore seems to be one of the better scales to measure smoking expectancies in adolescents.

Racicot, McGrath, Hine, O'Loughlin, and Guyon (2008) validated the French-Canadian version of the SESA (SESA-FC). Using the back-translation procedure, three independent translators translated the SESA into Canadian French; any discrepancy was resolved through discussion. This instrument was tested in a French-speaking sample of 276 6th graders (M_{age} = 12.2; SD_{age} = 0.4; 63% female; 97% non-smokers). Principal components analysis with varimax rotation was used; items with factor loadings equal to or greater than .40 were retained for the interpretation of the factors. Analyses indicated the expected benefits and expected costs factors were replicated. In addition, 6 of the 8 factors were satisfactorily replicated with factor loadings averaging .55 (see Table 1). Students in the French-speaking sample did not differentiate appearance costs from health costs and instead perceived these two categories of expectancies as only one factor. Compared to the original validation sample of the SESA (Hine et al., 2007), age or previous tobacco experience may explain this different factor structure. Alternatively, cultural differences may account for differences in smoking expectancies. In summary, the SESA-FC largely replicated the two higher-order factors of the original SESA and could be used in a French-speaking sample of adolescents. Further research on the SESA-FC is required to assess the suitability of its eight-factor model.

Studies have shown an association between social smoke exposure via parents or peers and smoking expectancies. Lewis-Esquerre and colleagues (2005) asked participants about the number of parents and friends who smoke. Using the ASCQ in a sample of 437 non-smokers and smokers attending high schools, aged 11 to 19 years, exposure to parental smoking was significantly associated with lower

Table 1

Factor loadings of smoking expectancy scale for adolescents: French-Canadian version

	First-order factors									Second-order factors	
Items	Affect control	Appearance costs	Social costs	Health costs	Addiction	Social benefits	Boredom reduction	Weight control	Cannot interpret	Expected costs	Expected benefits
Feel relaxed	**.69** **(.83)**	.03 (-.08)	.03 (-.08)	.17 (.03)	.15 (-.04)	.40 (.09)	.06 (.07)	-.01 (-.02)	.13 (.12)	.03	**.79**
Feel calm	**.82** **(.66)**	.11 (-.04)	.17 (-.01)	.06 (.02)	.11 (-.11)	.15 (.03)	-.20 (.04)	.07 (-.10)	-.08 (.02)	.12	**.60**
Feel less stressed	**.57** **(.60)**	.22 (.00)	-.17 (.01)	.23 (-.04)	.09 (-.07)	.18 (-.07)	.17 (.08)	.34 (-.14)	.21 (-.05)	.19	**.68**
Distract negative feelings	**.36** **(.52)**	.31 (.06)	.49 (.09)	.20 (-.12)	.30 (-.06)	.08 (-.11)	-.01 (.05)	.02 (-.23)	.41 (-.07)	.39	**.56**
Smell bad	.02 (-.06)	**.77** **(.83)**	.10 (.07)	.07 (-.01)	.03 (.00)	-.05 (.04)	-.01 (.06)	.07 (.02)	.29 (-.04)	**.78**	.02

Note. Factor loadings on top come from Racicot et al. (2008), whereas factor loadings in parentheses come from Hine et al. (2007). Numbers in bold represent the items loading on a specific factor.

Table 1 *(Continued)*

Items	First-order factors									Second-order factors	
	Affect control	Appearance costs	Social costs	Health costs	Addiction	Social benefits	Boredom reduction	Weight control	Cannot interpret	Expected costs	Expected benefits
Bad taste in mouth	.02 (-.04)	**.83 (.64)**	.14 (.07)	-.04 (-.06)	.01 (-.05)	-.02 (.01)	-.02 (-.08)	.10 (-.05)	.18 (-.05)	**.84**	-.01
Bad breath	.05 (.07)	**.87 (.64)**	.17 (-.09)	-.02 (.19)	.02 (.02)	.02 (-.02)	.09 (-.07)	.01 (.01)	.17 (.10)	**.87**	.01
Stain fingers, teeth	-.04 (-.03)	**.74 (.60)**	-.02 (.00)	-.02 (.18)	.09 (-.12)	-.01 (-.01)	.21 (.08)	.09 (.07)	.19 (.01)	**.73**	.02
Become less popular	-.10 (-.03)	.45 (-.01)	**.23 (.75)**	.00 (-.01)	.11 (.06)	.11 (.04)	.52 (-.03)	.23 (-.06)	-.20 (.03)	**.49**	.14
Feel like outsider	.14 (.06)	.43 (.01)	**.33 (.65)**	-.09 (-.04)	-.02 (-.08)	.03 (.04)	.12 (.06)	.50 (.01)	-.14 (.02)	**.54**	.19
Lose respect of friends	.09 (-.14)	.36 (-.04)	**.71 (.52)**	-.10 (.14)	.11 (-.14)	.03 (-.15)	.09 (-.05)	.12 (.06)	.12 (-.07)	**.48**	.22

Note. Factor loadings on top come from Racicot et al. (2008), whereas factor loadings in parentheses come from Hine et al. (2007). Numbers in bold represent the items loading on a specific factor.

Table 1 *(Continued)*

Items	First-order factors									Second-order factors	
	Affect control	Appearance costs	Social costs	Health costs	Addiction	Social benefits	Boredom reduction	Weight control	Cannot interpret	Expected costs	Expected benefits
Get lung cancer	.07 (-.04)	.85 (.06)	-.03 (-.05)	**.01 (.80)**	.20 (-.10)	-.10 (.00)	.08 (.01)	.04 (-.07)	-.06 (-.05)	**.85**	-.07
Seriously damage health	.13 (-.01)	.67 (-.11)	.12 (.00)	**-.20 (.79)**	.33 (-.17)	.20 (.15)	-.08 (.01)	-.10 (-.05)	.15 (.07)	**.71**	.10
Hurt lungs	.08 (.02)	.86 (.16)	-.05 (.08)	**-.17 (.61)**	.01 (-.09)	.01 (-.09)	-.07 (-.10)	.05 (.02)	.07 (-.01)	**.83**	-.08
Get heart disease	.10 (-.02)	.82 (.22)	.07 (.07)	**.01 (.53)**	.00 (.01)	-.11 (.11)	-.04 (.06)	.02 (.01)	.01 (.08)	**.78**	-.04
Become addicted to cigarettes	.15 (.05)	.75 (.02)	-.05 (.08)	.11 (.08)	**.42 (-.69)**	-.09 (.03)	.07 (-.02)	.17 (-.04)	-.02 (.04)	**.83**	.10

Note. Factor loadings on top come from Racicot et al. (2008), whereas factor loadings in parentheses come from Hine et al. (2007). Numbers in bold represent the items loading on a specific factor.

Table 1 *(Continued)*

	First-order factors									Second-order factors	
Items	Affect control	Appearance costs	Social costs	Health costs	Addiction	Social benefits	Boredom reduction	Weight control	Cannot interpret	Expected costs	Expected benefits
Become dependent on nicotine	.17 (.06)	.56 (-.08)	.15 (-.05)	-.03 (.01)	**.70** **(-.67)**	.08 (.01)	.03 (.01)	.02 (-.01)	.08 (.03)	**.71**	.22
Get hooked	.23 (.02)	.54 (.05)	.00 (.01)	.06 (.19)	**.64** **(-.66)**	.02 (-.13)	.08 (.00)	.08 (-.07)	.03 (-.17)	**.68**	.26
Increase status	.27 (.10)	.06 (.07)	.12 (.00)	.66 (-.06)	.01 (.00)	**.42** **(-.80)**	.01 (.01)	.02 (-.03)	-.05 (-.07)	-.01	**.71**
Look more attractive	.21 (.04)	-.01 (.01)	.00 (-.02)	.62 (-.06)	.02 (-.04)	**.60** **(-.70)**	-.11 (.02)	-.10 (.01)	.09 (.02)	-.15	**.74**
Fit in better with friends	.22 (.02)	-.05 (-.05)	-.04 (-.02)	.33 (-.09)	.06 (-.02)	**.77** **(-.63)**	-.11 (.20)	.08 (-.02)	-.04 (.02)	-.16	**.74**
Feel less bored	.65 (-.04)	.11 (-.03)	-.11 (-.03)	.30 (-.01)	.04 (-.03)	.25 (.02)	**.24** **(.62)**	.21 (-.20)	.15 (.00)	.12	**.75**

Note. Factor loadings on top come from Racicot et al. (2008), whereas factor loadings in parentheses come from Hine et al. (2007). Numbers in bold represent the items loading on a specific factor.

Table 1 *(Continued)*

Items	First-order factors									Second-order factors	
	Affect control	Appearance costs	Social costs	Health costs	Addiction	Social benefits	Boredom reduction	Weight control	Cannot interpret	Expected costs	Expected benefits
Make life less dull	.30 (.15)	-.06 (.07)	.19 (.06)	.33 (.04)	.11 (.07)	.38 (-.06)	**.49 (.59)**	.14 (-.07)	.14 (-.19)	.02	**.66**
Help kill time	.40 (.17)	.00 (.01)	.12 (-.01)	.02 (.00)	.10 (-.08)	.52 (-.04)	**.43 (.57)**	.09 (-.01)	.09 (.01)	.07	**.66**
Prevent overeating	.12 (-.08)	.22 (.02)	.14 (-.12)	.15 (.05)	.49 (-.10)	.31 (.11)	-.01 (.10)	**.44 (-.77)**	-.05 (-.05)	.31	**.56**
Control weight	.21 (.12)	.08 (-.03)	.16 (.00)	.15 (.03)	.23 (-.04)	.44 (-.11)	-.46 (-.09)	**.39 (-.66)**	.13 (.03)	.09	**.64**
Prevent weight gain	.30 (.14)	.28 (-.03)	.14 (.11)	.08 (-.03)	-.09 (.01)	.21 (.00)	.02 (.04)	**.57 (-.65)**	.17 (.03)	.35	**.46**

Note. Factor loadings on top come from Racicot et al. (2008), whereas factor loadings in parentheses come from Hine et al. (2007). Numbers in bold represent the items loading on a specific factor.

endorsement of negative physical feelings and greater endorsement of negative affect reduction. The seven factors on the ASCQ explained 22.0% of the variance in the prediction of smoking status (i.e., non-smoking vs. smoking), after controlling for parental and peer smoking. Importantly, this study demonstrated that youth who were exposed to parental smoking were more likely to have more positive expectancies (e.g., believe that smoking helps manage negative emotions) and fewer negative expectancies (e.g., believe in the harmful effects in smoking) of smoking. In other words, social smoke exposure predicted expectancies, a risk factor for eventual cigarette smoking.

Smoking Susceptibility

Researchers have also examined other early precursors to smoking behaviour including smoking susceptibility, another smoking risk factor. By definition, never-smokers with greater susceptibility at baseline are more likely to become smokers at follow-up than never-smokers with lower susceptibility.

Pierce and colleagues (1996) validated the construct of smoking susceptibility as a cognitive predictor of smoking behaviour among never smoking adolescents. This concept was operationally defined as the absence of a strong commitment to not smoke. The authors conducted a longitudinal study with participants ($N = 4500$) aged 12 to 18 years who were never-smokers at baseline and at follow-up four years later. They answered questions about their own smoking status, as well as that of their family members and peers. Smoking susceptibility was assessed using the three following items: 1) "Do you think you will try a cigarette soon?"; 2) "If one of your best friends was to offer you a cigarette, would you smoke it?"; and 3) "Do you think you will be smoking cigarettes one year from now?". Thus, the construct of smoking susceptibility includes both smoking intentions and refusal self-efficacy. Smoking

experimentation four years later was predicted by social smoke exposure (family member OR = 1.25, peers OR = 1.60, family members and peers OR = 1.84) and susceptibility (low OR = 1.92, high OR = 3.15). These findings suggest susceptibility in never-smokers is a predictor of transitioning from never smoking at baseline to smoking experimentation four years later. Thus, smoking susceptibility is a smoking risk factor.

Unger, Johnson, Stoddard, Nezami, and Chou (1997) also assessed smoking susceptibility to validate this construct. At baseline, participants were 7th graders (N = 687) and were never-smokers. Participants were tested one and two years later. Smoking susceptibility was measured with the following questions: 1) "Do you think you would like to try smoking a cigarette"; 2) "Do you think you will ever smoke everyday?"; and 3) "Do you think you will ever smoke every month?". After controlling for the number of cigarettes smoked by participants, parental and peer smoking, perceived smoking prevalence among peers, number of offers of cigarettes, and positive and negative expectancies towards smoking, smoking susceptibility predicted which 7th graders tried inhaling a puff of smoke (OR = 2.40), smoked one whole cigarette (OR = 2.89), and smoked 2 to 4 cigarettes (OR = 2.88) one year later in 8th grade. Similarly, susceptibility predicted which 7th graders tried inhaling a puff of smoke (OR = 2.64) and smoked one cigarette (OR = 2.26) two years later in 9th grade. Susceptible students differed from non-susceptible students based on the number of smoking friends (t = 2.87, p < .005), endorsement of positive expectancies towards smoking (t = 2.98, p < .005), and the number of cigarette offers (t = 4.57, p < .0001). In sum, smoking susceptibility predicted which 7th graders became smokers one or two years later. Susceptibility could be a pertinent variable to use when predicting smoking initiation or experimentation. Similar to Pierce and colleagues

(1996), Unger and colleagues (1997) support smoking susceptibility is a construct which may help detect which never-smokers may become ever-smokers in the future.

Jackson (1998) investigated the effects of smoking susceptibility on ever smoking in children aged 8 to 10 years old. Children (N = 788) were surveyed at baseline, one year later, and two years later. Participants answered questions about their smoking status, as well as exposure to smoking by family members and peers. Similar to other studies, analyses were carried out on participants who were never-smokers at baseline. Susceptibility was measured with four items similar to those previously described (Pierce et al., 1996). Participants provided the number of cigarettes smoked by family members and the number of friends smoking among the three best friends. Smoking initiation (i.e., endorsing at least one puff or more) two years later was predicted by smoking by one family member (OR = 2.18, $p < .001$), smoking by two family members (OR = 2.34, $p < .001$), low susceptibility (OR = 1.83, $p < .001$), and high susceptibility (OR = 3.74, $p < .0001$). Surprisingly, peer smoking did not predict smoking initiation two years later. This study supported the role of smoking susceptibility as a predictor of smoking initiation in children aged 8 to 10 years who were never-smokers at baseline. It is possible peer smoking did not predict smoking initiation two years later because the authors asked the number of smoking friends among three close friends as opposed to asking the total number of friends who smoke.

Leatherdale, McDonald, Cameron, Jolin, and Brown (2006) studied exposure to smokers as a predictor of smoking susceptibility among never-smokers (N = 2478) in 6th and 7th grade. Smoking susceptibility was assessed with similar questions to those of Pierce and colleagues (1996). Participants also answered questions about exposure to parental and peer smoking. Exposure to smoking by older schoolmates

was measured with the smoking prevalence among 8th graders. Susceptibility to smoking among never-smokers was predicted by mother smoking (OR = 1.63, $p < .01$) and by peer smoking (OR = 2.11, $p < .001$). The idea that mothers usually spend more time than fathers around their children may explain why father smoking was non-significant. Moreover, an interaction between peer smoking and smoking prevalence among older schoolmates significantly predicted susceptibility (OR = 1.03, p =.05). Specifically, the authors found that having two or more close friends smoking and attending a school with a high prevalence of smoking in 8th grade students was a risk factor for smoking susceptibility among 6 and 7th grade students. Hence, the authors explain having smoking friends and attending a school with a high percentage of smoking among older students is an important risk factor for increasing susceptibility among never smoking youth in 6th or 7th grade. This study also found social smoke exposure via mothers, peers, and older schoolmates contributes to greater smoking susceptibility.

Leatherdale and colleagues (2005) sought to evaluate the influence of the school environment on smoking susceptibility. Participants (N = 6679) were never-smokers in 9th to 13th grade. Participants were classified as susceptible or non-susceptible based on a measure of smoking susceptibility (cf. Pierce et al., 1996). Participants answered questions about social smoke exposure at school. The authors calculated the prevalence of daily smoking for each school; they recorded whether smoking occurred on school premises, the school periphery, or across from the school. Greater smoking susceptibility was associated with having more friends who smoked and perceiving students who smoked close to the school. Thus, social smoke exposure via school was a risk factor for smoking susceptibility. Being exposed to smoking on the school periphery is a source of social smoke exposure which includes

visual smoking cues (i.e., manipulating a lighter to light up a cigarette), but no pharmacological smoke exposure.

The literature reviewed above has largely relied on social smoke exposure to explain how smoking behaviour becomes established in children and adolescents. Interestingly, these theories do not consider the pharmacological component of tobacco smoking, which involves exposure to nicotine (Benowitz, 1996b). New lines of research have started to suggest that pharmacological exposure to nicotine may contribute to smoking initiation via a physiological pathway in youth. In the next section, an overview of the physiological effects of nicotine is presented.

Physiological Effects of Nicotine

Nicotine is the principal psychoactive or psychotropic substance in tobacco and has been found to alter the neural mechanisms associated with drug addiction (Laviolette & van der Kooy, 2004; USDHSS, 1988). Nicotine exerts its influence predominantly on nicotinic cholinergic receptors (nAChRs) of the central nervous system (Wonnacott, Sidhpura, & Balfour, 2005). Acetylcholine (ACh) is the natural neurotransmitter which binds to nAChRs and nicotine is an agonist on this type of receptor. Mineur and Picciotto (2008) reported in a review article that nAChRs have different subunits with nine α-subtypes (i.e., α2 to α10) and three β-subtypes (i.e., β2 to β4). Like other drugs, nicotine administration has been found to release dopamine (Balfour, Wright, Benwell, & Birrell, 2000; Di Chiara, 2000), a neurotransmitter associated with pleasure and reward (Domino, 1998). Dopamine is released in the ventral tegmentum area located in the mesolimbic system (Wonnacott et al., 2005). The ventral tegmentum sends its projections to the nucleus accumbens in the mesolimbic system (Balfour, 2002; Balfour, Benwell, Birrell, Kelly, & Al-Aloul,

1998). The pathway from the ventral tegmentum area to the nucleus accumbens has been associated with nicotine addiction.

Recent research on animals suggested the adolescent brain may be particularly vulnerable to nicotine exposure. Slotkin (2002) reported in a review article that nicotine exposure adversely affects the natural maturation of cells in the central nervous system or contributes to cellular death (i.e., apoptosis) in adolescent rats. Upregulation of nAChRs, which signifies an increase in the number of receptors on the surface of dopaminergic neurons (Littleton, 2001; Wonnacott, 1990), has been observed in animal studies. As example, Abreu-Villaça and colleagues (2003) administered nicotine via two experimental conditions (infusion vs. injection) in rats for seven days, starting prepuberally, 30 days post-birth. Brain tissue from the cerebral cortex, midbrain, and hippocampus was assayed at three subsequent time points (+7, +15, +35 days). In the infusion condition, results showed that at the +7 day follow-up, nAChRs were upregulated by 30-40%. Upregulation of nAChRs remained high in the midbrain at the +35 day follow-up, as well as in the cerebral cortex and hippocampus at the +15 day and +35 day follow-ups. In the injection condition, results showed that nAChRs were upregulated by 15-20% when measured at the +7 day follow-up. Receptor upregulation remained high in the midbrain and hippocampus at the +35 day follow-up, but not in the cerebral cortex. Taken together, these results suggest that nicotine administered via two different routes contributes to receptor upregulation in the adolescent brain. Importantly, this study suggests nicotine-related neural changes are long-lasting in rats as nAChRs upregulation persisted for a month after the last nicotine exposure.

Similarly, Trauth, Seidler, McCook, and Slotkin (1999) evaluated the neural effects of nicotine administration on the adolescent brain. Adolescent rats aged 30

days were administered a daily dose of nicotine of 6 mg/kg/day for a period of 17 days. Results showed that after 15 days of daily nicotine administration, similar upregulation of nAChRs was observed in the midbrain, cerebral cortex, and hippocampus. At the +20 day follow-up, nAChR upregulation was observed in the cerebral cortex, hippocampus, and midbrain, but upregulation in the midbrain was significantly smaller that that observed in the cerebral cortex and hippocampus. At the +30 day follow-up, upregulation was observed in all three regions, but greater upregulation was observed in the cerebral cortex when compared to the midbrain and greater upregulation was observed in the midbrain when compared to the hippocampus. At the +45 day follow-up, all three regions were similarly upregulated. Similar to the findings of Abreu-Villaça and colleagues (2003), this study showed that neuronal adaptations to nicotine administration may persist for a month after the last nicotine dose. These animal studies confer strong evidence that nicotine has long-lasting neural effects in adolescent rats.

The neurophysiological effects of nicotine exposure have also been evaluated in humans and are often observed in autopsied brains (Breese et al., 1997). For example, Benwell, Balfour, and Anderson (1988) compared the density of nAChRs binding in the postmortem brains of smokers and non-smokers. Smokers had significantly greater nAChRs density in the hippocampal formation, the hippocampal neocortex, the cerebellar cortex, the gyrus rectus, and the median raphe nuclei; in these brain regions, nAChRs upregulation had increased by 50% to 100%. There were no differences in the medulla oblongata. Similarly, Perry, Dávila-García, Stockmeier, and Kellar (1999) assessed the effects of nicotine in humans. Postmortem brains of eight smokers and eight non-smokers were autopsied. Density of nAChRs was evaluated with autoradiography in three brain regions: prefrontal

cortex, temporal cortex, and hippocampus. When compared to non-smokers, density of nAChRs in smokers was increased by 400% in the prefrontal cortex, 300% in the temporal cortex, and 160-290% in different regions of the hippocampus ($p < .001$). Overall, the results of animal and human studies provide evidence that nicotine exposure is associated with neural changes in the brain. Although nicotine exposure has been linked to nAChRs upregulation, the amount of nicotine exposure necessary to produce these changes is not well understood. It is also unknown whether other types of smoke exposure, such as second-hand smoke exposure in humans, are sufficient to precipitate such neuronal changes in the brain. Further research is required to answer these questions.

Sensitization-Homeostasis Model

Different theoretical models have been developed to explain the neuroadaptations that underlie drug-related behaviour (Robinson & Berrigde, 1993, 2001). DiFranza and Wellman (2005) proposed the *Sensitization-Homeostasis Model* to explain how sensitization develops in smokers. Sensitization is a phenomenon that occurs when repeated exposure to a drug contributes to greater behavioural responses. Thus far, sensitization has been observed in animals and has been associated with greater locomotor activity and nicotine self-administration (DiFranza & Wellman, 2007; Vezina, McGehee, & Green, 2007). DiFranza and Wellman (2005) suggested the Sensitization-Homeostasis Model may be helpful in understanding how sensitization develops in experimental smokers who are sporadically exposed to nicotine. In fact, sensitization among experimental smokers is thought to be one of the factors that contributes to more regular tobacco use (Vezina et al., 2007).

The Sensitization-Homeostasis Model posits that the principal function of nicotine is to suppress craving (DiFranza & Wellman, 2005). These researchers

contend that there are two opposite craving systems in the nervous system: a craving inhibition system and a craving generation system. Nicotine exposure is thought to be associated with nAChRs activation and the release of dopamine in the craving inhibition system, given that dopamine is a neurotransmitter that may have inhibitory effects (Kalat, 2001). Thus, nicotine exposure leads to nAChRs inactivation, which then leads to nAChRs upregulation (Littleton, 2001). Inactivation of nAChRs may last about a day after heavy nicotine exposure (Girod & Role, 2001). DiFranza and Wellman (2005) explained that when experimental smokers wait more than 24 hours to smoke their next cigarette, their upregulated nAChRs re-activate. When they smoke their next cigarette more than 24 hours after the previous one, nicotine activates upregulated nAChRs in the craving inhibition system and further inhibits the craving generation system. Because of the over-inhibition of the craving generation system by the craving inhibitory system, the craving generation system develops compensatory mechanisms to return to normal levels of functioning. The authors further propose that situational cues, such as observing one's father light up a cigarette, promote endorphin release. Endorphins are thought to activate the craving generation system.

Once neuroadaptations like tolerance and withdrawal have become established with repeated tobacco use, the craving generation system becomes increasingly stimulated; thus, it takes greater doses of nicotine for the craving inhibition system to inhibit the craving generation system. In the first 24 hours of nicotine abstinence, nAChRs are inactive and ACh is not sufficient to activate the craving inhibition system. Thus, the craving generation system is not inhibited and leads to craving. DiFranza and Wellman (2005) then explain that when nAChRs re-activate after 24 hours of inactivity, it takes stronger activation in the craving inhibition system to

inhibit the craving generation system. Moreover, situational cues that have been paired with nicotine administration activate the neuronal pathways of the craving generation system, even in the absence of nicotine (Schroeder, Binzak, & Kelley, 2001). In summary, the Sensitization-Homeostasis model posits nicotine activates the craving inhibition system and inhibits the craving generation system. The craving generation then develops homeostatic adaptations to counteract over-inhibition. When nicotine is administered intermittently, as is the case with experimental smokers, the craving inhibition system does not succeed in inhibiting the craving generation system, which results in craving.

Contrary to more general models of drug addiction (Robinson & Berridge, 1993, 2001), the Sensitization-Homeostasis Model was derived specifically from the nicotine literature and is not directly generalizable to other drugs like cocaine or heroin. As such, DiFranza and Wellman (2005) specify that the Sensitization-Homeostasis Model pertains to the hypothesis that the function of nicotine is to suppress craving and not to provide pleasure. Like other models, this model proposes situational cues independently activate the craving generation system, that is, even in the absence of nicotine administration. The Sensitization-Homeostasis Model provides an explanation as to how sensitization develops in new smokers who smoke irregularly with periods of abstinence lasting more than 24 hours. However, the model remains to be tested empirically with human samples. The researchers acknowledge that some arguments are speculative or have been observed exclusively in animals. Intriguingly, like experimental smokers, never-smokers who are exposed to second-hand smoke are also intermittently exposed to nicotine. However, the Sensitization-Homeostasis model does not explicitly provide any explanation as to the possibility of nicotine sensitization in never-smokers via second-hand smoke

exposure. Nonetheless, it sheds light on the neural mechanisms that explain how sensitization develops in novice smokers. The results of further research may be able to extend this model to never-smokers as well.

Measurement of Second-Hand Smoke Exposure

Second-hand smoke exposure has become an important public health issue because of the documentation of its adverse health outcomes (USDHHS, 2006). Recently, Okoli, Kelly, and Hahn (2007) demonstrated second-hand smoke exposure is an important source of nicotine and other carcinogenic chemicals, which supports its relation to diseases. However, there are few data about the relationship between second-hand smoke exposure and smoking behaviour. Jaakkola and Jaakkola (1997) reported in a review article that factors affecting second-hand smoke exposure values include smoke concentration (e.g., number of exposure sources, volume of space, and air ventilation), duration, and frequency of second-hand smoke exposure. Second-hand smoke exposure can be measured with different devices. Jaakkola and Jaakkola (1997) reported that popular measures of second-hand smoke exposure include personal monitors, indoor stationary monitors, interviews and questionnaires, and biomarkers. Of all these measures, biomarkers are the only method that permits the measurement of the amount of second-hand smoke exposure absorbed by an individual. Biomarkers are usually derived from bodily fluids and reflect bodily functions and processing, such as metabolism and elimination (U.S. Environmental Protection Agency, 1992). Thus, biomarkers are the only true measure of one's pharmacological dose of nicotine.

Iwase, Aiba, and Kira (1991) estimated the absorption and intake of nicotine in 17 non-smoking females (aged 18-22 years old). Participants were asked to wear a mask with an inlet and outlet valve, each of which contained a nicotine sampler. Each

participant wore the face mask while in a room where 30 cigarettes were burnt in an hour. Nicotine concentration in both inspired and expired air was measured 10 minutes before exposure, and 10, 30, and 50 minutes after the beginning of the experiment. Nicotine concentrations were determined by gas chromatography. The authors calculated nicotine absorption with the formula: ((nicotine concentration in inspired air – nicotine concentration in expired air) / nicotine concentration in inspired air) X 100. Results showed the average of nicotine absorption was 71.3% (*SD* = 10.2%), with a range of 60% to 80% for the 17 participants. Based on these results, the authors estimated nicotine intake would equal .026 mg/h if a non-smoker was exposed for an hour to a concentration of 100 $\mu g/m^3$. Importantly, this study showed considerable amounts of nicotine contained in second-hand smoke were absorbed by adult non-smokers (60%-80%).

After nicotine has been absorbed into the body, it is metabolized by the liver predominantly with the enzyme CYP2A6 and is transformed into cotinine (Hukkanen, Jacob, & Benowitz, 2005). Approximately 75% of nicotine is converted into cotinine (Benowitz & Jacob, 1994). Nicotine has a half-life of 2-3 hours, whereas cotinine has a half-life of 16-17 hours in adults (Benowitz, 2008). However, Jaakkola and Jaakola (1997) reported the half-life of cotinine is longer in children (i.e., 32-82 hours) and newborns (i.e., 160 hours). Okoli and colleagues (2007) reported that studies using biomarkers have collected nicotine in hair (Al-Delaimy, Crane, & Woodward, 2002), serum and semen (Pacifici et al., 1995), and toenails (Al-Delaimy, Mahoney, Speizer, & Willet, 2002). Cotinine has been collected in hair (Matt et al., 2004), urine (Al-Delaimy et al., 2002), saliva (Scherer, Meger-Kossien, Riedel, Renner, & Meger, 1999), serum (Seccareccia et al., 2003), and semen (Pacifici et al., 1995). Okoli and colleagues (2007) further reviewed studies indicating an association between self-

reported second-hand smoke exposure and biomarkers (Al-Delaimy, Fraser, & Woodward, 2001; Berman et al., 2003), and between air nicotine and biomarkers (Chan, Chen, & Wang, 1995; Johnsson et al., 2003). In sum, using biomarkers to measure second-hand smoke exposure is considered valid and efficient, given that biomarkers are highly correlated with self-report questionnaire and air monitoring. Compared to nicotine, cotinine is a metabolite that is quite stable throughout the day, which explains why many researchers favour cotinine biomarkers over nicotine (Benowitz, 1996a). Because of its longer half-life, cotinine is usually preferred over nicotine when it is measured in bodily fluids. However, cotinine provides only a measure of short-term second-hand smoke exposure (3-4 days; Benowitz, 1999), while hair nicotine has been found to provide a measure of longer term (i.e., one month) exposure (Al-Delaimy, 2002). The choice of a specific biomarker depends on both scientific and practical reasons.

Second-Hand Smoke Exposure and Biomarkers

Al-Delaimy and colleagues (2001) assessed second-hand smoke exposure in relation to smoking policies in the workplace. Employees of bars ($n = 22$), restaurants ($n = 76$), and other workplaces ($n = 16$) answered questions about their smoking status, the number of cigarettes they smoked on a daily basis, and sources of exposure to second-hand smoke other than the workplace (e.g., exposure at home). Questions pertaining to smoking policies (i.e., no smoke-free policy, 50% smoke-free policy, and 100% smoke-free policy) were answered by the manager of each venue. Each participant provided a hair sample for nicotine assay. In non-smokers, nicotine levels differed based on the smoking policies of venues ($\chi^2 = 26.4$, $p = .0001$). In smokers, nicotine levels corresponded to their own cigarette consumption ($r = .45$, $p = .0018$). Hair nicotine levels differed according to the smoking policies at work even in non-

smokers who were exclusively exposed to second-hand smoke in their workplace (χ^2 = 15.4, p = .0004). Importantly, these results showed that non-smokers working in venues with no smoke-free policy had hair nicotine levels (6.69 ng/mg) comparable to those of smokers (7.92 ng/mg); this difference was not statistically significant (χ^2 = .03, p = .86). Thus, non-smokers who are highly exposed to second-hand smoke have similar levels of nicotine in their body as active smokers.

Dimich-Ward, Gee, Brauer, and Leung (1997) also studied second-hand smoke exposure in relation to the level of exposure in the workplace. Participants were 26 employees (M_{age} = 35.2 years) classified into four groups. In the first group, five participants were smokers with an average consumption of 20 cigarettes per day; four of these five smokers worked in places where smoking was forbidden. In the second group, five non-smokers were exposed to high levels of smoke at work with an average of 38 hours of exposure per week. In the third group, eight non-smokers were exposed to moderate levels of smoke at work with an average of 21 hours of exposure per week. In the fourth group, eight non-smokers worked in places with no second-hand smoke exposure. Every participant provided hair samples for nicotine assay. Hair nicotine discriminated non-smokers based on their level of exposure. Mean value for non-smokers not exposed to second-hand smoke was 0.10 ng/mg; mean value for non-smokers moderately exposed to second-hand smoke was 0.34 ng/mg; and mean value for non-smokers highly exposed to second-hand smoke was 1.03 ng/mg (p = 0.019). Mean value for hair nicotine among smokers was 1.19 ng/mg and nicotine values were strongly associated with daily consumption of cigarettes (r = 0.97, p = 0.01), given that four of the five smokers were not exposed to smoke at work. Importantly, these results showed non-smokers highly exposed to smoke had hair nicotine values (M = 1.03 ng/mg) that were similar to those of smokers (M = 1.19

ng/mg). This study demonstrated a dose-response relationship between hair nicotine levels and level of exposure to second-hand smoke (i.e., low, moderate, and high) in non-smokers. Similar to the findings of Al-Delaimy and colleagues (2001), non-smokers who were highly exposed to second-hand smoke had hair nicotine values comparable to those of smokers.

The results of these studies suggest that non-smokers exposed to second-hand smoke passively consume similar amounts of nicotine as smokers. Given that nicotine is a psychoactive substance associated with addiction (USDHHS, 1988) and sensitization (Vezina et al., 2007), this raises the question of whether nicotine intake in non-smokers, via high levels of second-hand smoke exposure, contributes to neuroadaptations. However, these findings were from adults with high levels of workplace smoke exposure. Recent studies have examined how household smoke exposure may affect children and adolescents. It is also unclear whether the pharmacokinetics of nicotine differs for children and adults.

Willers, Skarping, Dalene, and Skerfving (1995) measured second-hand smoke exposure in non-smoking children and adults. Participants were 7 adults (M_{age} = 40 years) and 14 children (M_{age} = 8.1 years) who were exposed to second-hand smoke on a bus. A total of four smokers smoked a total 78 cigarettes with 1.1 mg of nicotine per cigarette for a period of two consecutive hours. After the two-hour exposure period, the bus was ventilated. One sample of urine cotinine was collected one week before exposure, on the morning before exposure, and from the third to seventh morning after exposure. Additionally, total urine was collected over 24 hours, from the beginning of the exposure on the first day until the end of the second day after exposure. Air nicotine in the bus was measured before, during, and after exposure. Maximum levels of urine cotinine during and shortly after exposure were

22 mg/l in children and 13 mg/l in adults, which signifies that children had higher peak levels of cotinine than adults. Cotinine values formed a plateau after exposure and were also higher in children (18 mg/l) than adults (11 mg/l). The authors estimated the dose of nicotine inhaled by children (63 mg) and adults (129 mg) and found that children obtained higher values (2.3 mg/kg) than adults (1.7 mg/kg) when adjusting for weight. When dividing the total amount of urinary cotinine measured throughout the seven days post-exposure with the estimated nicotine dose, children also obtained higher values than their adult counterparts (27% vs. 16%).

Importantly, this study showed children who are exposed to the same absolute amounts of second-hand smoke as adults absorb relatively higher doses of nicotine. Given that nicotine is associated with neuroadaptations (Benowitz, 2008; DiFranza & Wellman, 2005) and cell damage in youth (Slotkin, 2002), these findings provide evidence second-hand smoke exposure may be a greater risk factor for neuroadaptations in children than adults. In addition to the absorption of nicotine by never-smokers, second-hand smoke exposure represents a source of smoking cues, such as the smell of tobacco smoke.

Smoking Cues

Although nicotine is the primary psychoactive substance in tobacco smoke, researchers have argued the pharmacological effects of nicotine alone do not suffice to provide a comprehensive explanation of the mechanisms underlying smoking behaviour (Rose, Behm, Westman, & Johnson, 2000). One argument frequently used to support this conclusion is that nicotine replacement therapies often fail to help smokers remain abstinent (Rose, 2006). Smoking cues, such as the smell, taste, sight of tobacco smoke, or the situations wherein smoking takes place, are non-nicotinic

stimuli that co-occur with nicotine intake and may be associated with smoking behaviour (Caggiula et al., 2002).

Smoking cues have been found to be paired with nicotine self-administration. Caggiula and colleagues (2001) assessed the effects of nicotine and smoking cues on extinction and reacquisition of self-administration by rats. Rats were placed in a chamber which contained a chamber light, an active and inactive lever, and a cue-light situated above the active lever. Responses on the active lever were associated with nicotine infusion; responses on the inactive lever had no effect. Each infusion was paired with a one-second cue light above the active lever and the commencement of a one-minute period where the chamber light was turned off. During this minute, researchers measured responding, but did not reinforce the act of pressing the lever. After 20 days of self-administration of nicotine with cues, rats were allocated to three different groups during extinction: saline with cues, saline without cues, and nicotine without cues. Infusion rates decreased among rats receiving saline with cues (-58%); infusion rates almost reached extinction among rats receiving saline without cues (-100%); and infusion rates decreased among rats receiving nicotine without cues (-63%). During reacquisition, rats receiving saline with cues were divided into two groups: nicotine with cues (infusion rates reached acquisition levels) and saline with cues (infusion rates reached 50% of the levels reached during acquisition). Rats receiving nicotine without cues received nicotine with cues during reacquisition (infusion rates increased to levels reached during acquisition). Rats receiving saline without cues were divided into three groups during reacquisition: nicotine with cues (infusion rates increased to levels reached during acquisition), saline with cues (infusion rates significantly increased from extinction), and nicotine with cues (infusion levels remained at extinction). This study provides evidence that both

nicotine and smoking cues are associated with self-administration; while the combination of nicotine and smoking cues was most strongly associated with self-administration.

Extending their work in this area, Caggiula and colleagues (2002) next examined the effects of nicotine and smoking cues on nicotine self-administration in drug-naïve rats. Rats were divided into two groups: nicotine with cues and nicotine only. Rats receiving nicotine with cues quickly self-administered nicotine. Rats receiving nicotine only did not increase responding on the active lever; the percentage of responding on the active lever was 73% lower than the in the group where rats received both nicotine and cues. However, the number of infusions in rats receiving nicotine only increased (70%) from day 4-5 to day 19-20. Still, rats receiving nicotine with smoking cues self-administered nicotine significantly more frequently than rats receiving nicotine only. Thus, while nicotine seems necessary for self-administration, the combination of nicotine with smoking cues enhances nicotine self-administration in drug-naïve rats.

The two studies described above provide evidence that environmental cues are associated with nicotine self-administration in animals. These results partially support the hypothesis that smoking cues are risk factors that increased nicotine self-administration, in addition to the pharmacological effects of nicotine itself. Much research has also examined the role of environmental cues on smoking behaviour in humans (cf., Caggiula et al., 2002; Chaudhri et al., 2006; Shiffman et al., 1996). In humans, some smoking cues include observing another person smoking a cigarette, smelling tobacco smoke, or the taste of cigarettes. As example of the powerful effects of smoking cues, former smokers who were exposed to smoking cues were found to have higher intentions to smoke compared to those not exposed to cues. It is thus

plausible that pharmacological exposure via second-hand smoke exposure, combined with pairing of social cues, could exacerbate smoking risk in non-smokers, including youth.

Social Smoke Exposure

Social exposure to tobacco smoking is a consistent and robust risk factor which contributes to smoking initiation. Social smoke exposure refers to the idea of observing others smoking in one's own social environment. In the literature, social smoke exposure via parents, siblings, and friends have been associated with youth's smoking risk factors.

Parental Smoking

Parents are often one of the most important sources of influence on youth. Peterson and colleagues (2006) evaluated the differential contribution of two non-smoking parents, one smoking parent, and two smoking parents during childhood to daily smoking during adolescence. This longitudinal study comprised two cycles: when children were 8-9 years old (baseline) and 17-18 years old (nine years later). At baseline, one parent completed a questionnaire about his or her smoking status (current smoker vs. current non-smoker) and provided the smoking status of the second parent. Nine years later, children (N = 3012) were asked if they smoked cigarettes daily. Having one parent who smoked when the child was 8-9 years old was associated with a greater likelihood of daily smoking nine years later compared to children with two non-smoking parents (OR = 1.90, $p < .01$). Further, having two parents smoking at baseline increased the likelihood of daily smoking nine years later compared to children with one parent smoking (OR = 1.39, $p < .01$) and compared to children with two non-smoking parents (OR = 2.65, $p < .01$). The authors suggested a dose-response relationship appears to exist between the number of parents who were

current smokers when children were 8-9 years old and daily smoking during adolescence, nine years later. Hence, having one parent who smokes is a risk factor, but having two parents who smoke during childhood augments the risk even more. This study evidences greater social exposure to smoke in childhood via parental smoking is more strongly associated with prospective smoking. Notably, the dose-response relationship may partly be explained by greater pharmacological exposure to nicotine; however, the authors did not measure second-hand smoke exposure.

Otten, Engels, van de Ven, and Bricker (2007) evaluated the effects of both current and former parental smoking on five smoking transitions during adolescence (i.e., never smoking to trying, never smoking to monthly smoking, never smoking to daily smoking, trying to monthly smoking, and trying to daily smoking). Participants (N = 7426) were surveyed when they were 11-16 years old at baseline and two years later. They answered questions about their own smoking status (i.e., never-smoker, trier, monthly smoker, and daily smoker) and that of their parents (i.e., current smoker, former smoker, never-smoker). Children of former smokers either provided their own age when their parents ceased smoking or answered that their parents ceased smoking before they were born. Overall, having a higher number of parents who currently smoked was associated with a greater risk of escalating through the smoking transitions, compared to having former smoking parents. Similarly, having a higher number of former smoking parents was associated with a greater risk of escalating through the smoking transitions, compared to having never smoking parents. Compared to children of never smoking parents, the risk for smoking was greater among children of former smokers who ceased smoking when their children were 0 to 7 years of age (OR = 1.76, $p < .01$), 7 to 10 years of age (OR = 1.87, $p <$

.01) and 10 years or older (OR = 1.53, $p < .01$) than children of former smokers who ceased smoking before their birth (OR = 1.18, *n.s.*).

Similar to the previously described study by Peterson and colleagues (2006), Otten and colleagues (2007) assert a dose-response relationship exists between parental smoking and progressing through the smoking transitions. The effects of parental smoking on smoking behaviour among adolescents have been reported in many studies (see reviews: Avenevoli & Merikangas, 2003; Tyas & Pederson, 1998). Other studies have found that parental smoking may not be as important as other risk factors, such as peer smoking in predicting smoking behaviour in adolescents (Distefan, Gilpin, Choi, & Pierce, 1998; O'Loughlin et al., 1998; Vink, Willemsen, & Boomsma, 2003). Conversely, Vitaro, Wanner, Brendgen, Gosselin, and Gendreau (2004) showed that parents may be more important when youth are younger, whereas peer smoking may be more important when youth are older, suggesting a critical window when parents are more important. However, the influence of other family members is also important.

Sibling Smoking

Though studied less frequently than parents, sibling smoking has been shown to predict smoking behaviour among adolescents. Rajan and colleagues (2003) investigated the relationship between childhood exposure to sibling smoking and smoking during adolescence. In a nine-year longitudinal study, the first data collection occurred when participants were in 3rd grade and the second collection occurred when they were in 12th grade (N = 2981). During 3rd grade, one parent provided his or her own smoking status, that of the other parent, and that of their older children. During 12th grade, participants answered questions to determine whether they were daily smokers. After controlling for parental smoking, smoking by older

siblings when children were in 3rd grade was associated with a greater likelihood of daily smoking in these children nine years later (OR = 1.60, $p < .004$). Furthermore, the association between older sibling smoking in 3rd grade and daily smoking among participants in 12th grade did not differ based on parental smoking status. This longitudinal study supported the association between childhood exposure to older sibling smoking and likelihood of smoking in adolescence, regardless of parental smoking. One limitation of this study was that sibling smoking status was reported by parents.

Harakeh, Engels, Vermulst, de Vries, and Scholte (2007) evaluated the differential contribution of sibling smoking and best friend smoking to smoking initiation in a cross-lagged panel design (baseline and one year later). Pairs of siblings included an older sibling (14-17 years of age) and younger sibling (13-15 years of age). Siblings were asked about their smoking status, but only never-smokers at baseline were retained for the analyses. The older and younger siblings answered questions about their respective best friend's smoking status. Using SEM analyses, results indicated that older sibling smoking significantly predicted younger sibling smoking, but younger sibling smoking failed to predict older sibling smoking.

An association between sibling smoking and smoking behaviour among adolescents has been found in other studies (O'Loughlin et al., 1998; Slomkowski, Rende, Novak, Lloyd-Richardson, & Niaura, 2005; Vink et al., 2003). Some studies have also shown that sibling smoking appears to be more strongly associated with smoking behaviour than parental smoking (cf. Vink et al., 2003), but this may in part depend on the age of the child.

Peer Smoking

Peer smoking has been identified as a risk factor for smoking during adolescence. O'Loughlin and colleagues (1998) studied the predictors of two smoking transitions (i.e., from never smoking to ever smoking and from ever smoking to continued smoking) in 4th and 5th grade children (aged 9 to 12 years) during a one-year longitudinal study. Children (N = 1824) provided their smoking status (i.e., never-smoker vs. ever-smoker), as well as that of their parents, siblings, and peers. Additionally, parents provided self-report about their own smoking status and that of their children. Predictors of the transition from never smoking to ever smoking in children included peer smoking (OR = 2.3), parental smoking (OR = 2.2), and sibling smoking (OR = 1.9). Consistent with other studies, these findings provide support for social smoke exposure, via parents, siblings, and peers, as an important predictor of smoking risk.

Vink and colleagues (2003) studied the effects of parental, sibling, and peer smoking on current smoking during two periods of adolescence (12-15 years and 16-20 years) and adulthood (21-40 years). This longitudinal study comprised five measurement points from 1991 to 2000; however, new participants were added at each measurement point. Participants (N = 7196) were monozygotic and dizygotic twins who answered questions about their smoking status (i.e., current smoking vs. non-smoking) and the smoking status of their parents, co-twin, siblings, and peers. Peer smoking represented a greater risk for current smoking than sibling smoking (excluding co-twin smoking). In turn, sibling smoking represented a greater risk for current smoking than parental smoking. As an example, risk ratios for current smoking in males in the 12-15 year-old group were 3.06 for smoking by both parents; 5.23 for older brother smoking; and 7.53 for peer smoking. Although the risk

associated with peer smoking was higher than parental and sibling smoking, the risk associated with co-twin smoking was similar to the risk associated with peer smoking. Interestingly, risk ratios for current smoking in 12-15 year-olds were 25.60 if the co-monozygotic twin smoked, but only 6.13 if the co-dizygotic twin smoked; this later risk ratio (RR) is similar to the risk associated with peer smoking (RR = 16.65). These results lend some support for a potential genetic role in smoking behaviour. Recently, researchers have isolated genes associated with nicotine metabolism (see further discussion below).

Taken together, social smoke exposure via parental, sibling, and peer smoking is a strong predictor of smoking risk or smoking behaviour among children and adolescents. Cross-sectional studies have found an association of peer (Alexander, Piazza, Mekos, & Valente, 2001), schoolmate (Leatherdale, Cameron, Brown, Jolin, & Kroeker, 2006), and teacher smoking (Poulsen et al., 2002) with smoking behaviour in youth. Longitudinal studies have shown that parental (Otten et al., 2007; Peterson et al., 2006), sibling (Harakeh et al., 2007; Rajan et al., 2003; Vink et al., 2003), peer (Distefan et al., 1998; O'Loughlin et al., 1998), and schoolmate smoking (Bricker, Anderson, Rajan, Sarason, & Peterson, 2007) at baseline significantly predict prospective smoking behaviour in youth. Some studies have found that peer smoking is a stronger predictor than parental smoking (Distefan et al., 1998; O'Loughlin, 1998), whereas other studies have found that parental and peer smoking are similar predictors (Bricker et al., 2006) or influence youth smoking differentially based on age (Vitaro et al., 2004). These discrepant findings may be partly explained by differences in the outcome variables (e.g., ever smoking vs. daily smoking), different lengths of longitudinal studies (e.g., nine years vs. four years), different cultures (English-speaking vs. French-speaking), or age of children. Despite these

differences, these studies suggest that youth who are exposed to a greater number of smokers in their social environment are more likely to become smokers or to progress to higher levels of smoking.

While many studies have demonstrated that greater social smoke exposure to parents, peer, and sibling smoking is associated with greater risk for smoking initiation, few have considered that social smoke exposure is also confounded by pharmacological exposure. In other words, social smoke exposure is largely associated with greater exposure to second-hand smoke and the physiological effects of nicotine. Thus, it is of importance for researchers to measure both social and pharmacological exposure to smoke to consider the unique contributions of each on smoking initiation. Based on the findings that never-smokers may absorb as much nicotine as smokers (Al-Delaimy et al., 2001) and nicotine is associated with neuronal sensitization (DiFranza & Wellman, 2005), one might argue it is pertinent to measure pharmacological smoke exposure when investigating smoking behaviour. Because pharmacological exposure has not been measured in the studies reviewed above, it is impossible to determine if the relationships are explained entirely by social smoke exposure alone or by a combination of social and pharmacological smoke exposure.

Second-Hand Smoke Exposure and Smoking Behaviour

Considerable research has examined the relationship between exposure to second-hand tobacco smoke and childhood health problems, including asthma, bronchitis, otitis media, or sudden infant death syndrome (cf. USDHHS, 2006). However, there is a paucity of studies that have investigated the relationship between second-hand smoke exposure and smoking behaviour.

Andreeva, Krasovsky, and Semenova (2007) studied the risk factors for smoking initiation in adolescents and young adults aged 15 to 29 years (N = 609).

Participants answered questions about their age of initiation when they smoked their first cigarette, frequency of second-hand smoke exposure, their knowledge about smoking, exposure to advertisement about tobacco, and smoking restriction at home. Males were more likely to initiate smoking at a younger age if they reported no smoking restrictions at home (Cox proportional Hazard Ratios [HR] = .78) and greater exposure to second-hand smoke (HR = .84). Females were more likely to have initiated smoking at a younger age if they reported no smoking restrictions at home (HR = .39) and greater second-hand smoke exposure (HR = .79). This study provides some evidence there is a relationship between second-hand smoke exposure and smoking initiation in adolescents and young adults aged 15 to 29 years. The absence of home smoking restrictions, which may indirectly imply greater second-hand smoke exposure, is also related to smoking initiation. However, no pharmacological measurement of second-hand smoke exposure was collected. Hence, the association may be the unique consequence of social smoke exposure.

As part of the 2002 Youth Lifestyle Study, Darling and Reeder (2003) examined the link between second-hand smoke exposure at home and the prevalence rates of daily smoking in adolescents. Participants were 3,434 high school students (M_{age} = 15.0 years; 51.7% boys; 12.7% daily smokers). There was evidence of a dose-response relationship between the number of days of second-hand smoke exposure and daily smoking in adolescents: no exposure (OR = 1, *n.s.*); 1 to 2 days (OR = 3.02, p = 0.001); 3-4 days (OR = 3.95, p = 0.001); 5-6 days (OR = 4.77, p = 0.001), and 7 days (OR = 6.71, p = 0.001). Greater exposure to second-hand smoke at home was more strongly related to the likelihood of becoming a daily smoker during adolescence. However, this study only measured second-hand smoke exposure as the number of days where adolescents were exposed; they did not measure how many

smokers or which family members smoked inside the house, nor did they collect any biomarker. It is therefore not possible to determine whether social, pharmacological, or both types of smoke exposure contribute to the dose-response relationship observed in this study.

Okoli, Browning, Rayens, and Hahn (2008) investigated the relationship between exposure to second-hand smoke, nicotine dependence, as well as intentions and attempts to cease smoking among 822 adult participants who responded affirmatively to "Do you currently smoke cigarettes, even just once in a while?". Participants answered questions about the number of cigarettes they smoke daily, their age when they smoked their first whole cigarette, and the duration of their last quit attempt. Nicotine dependence was measured with the Fagerstrom Tolerance Questionnaire (FTQ: Fagerstrom & Schneider, 1989). Participants were asked if they were exposed to second-hand smoke in their home and car; the number of sources of exposure (i.e., none, one, or both) represented the measure for second-hand smoke exposure. Attempts and intentions to cease smoking were also measured. When compared to participants with no exposure, participants with two sources (OR = .51, $p < .008$) or one source (OR = .69, *n.s.*) of exposure to second-hand smoke were less likely to intend to cease smoking. Likewise, participants with two (OR = .49, $p < .001$) and one source of exposure (OR = .55, $p < .017$) were less likely to have succeeded in remaining abstinent for 24 hours than those with no exposure to second-hand smoke. A higher number of sources of exposure was associated with higher scores on the FTQ ($r = .26$, $p < .0001$). Even after controlling for daily smoking frequency, age of initiation, and the duration of the last quit attempt, a higher number of sources of exposure continued to be associated with higher scores on the FTQ ($\beta = .11$; Adjusted $R^2 = 0.46$, $p < .0001$). This result suggests second-hand smoke

exposure among active smokers was associated with greater difficulty to cease smoking and a higher likelihood of scoring high on a measure of nicotine dependence, over and above one's own smoking frequency. Limitations of this study include the lack of an objective measure of second-hand smoke exposure. Thus, the results described above could be explained solely in terms of greater social smoke exposure.

Okoli, Rayens, and Hahn (2007) next examined the effects of second-hand smoke exposure on current smoking and non-smoking adults working in bars and restaurants. Participants (N = 105) answered questions about their cigarette use and whether they were exposed to second-hand smoke in different places, such as work, home, vehicle, or elsewhere. They also reported symptoms they experience when they are in an environment with tobacco smoke. To do so, participants used a list of eight physical sensations related to second-hand smoke exposure (e.g., difficulty concentrating, difficulty sleeping, anxiety feelings, etc.). These symptoms were largely derived from the DSM-IV withdrawal syndrome for nicotine (APA, 1994). Second-hand smoke exposure was derived from nicotine assays of hair samples. The number of physical sensations reported by non-smokers was not significantly different from the number reported by smokers (χ^2 = .04, *n.s.*). Importantly, non-smokers with higher hair nicotine values were 2.2 times more likely to endorse four or more symptoms than non-smokers with lower hair nicotine values (p < .019). The number of sources of second-hand smoke exposure did not correlate with the number of physical sensations endorsed by non-smokers. By using a biomarker in addition to self-report, this study demonstrated second-hand smoke exposure was associated with physical sensations in non-smokers. In fact, non-smokers exposed to high levels of second-hand smoke exposure were as likely as smokers to report physical sensations. However, these findings must be interpreted with prudence, given that the physical

sensations were physical symptoms of second-hand smoke exposure even though they are also DSM withdrawal symptoms. The strength of this study pertains to the simultaneous use of self-report and an objective measurement of second-hand smoke exposure.

Becklake and colleagues (2005) conducted a longitudinal study to assess exposure to second-hand smoke in a sample of children. They evaluated the childhood predictors of eventual smoking uptake. Four years later children completed a self-report questionnaire about their health and smoking behaviour. Results indicated that 84 of these 191 children (44.0%) became smokers by the time of the second data collection. That is, they responded affirmatively to the question "Have you ever smoked as much as one cigarette a week for a month?". Cotinine significantly predicted smoking uptake four years after the first data collection in both pre- and post-pubertal participants. Importantly, this finding remained significant after controlling for potential covariates, such as gender, number of siblings, socioeconomic status, the number of smokers at home, and the number of cigarettes smoked inside the household (pre-pubertal: OR = 2.1, p = .052; post-pubertal: OR = 1.9, p = .007). Impressively, exposure to second-hand smoke, measured objectively in non-smoking children, predicted smoking uptake during adolescence over and above that of other robust social predictors, such as the number of smokers in the household. This is the first longitudinal study to show exposure to second-hand smoke in non-smoking children is a risk factor for smoking uptake during adolescence. However, these results need to be replicated in other longitudinal studies, especially studies with larger sample sizes.

Based on the study by Becklake and colleagues (2005), Anthonisen and Murray (2005) suggested a physiological pathway may exist between childhood

exposure to nicotine and smoking during adolescence. The latter authors suggested smoking uptake in adolescence cannot be completely explained by social factors, given that cotinine is not a social measure, but rather a pharmacological measure of second-hand smoke exposure.

Most recently, Bélanger and colleagues (2008) evaluated the relationship between exposure to second-hand smoke in never smoking 5th graders (N = 1843) and perceived nicotine dependence. Perceived nicotine dependence was measured with items derived from the Nicotine Dependence Scale for Adolescents (NDSA; Nonnemaker et al., 2004), the Hooked on Nicotine Checklist (HONK; Wheeler, Fletcher, Wellman, & DiFranza, 2004) and the ICD-10 criteria (World Health Organization [WHO], 1994). Participants provided the number of parents, siblings, and peers who smoked, and answered questions about smoking susceptibility. Exposure to second-hand smoke was based on the number of smokers in the house and the number of days spent with a smoker in a car. Results indicated 4.6% of never-smoking participants endorsed at least one symptom of nicotine dependence. Participants with greater exposure to smoke in a car were 1.2 time more likely to endorse at least one nicotine dependence symptom after controlling for sibling and peer smoking, and smoking susceptibility. One major limitation with this study is that second-hand smoke exposure was not measured with a biomarker; thus, an objective pharmacological measure was not used. The relationship between second-hand smoke exposure in a car and perceived nicotine dependence may be the result of social modeling. Importantly, statistically controlling for social exposure via peer and sibling smoking was a *first* step in trying to document a possible physiological pathway. Future studies should replicate this finding using biomarkers to better understand why never-smokers exposed to second-hand smoke perceive and endorse

nicotine dependence symptoms which are, by definition, restricted to smokers (APA, 1994). Controlling for social smoke exposure and assessing pharmacological smoke exposure with a biomarker would be the *next* step in trying to convincingly examine whether pharmacological exposure uniquely contributes to smoking initiation risk among never-smokers.

Taken together, the studies reviewed in this section lend preliminary support to the hypothesis that there is a relationship between second-hand smoke exposure and smoking behaviour in youth (Andreeva et al., 2007; Becklake et al., 2005; Bélanger et al., 2008; Darling et al., 2003) and adults (Andreeva et al., 2007; Okoli et al., 2007). This relationship has been observed for smoking initiation (Andreeva et al., 2007; Becklake et al., 2005), daily smoking (Darling et al., 2003), and smoking cessation and nicotine dependence (Okoli et al., 2008). The study by Becklake and colleagues (2005) showed pharmacological smoke exposure in never-smokers uniquely predicted prospective smoking in youth. Except for the studies by Becklake and colleagues (2005) and Okoli and colleagues (2007), the majority of the studies relied on self-report as a measure of second-hand smoke exposure and did not use a biomarker. Nonetheless, these studies suggest there is a link between second-hand smoke exposure and smoking behaviour and prompt researchers to further investigate this association. In order to evaluate the unique contribution of pharmacological smoke exposure to smoking behaviour, it is imperative to use a strategy similar to that of Becklake and colleagues (2005), by having a biomarker of smoke exposure and statistically controlling for social exposure. While longitudinal studies which examine smoking initiation prospectively are ideal, even cross-sectional studies examining smoking risk factors, such as smoking expectancies and susceptibility,

would provide important new knowledge about the differential roles of pharmacological and social smoke exposure.

Summary

Past research has largely emphasized a model of social smoke exposure to explain smoking initiation in youth. Namely, social smoke exposure models consider that having more exposure to individuals that smoke in one's own social environment increases risk for smoking. As such, research has shown an association between social smoke exposure and smoking uptake (e.g., Avenoveli et al., 2003; Kobus, 2003). Other studies have found that social smoke exposure was associated with precursors to smoking behaviours, or smoking risk factors such as endorsement of smoking expectancies (e.g., Lewis-Esquirre et al., 2005), perceived nicotine dependence (e.g., Bélanger et al., 2008), and smoking susceptibility (e.g., Leatherdale et al., 2006). Like social smoke exposure, smoking expectancies (Hine et al., 2007) and smoking susceptibility (Pierce et al., 1996) have been found to be associated with eventual smoking status; thus, these two latter measures represent a risk for smoking among never-smokers. As an example, smoking expectancies have been found to partially mediate the relationship between social smoke exposure and smoking status (Hine, McKenzie-Richer, Lewko, Tilleczek, & Perreault, 2002).

Although a higher number of smokers within one's social environment is evidently linked to greater social smoke exposure, it is also associated with greater pharmacological exposure. Given that exposure to second-hand smoke is associated with nicotine absorption (Iwase et al., 1991); that nicotine levels in never-smokers exposed to smoke can be similar to those of smokers (Dimich-Ward et al., 1997); that children exposed to second-hand smoke have higher levels of absorbed nicotine than similarly exposed nicotine than adults (Willers et al., 1995); and that nicotine has

been found to be associated with sensitization among experimental smokers (DiFranza & Wellman, 2005), it is plausible that pharmacological smoke exposure may confer risk for smoking among never-smokers. As an example, Okoli and colleagues (2007) have shown that increased pharmacological smoke exposure was associated with greater endorsement of DSM withdrawal symptoms among non-smokers.

The Present Study

The goal of the present study was to evaluate whether pharmacological and social smoke exposure are differential predictors of smoking risk in youth. Specifically, the overarching aim was to statistically tease apart the unique contributions of both pharmacological and social smoke exposure. Because social smoke exposure is confounded by pharmacological exposure, we believed that controlling for social smoke exposure would partly enable us to measure the unique contribution of pharmacological exposure. The aim of the current study was to conduct a *preliminary* test of the research question that pharmacological smoke exposure directly influences smoking risk in never-smokers, after controlling for social smoke exposure. Preliminary support for this hypothesis came from the longitudinal study by Becklake and colleagues (2005) which found that cotinine measured in never-smoking children uniquely predicted smoking uptake four years later during adolescence. Based on this finding, Anthonisen and Murray (2005) suggested a physiological pathway may exist between pharmacological smoke exposure and smoking behaviour. Bélanger and colleagues (2008) found that never smoking youth reporting greater second-hand smoke exposure also reported greater perceived nicotine dependence; however, no objective measure of pharmacological smoke exposure was collected. Extending the work of DiFranza and Wellman (2005)

about neural sensitization in experimental smokers, one might postulate sensitization occurs among never-smokers exposed to nicotine via second-hand smoke exposure. Taken together, this current study sought to simultaneously evaluate pharmacological and social smoke exposure in order to tease apart their unique effects on smoking risk factors among never-smoking youth in Québec.

Given this objective, three specific hypotheses were tested. First, it was hypothesized that greater pharmacological exposure (cotinine) would predict greater smoking risk factors (expectancies, smoking susceptibility, perceived nicotine dependence). Second, it was hypothesized that greater social smoke exposure (parent, sibling, peer, school) would predict greater smoking risk factors. Third, it was hypothesized that both greater pharmacological exposure and greater social exposure would each uniquely contribute to the model to predict smoking risk factors in youth.

Method

Participants

Participants were 655 students (53.2% female; M_{age} = 12.4 years; SD_{age} = .6 year) in 6th or 7th grade (50.4% in 6th grade) attending a public school. This study was conducted exclusively in French-speaking schools because the prevalence of smoking is usually 5 to 10% higher among French-speaking Canadians than English-speaking Canadians (Wharry, 1997). Thus, youth in Québec are more likely to be exposed to second-hand smoke than their counterparts in other Canadian provinces. Based on the 2004-05 Youth Smoking Survey (YSS; Health Canada, 2007b), students in 6th and 7th grades were selected because there is a lower probability they ever tried smoking (6th grade = 17.1%; 7th grade = 36.2%) compared to older students (8th grade = 45.6%; 9th grade = 48.9%), which facilitated recruitment of never-smokers, our target sample.

Primary and secondary schools were recruited with convenience sampling within these four school boards: 1) *Commission scolaire Marie-Victorin* (four schools; n = 365); 2) *Commission scolaire de Laval* (six schools; n = 221); 3) *Commission scolaire des Hautes-Rivières* (one school; n = 44); and 4) *Commission scolaire des Samares* (one school; n = 25). These four school boards were selected because of their participation in the larger AdoQuest study and because their principals agreed to participate. AdoQuest is a longitudinal, cohort-design study which originated in 2005 with 29 Montréal schools with 1800 schoolchildren aged 10 to 12 years old. AdoQuest investigates smoking trajectories in youth and is a multi-site collaboration, including McGill University, Concordia University, Université de Montréal, Centre de recherche du Centre hospitalier de l'Université de Montréal, and Institut national de santé publique du Québec. Only AdoQuest smoking-related measures which are relevant to the present study are described below. AdoQuest and

the present study have been approved by the ethical review committee of Concordia University (UH2006-063-1).

Procedure

Data collection was conducted in two cycles. The first cycle took place in Spring 2007 with 6th graders (N = 278) in seven primary schools: *École primaire d'Iberville*, *École primaire Notre-Dame-de-Lourdes*, *École primaire Saint-Paul*, *École primaire Saint-Louis*, *École primaire Marcelle-Gauvreau*, *École primaire Cœur-Soleil*, and *École primaire l'Orée-des-bois*. The goal of the first cycle was to validate the French-Canadian version of the SESA to determine if its factor structure replicated that of the original. The second cycle took place in Fall 2007 and Winter 2008 with 6th and 7th graders (N = 377) in two elementary schools and three secondary schools: *École primaire des Mille-Fleurs*, *École primaire La Source*, *École secondaire Poly-Jeunesse*, *École secondaire Antoine-Brossard*, and *École secondaire André-Laurendeau*. The goal of the second cycle was to collect data to test the research hypotheses. During the second cycle, additional questions were added to more precisely measure social smoke exposure (e.g., parental smoking, peer smoking, sibling smoking, etc.) and smoking risk factors (e.g., smoking susceptibility). With the exception of the additional questions in the second cycle, the method for data collection was identical in both cycles.

School principals and teachers from targeted school boards were contacted to obtain their permission to collect data in their classrooms during class time, lasting between 60 and 75 minutes. Consent forms and permission slips were sent home to parents or legal guardians with the schoolchildren. Students who returned signed consent forms and who provided assent were allowed to participate in the study.

On the day of data collection in the classroom, researchers provided standardized instructions and reminded participants that their results would be kept confidential so as to maximize honest responding. Participants were asked to complete questionnaires in silence and on their own. Students who did not receive consent or did not provide assent were asked to do individual work in silence.

As participants were completing the questionnaires, research assistants collected saliva and breath carbon monoxide samples. Each participant provided one saliva sample to measure their level of salivary cotinine as a measure of nicotine intake via second-hand smoke exposure. Saliva samples were obtained by inserting a cotton swab (Salimetrics Oral Swab) under the tongue of each participant for a two-minute period. Participants were then asked to put the cotton swab into a swab storage tube with their teeth and tongue so as to avoid any contamination of the sample from their fingers. The participants' swab storage tubes were stored in a freezer in the Pediatric Public Health Psychology Laboratory prior to being shipped to Salimetrics Laboratories for assaying. Participants provided a breath sample to measure their level of expired carbon monoxide as a measure of secondhand smoke exposure. Participants were asked to hold their breath for 15 seconds and to slowly blow into the monitor, through a cardboard mouthpiece, until their lungs were completely empty of air. A new disposable cardboard mouthpiece was used for each participant for hygienic purposes. All of these measures were taken individually within a timeframe of 60 to 75 minutes while the remaining participants were completing the questionnaires.

Measures

Smoking Status

Smoking status was divided into two categories: never-smokers and ever-smokers. Smoking status was determined with the item: *Have you smoked a whole cigarette in the last 6 months?* Based on the criteria used by the 2006 *Enquête québécoise sur le tabac, l'alcool, la drogue et le jeu chez les élèves du secondaire* (Dubé & Camirand, 2007), participants who answered *no* were classified as never-smokers; participants who answered *yes* were classified as ever-smokers.

Social Smoke Exposure

Number of Smokers Inside of Household. Number of smokers inside the household was measured with one item derived from the YSS: *Excluding yourself, how many people smoke inside your home every day or almost every day? Do not count those who smoke outside.* Possible scores ranged from 0 smokers to 5 or more smokers. Given that the items were derived from the YSS, our results could be compared to normative data in Canada.

Number of days spent with a smoker in a car. Number of days spent with at least one smoker in a car was measured with one item derived from the YSS: *During the past 7 days, on how many days did you ride in a car with someone who was smoking cigarettes?* Possible scores ranged from 0 day to all 7 days.

Number of Smokers among Parents, Siblings, and Peers. Number of smokers among parents was defined as the number of parents who currently smoked. Two items were derived from the YSS: *Does your father (mother), or the person who is like your father (mother), smoke cigarettes?* Possible scores ranged from 2 (both parents smoke now) to 0 (neither parent smokes). Similarly, sibling smoking was measured using two items derived from the YSS: *Do any of your sisters (brothers)*

smoke cigarettes? Possible scores ranged from 2 (at least one brother and at least one sister smoke now) to 0 (no sibling smokes). Peer smoking was measured with one item derived from the YSS: *Your closest friends are the friends you like to spend the most time with. How many of your closest friends smoke cigarettes?* Possible scores ranged from 0 (no friend smokes) to 5 (five or more friends smoke).

Number of Situations of Smoke Exposure. Participants endorsed situations in which their parents smoked cigarettes (e.g., *When they drink a coffee*, *When they have guests*, *When they watch TV*, etc.). The total number of parental smoking situations ranged from 0 to 30. Likewise, participants endorsed situations in which their siblings smoked cigarettes (e.g., *When they eat breakfast*, *When they come back from school or work*, *When they do their homework*, etc.). The total number of sibling smoking situations ranged from 0 to 30. Participants also endorsed situations in which their peers smoked cigarettes (e.g., *Before classes*, *During recess*, *During lunch hour*, etc.). The total number of peer smoking situations ranged from 0 to 27. This new measure (i.e., number of situations of smoke exposure) was created for the present study to more precisely evaluate social smoke exposure.

Number of Cigarettes Smoked by Parents, Siblings, and Peers. Using an open-ended question, participants estimated the number of cigarettes their parents, siblings, and peers smoked daily. Other researchers (e.g., Hine et al., 2002) have successfully used categorical response options to measure parental smoking frequency (e.g., No cigarette; 1 to 7 cigarettes per day; Half a pack per day; One pack per day; One and a half pack per day; Two or more pack per day).

School Smoking. School smoking was measured with schoolmate and teacher smoking. Schoolmate smoking was measured with one item derived from the YSS: *Choose the answer that best describes what you think. I see students smoking near*

my school. Participants used a five-point scale ranging from *never* to *very often.* Similarly, teacher smoking was measured with one item derived from the YSS: *Choose the answer that best describes what you think. I see teachers or staff members of the school smoking near the school.* Participants used a five-point scale ranging from *never* to *very often.*

Smoking Expectancies. Using the French-Canadian version (Racicot and colleagues, 2008) of the SESA (Hine and colleagues, 2007), smoking expectancies were categorized into expected benefits and expected costs. These two categories were higher order factors that were replicated in the validation study of the French-Canadian version. Using a ten-point Likert scale (i.e., 0 = completely unlikely, 9 = completely likely), participants rated the likelihood of each item. There were 21 items measuring expected costs and 22 items measuring expected benefits. The scores for expected costs and expected benefits were calculated as the average of the items for each category. The original version of the SESA has good internal consistency, with Cronbach alphas ranging from .88 to .89 for expected benefits and .86 for expected costs (Hine et al., 2007). Like the original version, the French-Canadian version of the SESA showed good internal consistency with Cronbach's alphas of .94 for expected costs and .92 for expected benefits (Racicot and colleagues, 2008).

Perceived Nicotine Dependence. Given that studies have shown never-smokers do endorse nicotine dependence symptoms (Bélanger et al., 2008; Okoli et al., 2007), perceived nicotine dependence among never-smokers was measured with the Nicotine Dependence Scale for Adolescents (NDSA; Nonnemaker et al., 2004). The NDSA is a reliable instrument (Cronbach's α = .81) that correlates with daily cigarette consumption (r = .61, p < .01) and duration of quit attempts (r = -.22, p <

.01). The NDSA consists of six items: 1) *How soon after you wake up do you usually smoke your first cigarette on a weekday (Monday through Friday)?*; 2) *How soon after you wake up do you usually smoke your first cigarette during the weekend (Saturday and Sunday)?*; 3) *If you are sick with a bad cold or sore throat, do you smoke cigarettes?*; 4) *How true is this statement for you? When I go without a smoke for a few hours, I experience cravings*; 5) *How true is this statement for you? I sometimes have strong cravings for cigarettes where it feels like I am in the grip of a force that I cannot control*; and 6) *Do you think you would be able to quit smoking cigarettes if you wanted to?* Each item is answered on a Likert scale (ranging from 3-6 points). For every item, there is the response option "I do not smoke". A composite score, created by summing items, ranges from 0 (no perception of nicotine dependence) to 16 (high perception of nicotine dependence).

Pharmacological Smoke Exposure

Salivary Cotinine. Pharmacological exposure to nicotine via second-hand smoke was measured with cotinine. Saliva samples were assayed by Salimetrics Laboratories (Salimetrics, LLC, State College, PA, USA) for cotinine assays completed in duplicate. All samples were tested with a high sensitivity enzyme immuno-assay which has a minimal detection level of 0.05ng/mL; 20ul of saliva sample were used for each determination. The average of the two assays was used. Mean intra-assay coefficient of variation was 4.1% and mean inter-assay coefficient of variation was 6.6% (Salimetrics, 2007). Values below the minimal detection level were set to 0.04 ng/mL.

Breath Carbon Monoxide. Pharmacological exposure to second-hand smoke was also measured by a breath carbon monoxide (CO) sample. CO is a toxic gas that competes with and replaces oxygen in blood, which results in carboxyhemoglobin

(COHb). The Bedfont Scientific's Micro 4 Smokerlyzer (Bedfont, 2006) is a hand-held CO monitor that instantly provides a 2-line digital alphanumeric readout of expired CO in parts per million (ppm) and the percentage of COHb (%COHb).

Smoking Susceptibility. Smoking susceptibility was defined as a lack of a firm commitment to not smoke among never-smokers (Pierce et al., 1996). This definition has been used in other studies (e.g., Leatherdale et al., 2005, 2006). Smoking susceptibility was measured using five items derived from the YSS: 1) *Have you ever been curious about smoking a cigarette?*; 2) *Have you ever seriously thought about trying cigarette smoking?*; 3) *Do you think in the future you might try smoking cigarettes?*; 4) *If one of your best friends was to offer you a cigarette would you smoke it*?; and 5) *At anytime during the next year do you think you will smoke a cigarette?* The total score ranged from 0 (i.e., non-susceptibility) to 11 (i.e., high susceptibility to smoking).

Data Analysis

Given the aim of the present study, analyses were restricted to never-smoking youth. Of the 377 participants recruited for the second cycle, 338 answered "no" to the question *Have you smoked a whole cigarette in the last 6 months?* and thus, were retained for data analyses. Participants classified as ever-smokers (i.e., answering "yes" to the above question) were not included. All variables were inspected for normality. Cotinine was not normally distributed and was log transformed. Studies collecting biomarkers typically use statistical transformations (e.g., Becklake et al., 2005; Okoli et al., 2007). Missing values on each variable were replaced with the series mean.

Structural equation modeling (SEM) was used to test the research hypotheses as this statistical technique permits the measurement of relationships between latent

variables. Informed by previous work, this study comprised three latent factors: pharmacological exposure (cotinine), social smoke exposure (parent, sibling, peer, school), and smoking risk factors (expectancies, susceptibility, perceived nicotine dependence). Pearson and Spearman correlations were initially conducted to examine the associations between the measured manifest variables, to help guide decisions regarding development of the measurement models. (The manifest variables selected to make up the three latent factors are further described in the results.) The following indices were used to determine the goodness-of-fit of the models: 1) Goodness-of-Fit Index (GFI > .90) 2) Comparative Fit Index (CFI > .90), and 3) Root Mean Square Error of Approximation (RMSEA < .05 to .08; Bentler, 1990). Following identification of measurement models with appropriate fit indices, the structural models were tested in accordance with our three hypotheses. Exploratory post-hoc analyses were also conducted.

Results

Participant Characteristics

There were 377 students who participated in the *Cycle 2* data collection (see Tables 2-5). Mean age of the participants was 12.68 years (*SD* = 0.67). The majority of participants attended 7th grade (84.6%); the remaining attended 6th grade (15.4%). The participants included 181 girls (53.5%) and 152 boys (45.0%); five students did not report their sex (1.5%). Ninety percent (n = 338, 89.66%) of the participants were classified as never-smokers based on the criteria used by the *Institut de la statistique du Québec* (Dubé & Camirand, 2007). This percentage is consistent, but slightly higher than that found in the YSS (81.5%; Health Canada, 2008), which may be attributable to our younger sample.

The majority of participants reported that neither parent smoked (66.9%), compared to one parent who smoked (21.0%) or both parents (11.5%; see Table 6). The average number of situations in which parents smoked was 7.15 (*SD* = 5.55). The three most commonly endorsed situations in which parents smoked included smoking in a car (64.1%), smoking when coming back from work (64.1%), and smoking outside of the household (56.3%). The average number of cigarettes smoked daily by parents was 4.83 (*SD* = 6.18).

The majority of participants reported having no siblings who smoked (90.8%), compared to having at least one sister or brother who smoked (6.8%) or at least two siblings who smoked (1.5%; see Table 7). The average number of situations in which siblings smoked was 4.88 (*SD* = 5.80). The three most commonly endorsed situations in which siblings smoked included smoking outside the home (61.5%), smoking in a car (38.5%), and smoking when coming back from school or work (34.6%). The average number of cigarettes smoked daily by siblings was 3.63 (*SD* = 3.25).

Table 2

School Characteristics

Variable	Cycle 1 n (%)	Cycle 2 n (%)
Number of Participants	261	338
Gender		
Male	127 (48.7)	152 (44.97)
Female	134 (51.3)	181 (53.55)
Unspecified	0 (0.00)	5 (1.48)
School		
École des Mille-Fleurs	-	12 (3.6)
École La Source	-	40 (11.8)
École Poly-Jeunesse	-	49 (14.5)
École Antoine-Brossard	-	21 (6.2)
École André-Laurendeau	-	216 (63.9)
École Saint-Louis	22 (8.4)	-
École Notre-Dame-de-Lourdes	40 (15.3)	-
École Saint-Paul	29 (11.1)	-
École d'Iberville	78 (29.9)	-
École Marcelle-Gauvreau	36 (12.6)	-
École Cœur-Soleil	33 (13.8)	-
École L'Orée des Bois	23 (8.8)	-
Grade		
Sixth	261 (100.0)	52 (15.4)
Seventh	0 (0.0)	286 (84.6)

Note. Dashes indicate there is no value for the variable on a specific cycle.

Table 3

Participant Characteristics

Variables	Cycle 1 *M* (*SD*)	Cycle 2 *M* (*SD*)
Age (Years)	12.18 (.40)	12.68 (.67)
Number of Situations of Smoke Exposure Via		
Parents	-	7.15 (5.55)
Siblings	-	4.88 (5.80)
Peers	-	1.09 (1.15)
Estimated Number of Cigarettes Smoked Daily by		
Parents	-	4.83 (6.18)
Siblings	-	3.63 (3.25)
Peers	-	.99 (1.53)
Cotinine Value (ng/mL)	.56 (1.69)	.90 (2.12)
Carbon Monoxide (ppm)	.31 (.75)	.11 (.70)
Carboxyhemoglobin (%COHb)	.14 (.33)	.10 (1.10)
Expected Costs (Average)	5.15 (2.32)	6.10 (2.05)
Expected Benefits (Average)	2.53 (1,78)	2.18 (1.44)
Nicotine Dependence Scale for Adolescents (Score)	.17 (1.04)	.31 (1.10)
Smoking Susceptibility (Sum)	-	1.31 (1.94)

Note. Dashes indicate there is no value for the variable on a specific cycle.

Table 4

Smoking Status

Question	Cycle 1 *n* (%)	Cycle 2 *n* (%)
Tried smoking, even a few puffs (last 6 months)		
Yes	17 (6.5)	29 (8.6)
No	244 (93.5)	308 (91.4)
Smoked whole cigarette (last 6 months)		
Yes	0 (.0)	0 (.0)
No	261 (100.0)	338 (100.0)

Table 5

Exposure to Smoke at Home and in Car

Question	Cycle 1 *n* (%)	Cycle 2 *n* (%)
People smoking inside home daily		
None	173 (67.1)	227 (67.4)
1 person	41 (15.9)	54 (16.0)
2 people	33 (12.8)	45 (13.4)
3 people	7 (2.7)	7 (2.1)
4 people	1 (.4)	2 (.6)
5 or more people	3 (1.2)	2 (.6)
Number of days where exposure took place in car (past 7 days)		
0 days	181 (69.6)	243 (72.1)
1 or 2 days	49 (18.8)	51 (15.1)
3 or 4 days	14 (5.4)	23 (6.8)
5 or 6 days	6 (2.3)	9 (2.7)
All 7 days	10 (3.8)	11 (3.3)

Table 6

Parental Smoking

Question	Cycle 2 *n* (%)
Does father smoke cigarettes?	
I do not live with a father	6 (1.8)
He has never smoked	137 (40.9)
He used to smoke	92 (27.5)
He smokes now	77 (23.0)
I do not know	23 (6.9)
Does mother smoke cigarettes?	
I do not live with a mother	1 (.3)
She has never smoked	177 (52.7)
She used to smoke	71 (21.1)
She smokes now	72 (21.4)
I do not know	15 (4.5)
Number of parents who smoke	
Neither of my parents smoke now	226 (66.9)
One of my parents smokes now	71 (21.0)
Both of my parents smoke now	39 (11.5)

Note. This variable was only measured in cycle 2.

Table 7

Sibling Smoking

Question	Cycle 2 *n* (%)
Do sisters smoke cigarettes?	
I do not have any sisters	165 (49.3)
None of my sisters smoke	143 (42.7)
At least 1 of my sisters used to smoke	3 (.9)
At least 1 of my sisters smokes now	17 (5.1)
I do not know	7 (2.1)
Do brothers smoke cigarettes?	
I do not have any brothers	138 (41.2)
None of my brothers smoke	159 (47.5)
At least 1 of my brothers used to smoke	5 (1.5)
At least 1 of my brothers smokes now	16 (4.8)
I do not know	17 (5.1)
Number of siblings who smoke	
None of my siblings smokes now	307 (90.8)
At least one sister OR at least one brother smokes now	23 (6.8)
At least one sister AND at least one brother smokes now	5 (1.5)

Note. This variable was only measured in cycle 2.

The majority of participants reported none of their friends smoked (77.1%), compared to one friend who smoked (9.5%), two friends who smoked (6.3%), three friends who smoked (2.7%), four friends who smoked (1.5%) or five or more friends who smoked (3.0%; see Table 8). The average number of situations in which peers smoked was 1.09 (*SD* = 1.15). The three most commonly endorsed situations in which peers smoked included smoking during recess at school (55.6%), smoking after school (47.2%), and smoking before classes begin (30.6%). The average number of cigarettes smoked daily by peers was .99 (*SD* = 1.53). Participants endorsed they observed schoolmate smoking at school never (11.7%), rarely (6.3%), sometimes (15.6%), often (25.2%), and very often (41.1%). Similarly, participants also endorsed that they observed teachers smoking around school grounds never (25.2%), rarely (26.7%), sometimes (28.8%), often (12.1%), and very often (7.3%).

Of the 338 participants, one participant did not provide enough saliva to complete the assay and one participant refused to provide a saliva sample. Salivary cotinine values were below the detectable limit (0.05 ng/mL) for 61 participants (18.1%). Thus, for the remaining 275 participants, mean cotinine value was .90 ng/mL (*SD* = 2.12; see Table 3). According to Salimetrics (2007), cotinine values below 15 ng/mL suggest one is not smoking and not exposed to second-hand smoke exposure; values between 15 and 50 ng/mL suggests one is not smoking, but exposed to second-hand smoke; and values above 50 ng/mL suggest one is smoking. In this study, values below the detectable limit were replaced with .04 ng/mL to provide the most conservative estimate; missing values were replaced with mean exposure. Due to non-normality, cotinine values were log transformed ($Cotinine_{(log)}$; *M* = -.68, *SD* = .62). Mean value for carbon monoxide was .11 ppm (*SD* = .70) and for

Table 8

Peer, Schoolmate, and Teacher Smoking

Question	Cycle 2 *n* (%)
How many friends smoke cigarettes?	
None	259 (77.1)
1 friend	32 (9.5)
2 friends	21 (6.3)
3 friends	9 (2.7)
4 friends	5 (1.5)
5 or more friends	10 (3.0)
I see students smoking near my school.	
Never	39 (11.7)
Rarely	21 (6.3)
Sometimes	52 (15.6)
Often	84 (25.2)
Very often	137 (41.1)
I see teachers smoke near school.	
Never	83 (25.2)
Rarely	88 (26.7)
Sometimes	95 (28.8)
Often	40 (12.1)
Very often	24 (7.3)

Note. This variable was only measured in cycle 2.

carboxyhemoglobin was .10% COHb (SD = 1.10), suggesting little or no recent second-hand smoke exposure (2-3 hours).

Mean score for expected costs was 6.10 (SD = 2.05), while mean score for expected benefits was 2.18 (SD = 1.44). Broadly, these results suggest that youth endorsed that cigarette smoking is more likely to be associated with costs and less likely to be associated with benefits. Hine and colleagues (2007) reported similar means and standard deviations (expected costs: M = 6.20, SD = 1.66; expected benefits: M = 2.66, SD = 1.80). Mean score on the Nicotine Dependence Scale for Adolescents (NDSA; see Table 9) was low (M = .31, SD = 1.10), compared to the mean score observed in the NDSA validation study (M = 6.88, SD = 4.55; Nonnemaker et al., 2004). However, the validation study included smokers, which explains the higher mean. Mean score for smoking susceptibility was low (M = 1.31, SD = 1.94; see Table 10). However, our results suggest participants (47.9%) endorsed at least one item measuring smoking susceptibility, which is comparable to the percentage (41.0%) found in Bélanger and colleagues (2008).

Preliminary Analyses

To help inform which variables would be retained for the measurement models in SEM, Pearson (r) and Spearman (r_s) bivariate correlations were computed among the manifest variables measuring parental smoking (see Table 11), sibling smoking (see Table 12), peer and school smoking (see Table 13), pharmacological smoke exposure (see Table 14), and smoking risk factors (see Table 15).

Parental Smoking. The number of parents who smoked was associated with the number of smokers inside the household (r_s = .72, p < .01), the number of days spent with a smoker in a car (r_s = .63, p < .01), the number of situations in which parents smoked (r_s = .91, p < .01), and the number of cigarettes smoked daily by

Table 9

Perceived Nicotine Dependence

Question	Cycle 1 *n* (%)	Cycle 2 *n* (%)
When I go without a smoke for a few hours, I experience cravings		
Not at all true	51 (20.2)	52 (15.8)
Not very true	2 (.8)	6 (1.8)
Fairly true	4 (1.6)	9 (2.7)
Very True	3 (1.2)	4 (1.2)
I don't smoke	193 (76.3)	259 (78.5)
I sometimes have strong cravings for cigarettes where it feels like I am in the grip of a force that I cannot control		
Not at all true	55 (21.7)	50 (15.1)
Not very true	4 (1.6)	13 (3.9)
Fairly true	4 (1.6)	6 (1.8)
Very True	1 (.4)	4 (1.2)
I don't smoke	189 (74.7)	259 (78.0)

Table 9 *(Continued)*

Question	Cycle 1 *n* (%)	Cycle 2 *n* (%)
How soon after waking up do you smoke first cigarette on a weekday ?		
I do not smoke cigarettes	253 (97.7)	318 (94.9)
Less than 15 minutes	1 (.4)	2 (.6)
15 to 30 minutes	0 (.0)	0 (.0)
More than 30 but less than 60 minutes	0 (.0)	2 (.6)
1 to 2 hours	0 (.0)	0 (.0)
More than 2 hours but less than ½ a day	0 (.0)	0 (.0)
More than ½ day	0 (.0)	0 (.0)
I do not smoke during the weekdays	5 (1.9)	13 (3.9)
How soon after waking up do you smoke your first cigarette during the weekend ?		
I do not smoke cigarettes	254 (98.1)	316 (94.3)
Less than 15 minutes	2 (.8)	5 (1.5)
15 to 30 minutes	0 (.0)	0 (.0)
More than 30 but less than 60 minutes	0 (.0)	5 (1.5)
1 to 2 hours	0 (.0)	0 (.0)
More than 2 hours but less than ½ a day	0 (.0)	0 (.0)
More than ½ a day	0 (.0)	0 (.0)
I do not smoke during the weekends	3 (1.2)	9 (2.7)

Table 9 *(continued)*

Question	Cycle 1 *n* (%)	Cycle 2 *n* (%)
If sick with bad cold or sore throat, do you smoke cigarettes?		
I do not smoke cigarettes	256 (98.8)	320 (95.5)
No, I stop smoking	2 (.8)	14 (4.2)
Yes, but I cut down	0 (.0)	1 (.3)
Yes, I smoke the same amount	1 (.4)	0 (.0)
Do you think you would be able to quit smoking cigarettes?		
I do not smoke cigarettes	246 (95.0)	308 (92.2)
Yes, without a doubt	13 (5.0)	20 (6.0)
Probably yes	0 (.0)	6 (1.8)
Probably not	0 (.0)	0 (.0)
Definitely not	0 (.0)	0 (.0)

Table 10

Smoking Susceptibility

Question	Cycle 2 *n* (%)
Ever been curious about smoking cigarette?	
Yes	69 (20.8)
No	262 (79.2)
Ever seriously thought about trying a cigarette?	
I have already tried smoking	35 (10.4)
Yes	27 (8.1)
No	272 (81.2)
I already smoke	1 (.3)
In the future will you try smoking cigarettes?	
I have already tried smoking	23 (6.9)
Definitely yes	7 (2.1)
Probably yes	31 (9.3)
Probably not	63 (18.9)
Definitely not	209 (62.8)
If your best friend was to offer a cigarette, would you smoke it?	
Definitely yes	3 (.9)
Probably yes	16 (4.8)
Probably not	52 (15.5)
Definitely not	264 (78.8)

Note. This variable was only measured in cycle 2.

Table 10 (*Continued*)

Question	Cycle 2 *n* (%)
Do you think you will smoke a cigarette within next year?	
Definitely yes	5 (1.5)
Probably yes	26 (7.8)
Probably not	42 (12.6)
Definitely not	261 (78.1)

Note. This variable was only measured in cycle 2.

Table 11

Correlations for Parental Smoking (n = 338)

Subscale	1	2	3	4	5
1. People smoking inside home daily	-	.51** (.54)**	.68** (.72)**	.63** (.71)**	.41** (.67)**
2. Number of days where exposure took place in car (past 7 days)		-	.61** (.63)**	.65** (.66)**	.37** (.63)**
3. Number of parents who smoke			-	.75** (.91)**	.51** (.88)**
4. Total number of situations for parents				-	.67** (.90)**
5. Number of cigarettes smoked daily by parents					-

Note. Coefficients on top represent Pearson correlations, while coefficients in parentheses represent Spearman correlations.

** $p < .01$, two-tailed.

Table 12

Correlation for Siblings Smoking (n = 338)

Subscale	1	2	3	4	5
1. People smoking inside home daily	-	.51** (.54)**	.41** (.23)**	.37** (.27)**	.17** (.23)**
2. Number of days where exposure took place in car (past 7 days)		-	.18** (.18)**	.22** (.17)**	.19** (.20)**
3. Number of siblings who smoke			-	.66** (.66)**	.52** (.64)**
4. Total number of situations for siblings				-	.38** (.72)**
5. Number of cigarettes smoked daily by siblings					-

Note. Coefficients on top represent Pearson correlations, while coefficients in parentheses represent Spearman correlations.

** $p < .01$, two-tailed.

Table 13

Correlation for Peer and School Smoking (n = 338)

Subscale	1	2	3	4
1. Total number of situations for peers	-	.38** (.86)**	.12* (.17)**	.07 (.04)
2. Number of cigarettes smoked daily by peers		-	.12* (.21)**	.12* (.10)
3. I see students smoking near my school.			-	.09 (.15)**
4. I see teachers smoke near school.				-

Note. Coefficients on top represent Pearson correlations, while coefficients in parentheses represent Spearman correlations.

* $p < .05$, two-tailed. ** $p < .01$, two-tailed.

Table 14

Correlation for Pharmacological Smoke Exposure (n = 338)

Subscale	1	2	3
1. Cotinine Log	-	-.08 (-.06)	-.07 (-.06)
2. Carbon monoxide (PPM)		-	.86** (1.00)**
3. Carboxyhemoglobin (%COHb)			-

Note. Coefficients on top represent Pearson correlations, while coefficients in parentheses represent Spearman correlations.

** $p < .01$, two-tailed.

Table 15

Correlation for Smoking Risk Factors (n = 338)

Subscale	1	2	3	4
Average expected costs	-	.10	-.14**	-.18**
Average expected benefits		-	.12*	.14*
NDSA score			-	.29**
Sum susceptibility				-

* $p < .05$, two-tailed. ** $p < .01$, two-tailed.

parents (r_s = .88, p < .01). The number of situations in which parents smoked was associated with the number of cigarettes smoked daily by parents (r = .67, p < .01). Because these correlation coefficients were moderate to large, all variables were retained for the latent variable parental smoking.

Sibling Smoking. The number of siblings who smoked was associated with the number of smokers inside the household (r_s = .23, p < .01), the number of days spent with a smoker in a car (r_s = .18, p < .01), the number of situations in which siblings smoked (r_s = .66, p < .01), and the number of cigarettes smoked daily by siblings (r_s = .65, p < .01). The number of situations in which siblings smoked was associated with the number of cigarettes smoked daily by siblings (r = .38, p < .01). Coefficients for the number of smokers inside the household and the number of days spent with a smoker in a car were judged too low to be retained for the latent variable sibling smoking. Thus, the number of siblings who smoked, the number of situations in which siblings smoked, and the number of cigarettes smoked daily by siblings were used for the latent variable sibling smoking.

Peer and School Smoking. The number of peers who smoked was associated with the number of situations in which peers smoked (r_s = .79, p < .01) and the number of cigarettes smoked daily by peers (r_s = .84, p < .01). The number of situations in which peers smoked was associated with the number of cigarettes smoked daily by peers (r = .38, p < .01). The number of peers who smoked was not highly correlated with observing schoolmates smoking (r_s = .15, p < .01) or observing teachers smoking (r_s = .08, *n.s.*). Observing schoolmates smoking was not highly associated with observing teachers smoking (r_s = .15, p < .01). Based on these coefficients, it was decided that peer and school smoking variables were measuring two different constructs and should not be included for a single latent variable. Thus,

two latent variables were derived: peer smoking and school smoking. The number of peers who smoked, the number of situations in which peers smoked, and the number of cigarettes smoked daily by peers were thus selected to measure peer smoking. Observing schoolmates and teachers smoking were selected to measure school smoking.

Pharmacological Smoke Exposure. $Cotinine_{(log)}$ was not significantly associated with carbon monoxide (r_s = -.06, *n.s.*) or carboxyhemoglobin (r = -.06, *n.s.*). Carbon monoxide and carboxyhemoglobin were significantly associated (r_s = 1.0, $p < .01$). Given that neither carbon monoxide nor carboxyhemoglobin were significantly associated with $cotinine_{(log)}$, only $cotinine_{(log)}$ will be used to measure pharmacological smoke exposure. This was deemed appropriate as carbon monoxide and carboxyhemoglobin are indirect derivatives of recent smoke exposure, whereas cotinine is a metabolite of nicotine exposure from second-hand smoke over the last several days.

Smoking Risk Factors. Expected costs were not significantly associated with expected benefits (r = .15, *n.s*). Small correlations were observed for expected costs with perceived nicotine dependence (r = -.14, $p < .01$) and smoking susceptibility (r = -.18, $p < .01$). As well, small correlations were observed for expected benefits with perceived nicotine dependence (r = .12, $p < .05$) and smoking susceptibility (r = .14, $p < .05$). Perceived nicotine dependence was moderately associated with smoking susceptibility (r = .29, $p < .01$). Despite these small correlations, expected costs, expected benefits, perceived nicotine dependence, and smoking susceptibility were retained for the latent variable smoking risk factors. In fact, these four variables are the primary measures typically reported in the literature as risk factors for eventual smoking uptake.

Measurement Models

Parental Smoking. A measurement model was computed for the latent variable *parental smoking.* The number of smokers inside the household (factor loading = .72), the number of days spent with a smoker in a car (factor loading = .70), the number of parents who smoked (factor loading = .84), the number of situations in which parents smoked (factor loading = .92), and the number of cigarettes smoked daily by parents (factor loading = .55) were manifest variables used to measure parental smoking. Fit indices showed this model had minimally acceptable fit (GFI = .94; CFI = .96; RMSEA = .17). In an effort to improve model fit, the number of cigarettes smoked daily by parents was removed. This was found to improve model fit (GFI = .99; CFI = .99, RMSEA = .09; factor loadings not shown) and will be used in the structural models.

Sibling Smoking. A measurement model was computed for the latent variable *sibling smoking.* The number of siblings who smoked (factor loading = .59; error variance constrained to .1), the number of situations in which siblings smoked (factor loading = .95), and the number of cigarettes smoked daily by siblings (factor loading = .42) were manifest variables used to measure sibling smoking. Fit indices for this model indicated the fit was unacceptable (GFI = .88; CFI = .72; RMSEA = .39). In an effort to improve model fit, the number of smokers inside the household was entered into the model. This was deemed to improve model fit (GFI = .99; CFI = .99; RMSEA = .08; factor loadings not shown) and will be used in the structural models.

Peer Smoking. A measurement model was computed for the latent variable *peer smoking.* The number of peers who smoked (factor loading = .81, error variance constrained to .45), the number of situations in which peers smoked (factor loading = .72), and the number of cigarettes smoked daily by peers (factor loading = .58) were

manifest variables used to measure peer smoking. Fit indices showed this model had acceptable fit (GFI = .99; CFI = .99; RMSEA = .08) and will be used in the structural models.

School Smoking. A measurement model was computed for the latent variable *school smoking*. Observing schoolmates smoking (factor loading = .70; error variance constrained to .9) and observing teachers smoking (factor loading = .31; error variance constrained to .9) were manifest variables used to measure school smoking. Fit indices indicated this model had poor fit (GFI = .92, CFI = 0.0, RMSEA = .35). Despite several attempts to improve model fit (adjusting error variances based on reported reliability estimates), this model was the best we obtained in terms of goodness-of-fit. Thus, this will be used in the structural models.

Smoking Risk Factors. A measurement model was computed for the latent variable *smoking risk factors*. Expected costs (factor loading = -.25), expected benefits (factor loading = .18), perceived nicotine dependence (factor loading = .50; error variance set to .9), and smoking susceptibility (factor loading = .52) were manifest variables used to measure smoking risk factors. Fit indices for this model showed the fit was minimally acceptable (GFI = .99; CFI = .87; RMSEA = .08) and will be used in the structural models.

Structural Models

Hypothesis 1: Pharmacological Exposure Predicts Smoking Risk Factors

First, it was hypothesized that greater pharmacological exposure would predict greater smoking risk factors. Results did not support this hypothesis. Pharmacological exposure explained 1.2% of the variance in smoking risk factors, but did not significantly predict smoking risk factors (t = 1.22, *n.s.*). Fit indices revealed this structural model had acceptable fit (GFI = .99; CFI = .91; RMSEA = .05).

Hypothesis 2: Social Smoke Exposure Predicts Smoking Risk Factors

Second, it was hypothesized that greater social smoke exposure would predict greater smoking risk factors. Four structural models were tested, one with each of the social smoke exposure latent variables. Parental smoking explained 3.6% of the variance in smoking risk factors and significantly predicted smoking risk factors (t = 2.25, $p < .05$). Fit indices revealed this model had good fit (GFI = .98; CFI = .99; RMSEA = .04). Sibling smoking explained 2.1% of the variance in smoking risk factors and did not significantly predict smoking risk factors (t = 1.71, *n.s.*). Fit indices indicated this model had good fit (GFI = .97; CFI = .97; RMSEA = .05). Peer smoking explained 56% of the variance in smoking risk factors and significantly predicted smoking risk factors (t = 7.09, $p < .01$). Fit indices showed this model had good fit (GFI = .98; CFI = .97; RMSEA = .05). Finally, school smoking explained 0.35% of the variance in smoking risk factors and did not significantly predict smoking risk factors (t = 0.56, *n.s.*). Fit indices showed this model had poor fit (GFI = .95; CFI = .31; RMSEA = .12). Taken together, these results provided partial support for the hypothesis as both parental and peer smoking significantly predicted smoking risk.

Hypothesis 3: Pharmacological and Social Smoke Exposure Predict Smoking Risk Factors

Third, it was hypothesized that when considered simultaneously, greater pharmacological exposure and greater social smoke exposure would both uniquely contribute to predicting smoking risk factors. Four structural were tested, one with each of the social smoke exposure latent variables. Parental smoking (t = 1.58, *n.s.*) and pharmacological exposure (t = -.85, *n.s.*) explained 4.7% of the variance in smoking risk factors, but neither significantly predicted smoking risk factors (see

SEM model with standardized estimates and loadings in Figure 1). Fit indices showed this model had good fit (GFI = .97; CFI = .99; RMSEA = .04). Sibling smoking (t = 1.38, *n.s.*) and pharmacological exposure (t = .64, *n.s.*) explained 2.7% of the variance in smoking risk factors, but neither significantly predicted smoking risk factors (see Figure 2). Fit indices showed this model had poor fit (GFI = .91; CFI = .77; RMSEA = .12). Peer smoking (t = 7.03, $p < .01$) and pharmacological exposure (t = -1.59, *n.s.*) explained 58% of the variance in smoking risk factors, but only peer smoking significantly predicted smoking risk factors (see Figure 3). Fit indices showed this model had good fit (GFI = .98; CFI = .98; RMSEA = .04). School smoking (t = .39, *n.s.*) and pharmacological exposure (t = .39, *n.s.*) explained 1.4% of the variance in smoking risk factors, but neither school smoking nor pharmacological exposure significantly predicted smoking risk factors (see Figure 4). Fit indices revealed this model had good fit (GFI = .98; CFI = .93; RMSEA = .03).

Taken together, these results showed that when pharmacological exposure and social smoke exposure were simultaneously modeled to predict smoking risk, they did not support the hypotheses. However, there is some evidence for minimal mediation. (This interpretation is presented most tentatively, as power limitations preclude identification of mediation or formal testing with Sobel indices). More specifically, in the sibling and school smoking structural models, including both the pharmacological exposure and the social smoke exposure variable resulted in an increased R^2 while the path coefficient for the social smoke exposure decreased. As example, when compared to the bivariate structural models (social smoke exposure predicts smoking risk), sibling smoking had a lower path coefficient (-.02) while the R^2 increased (+1.1%) and school smoking had a lower path coefficient (-.10) while the R^2 increased (+1.05%).

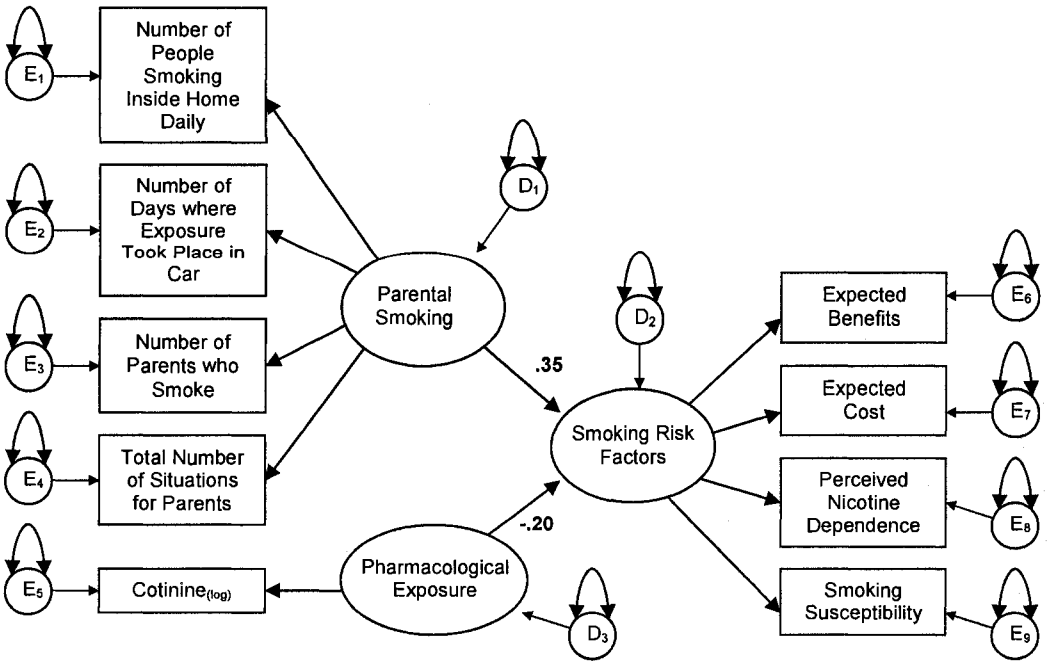

Figure 1. SEM of Social and Pharmacological Exposure of Smoking Risk: Parental Smoking.

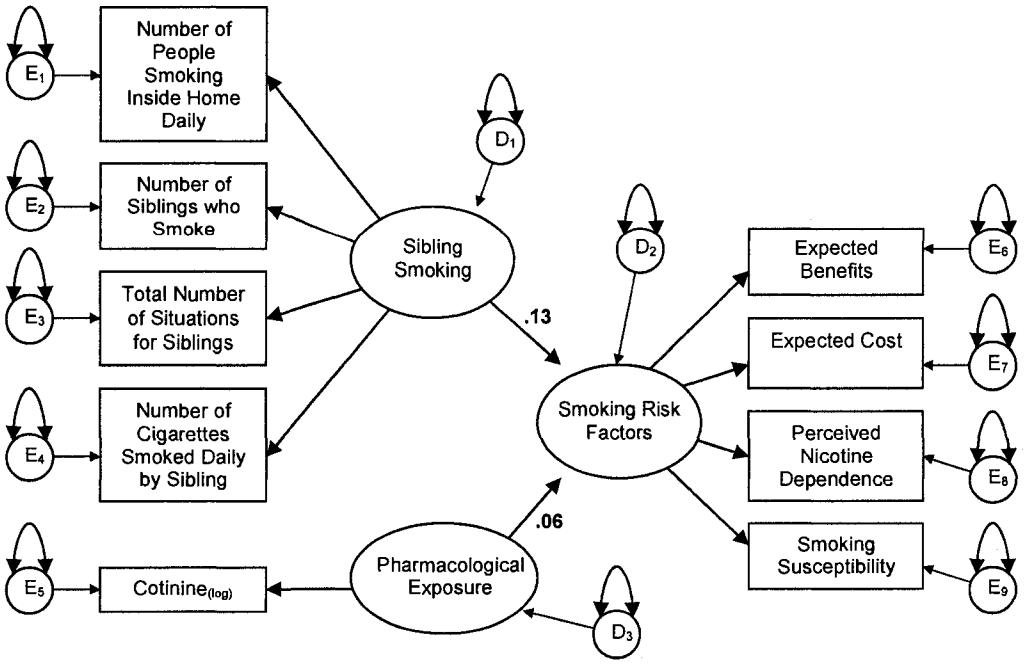

Figure 2. SEM of Social and Pharmacological Exposure of Smoking Risk: Sibling Smoking.

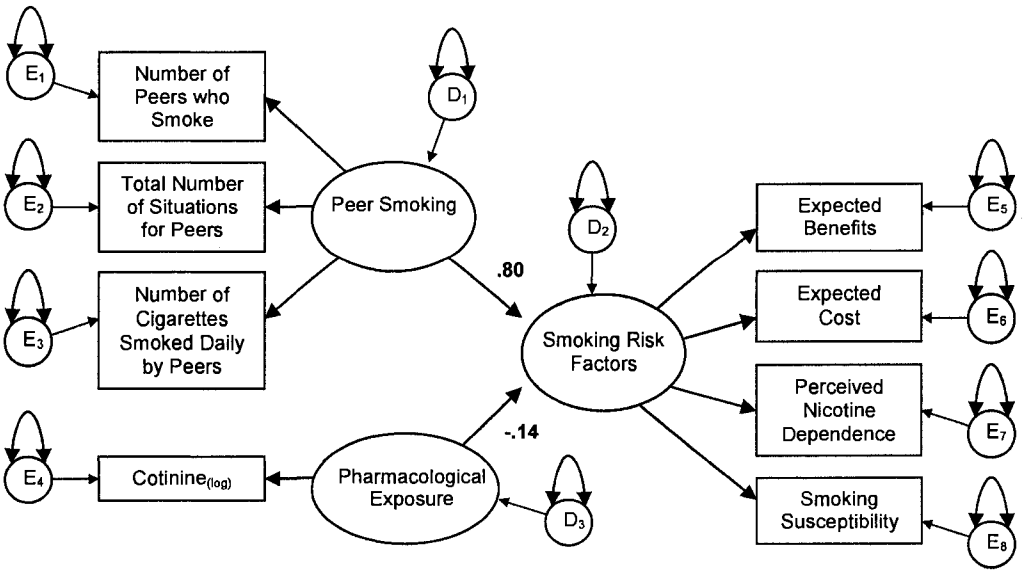

Figure 3. SEM of Social and Pharmacological Exposure of Smoking Risk: Peer Smoking.

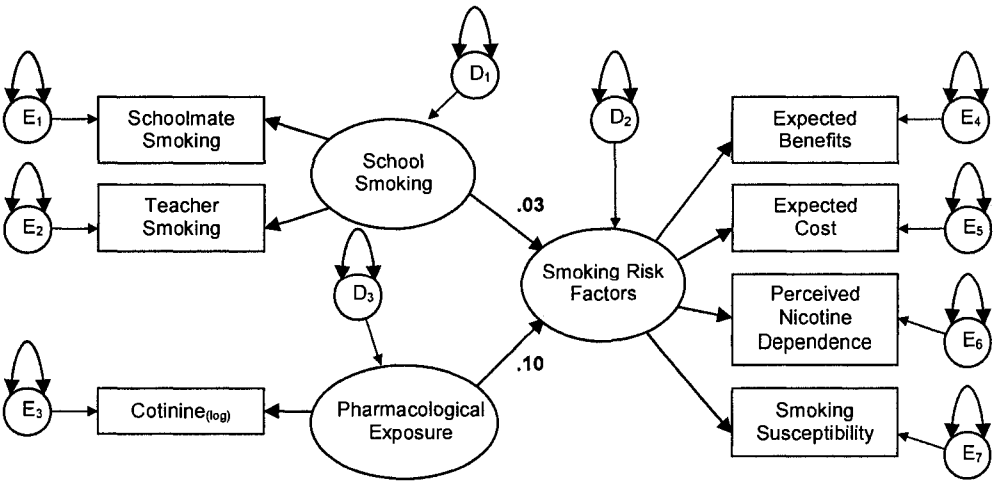

Figure 4. SEM of Social and Pharmacological Exposure of Smoking Risk: School Smoking.

Exploratory Post-Hoc Analyses

In SEM analysis, cotinine did not predict the latent variable smoking risk factors. Exploratory post-hoc analyses were conducted to determine if cotinine predicted any of the individual manifest variables that formed the latent variable. Univariate linear regression was used to test whether $cotinine_{(log)}$ predicted expected costs, expected benefits, perceived nicotine dependence, and smoking susceptibility. Consistent with the SEM analysis, $cotinine_{(log)}$ did not significantly predict any of the manifest variables individually (results not shown). The analyses were repeated in a multivariate regression using both $cotinine_{(log)}$ and carbon monoxide to predict smoking risk factors; all analyses were non-significant.

Similarly, given that the school smoke exposure latent variable had a poor fit, univariate linear regression was used to test whether schoolmate smoking or teacher smoking singularly predicted smoking risk. Neither variable was significantly associated with expected costs, expected benefits, perceived nicotine dependence, or smoking susceptibility.

Given that the sample largely include participants not exposed to pharmacological or social smoke exposure, exploratory post-hoc analyses were next conducted to examine how the restriction of range may have affected the results. Participants were categorized into four groups based on a composite score of parent, sibling, and peer smoke exposure. The groups were aggregated based on exposure level ranging from no exposure (exposed to 0 smokers) to high exposure (exposed to 3-9 smokers; see Table 16). The mean value for cotinine increased from .20 to 2.30 ng/mL as the number of smokers increased. Expected costs did not differ across the groups. In contrast, with increased exposure, expected benefits increase from 2.05 to 2.58, perceived nicotine dependence increased from .19 to 1.11, and smoking

Table 16

Exploratory Post-Hoc Analyses

Number of smokers	n (%)	Cotinine *M* (*SD*)	Expected Costs *M* (*SD*)	Expected Benefits *M* (*SD*)	Perceived Nicotine Dependence *M* (*SD*)	Smoking Susceptibility *M* (*SD*)
0	177 (52.4)	.20 (.53)	6.16 (2.09)	2.05 (1.35)	.19 (.79)	.87 (1.59)
1	64 (18.9)	.68 (1.10)	6.01 (2.24)	2.26 (1.51)	.14 (.59)	1.56 (2.20)
2	56 (16.6)	1.40 (2.95)	6.11 (1.92)	2.20 (1.57)	.30 (1.06)	1.50 (1.77)
3-9	41 (12.1)	2.30 (3.51)	5.89 (1.77)	2.58 (1.51)	1.11 (2.14)	2.59 (2.41)

Note. This table shows the means and standard deviations for cotinine and smoking risk factors based on the reported level of exposure.

susceptibility increased from .87 to 2.59. Follow-up ANOVA yielded that these group means were significantly different for perceived nicotine dependence ($F_{(3, 334)} = 9.08$, $p < .01$) and smoking susceptibility ($F_{(3, 334)} = 10.31$, $p < .01$). These results suggest that restriction of range in smoke exposure likely impacted the findings.

Discussion

The goal of the present study was to evaluate whether pharmacological and social smoke exposure differentially predicted smoking risk among never-smoking youth. Smoking risk included endorsement of fewer negative expectancies (i.e., expected costs) and more positive expectancies (i.e., expected benefits), greater perceived nicotine dependence, and greater smoking susceptibility. Based on previous studies demonstrating that reported second-hand smoke exposure was associated with smoking risk (cf., Becklake et al., 2005; Bélanger et al., 2008; Okoli et al., 2007), the aim of the present study was to extend these findings using an objective measure of second-hand smoke exposure.

In the current project, almost 90% of participants were categorized as never-smokers based on the classification criteria used by the provincial agency *Institut de la statistique du Québec* (Dubé & Camirand, 2007). This prevalence rate of never-smokers in our sample is consistent with those observed at the national level with the Youth Smoking Survey (Health Canada, 2008). While many participants reported they were not exposed to parental smoking (67%); one-third (33%) reported at least one parent smoked. This rate of exposure is consistent, but slightly higher than what has been observed in the Canadian Tobacco Use Monitoring Survey (CTUMS), with rates of exposure ranging from 18.4% to 25.8% for youth in the province of Québec (Health Canada, 2007). It is possible the rates observed in the present study were higher than those observed in the CTUMS because we only included French-speaking participants; the CTUMS included both French- and English-speaking participants (i.e., rates of smoking are higher among French-speaking persons). Additionally, these higher rates of exposure paradoxically may be explained by the new smoking ban which has been recently enforced in the province of Québec. Given that smokers

are no longer allowed to smoke in public places, some researchers have speculated they now smoke more frequently in private places, including their home and car. The high majority of participants reported no smoke exposure to sibling smoking (>90%) nor peer smoking (75%). Consistent with the low prevalence of self-reported social smoke exposure, values for biomarkers (i.e., cotinine, carbon monoxide, and carboxyhemoglobin) were low, suggesting no or little pharmacological exposure to nicotine via second-hand smoke. Altogether, our sample was largely comprised of never-smokers who were not exposed to second-hand smoke. As such, our sample likely did not include an adequate range of exposure to second-hand smoke (i.e., restriction of range). Ideally, we should have included a more heterogeneous sample with no, low, moderate, and high exposure across parents, siblings, and peers. Having too few participants with moderate to high exposure to second-hand smoke created challenges when testing our research questions.

Pharmacological exposure did not predict smoking risk. This finding did not support our first hypothesis and was inconsistent with previously reported research. Methodological differences between the current study and previous research may explain these discrepant findings. For example, in a longitudinal study, Becklake and colleagues (2005) found that cotinine levels in non-smoking children predicted smoking uptake four years later during adolescence. Smoking behavior was measured with both parental and student report; no measures of smoking risk were included. In the present study, we only used cross-sectional data. It is possible that over time, our participants with higher cotinine levels may be more likely to initiate smoking. Our measures were solely based on participants' self-report; using both parental and student report likely improved the accuracy of measuring smoking behavior. Importantly, our study investigated early risk factors for smoking (i.e., expected costs,

expected benefits, perceived nicotine dependence, smoking susceptibility) as the outcome variable among never-smokers, while Becklake and colleagues (2005) only used smoking status as their outcome variable. Hence, if we were to follow our participants for four years and evaluate their smoking status, cotinine may be uniquely associated with transitioning from never- to ever-smoking.

Similarly, Okoli and colleagues (2007) found that among never-smoking employees of bars and restaurants, greater second-hand smoke exposure measured with hair nicotine levels was associated with higher endorsement of physical sensations (e.g., depressed mood, trouble sleeping, feeling anxious). An important difference of this study was the choice of biomarker. Hair nicotine levels reflect second-hand smoke exposure over the past month (Al-Delaimy, 2002), while salivary cotinine reflects shorter term exposure to second-hand smoke because it has a half-life of approximately 17 hours (Benowitz, 1996a). Another important difference was the age and exposure level of the participant sample. In Okoli and colleagues study (2007), adults exposed to high levels of second-hand smoke in bars and restaurants were more likely to absorb greater amounts of nicotine than youth in our study who were exposed to lower levels of second hand-smoke at home, in a car, or on school grounds. In fact, Jaakkola and Jaakkola (1997) reported that smoke concentration (e.g., number of exposure sources, volume of space, air ventilation), duration, and frequency of second-hand smoke exposure are important variables to take into consideration when measuring second-hand smoke exposure. Taken together, these methodological differences may explain why pharmacological exposure was not associated with smoking risk, contrary to that found in previous studies.

Social smoke exposure of parents and peers predicted smoking risk. This finding largely supported our second hypothesis. Greater parental smoking was

associated with increased smoking risk; this is consistent with previous work showing an association between parental smoking and smoking status (Otten et al., 2007; Peterson et al., 2006); smoking expectancies (Hine et al., 2002, 2007); perceived nicotine dependence (Bélanger et al., 2008); and smoking susceptibility (Pierce et al., 1996). Sibling smoking was not associated with smoking risk. This finding does not corroborate previous findings showing an association between sibling smoking and smoking status (Rajan et al., 2003; Slomkowski et al., 2005). Our low percentage of youth who reported that at least one sibling smoked (<10%) may partly explain this finding. Greater peer smoking was strongly associated with greater smoking risk; this is consistent with other studies showing an association between peer smoking and smoking status (e.g., Bricker et al., 2006; Distefan et al., 1998; O'Loughlin et al., 1998; Vink et al., 2003), smoking expectancies (e.g., Hine et al., 2002, 2007), perceived nicotine dependence (Bélanger et al., 2008), and smoking susceptibility (e.g., Leatherdale et al., 2005, 2006). Finally, neither observing schoolmates nor teachers smoking was significantly associated with smoking risk, suggesting that more distal sources of influence may not be associated with smoking risk. This result is not consistent with previous findings that school smoke exposure (schoolmate smoking, observing teachers smoking, school smoking policies) is associated with greater smoking behaviour (e.g., Barnett et al., 2007; Poulsen et al., 2002). In sum, only parental and peer social smoke exposure were associated with smoking risk factors in our sample of 6th and 7th grade students. Further, peer smoking had a stronger influence on smoking risk than parents among youth in 6th and 7th grade. This finding is consistent with previous research showing that parents have greater influence during childhood, while peers have greater influence during adolescence.

Pharmacological and social smoke exposure did not uniquely contribute to predicting smoking risk. This finding did not support our third hypothesis. Altogether, the results largely showed that neither pharmacological exposure nor social smoke exposure were associated with smoking risk factors when they were modeled simultaneously. Given that there is much shared variance between pharmacological exposure and social smoke exposure, these findings are likely to be valid. It is possible that controlling for social smoke exposure automatically contributed to controlling for pharmacological exposure. Although pharmacological exposure and social smoke exposure probably have different mechanisms, it may not be possible to tease them apart with statistical procedures. It is possible that only an experiment may allow researchers to evaluate the differential contributions of pharmacological and social smoke exposure.

Integration of Findings

The tenet of the present study was that pharmacological exposure would uniquely contribute to predicting smoking risk factors (i.e., smoking expectancies, perceived nicotine dependence, smoking susceptibility), after controlling for social smoke exposure. Contrary to expectations and inconsistent with previous findings, cotinine, an objective and valid measure of nicotine intake, was not associated with smoking risk. Based on our findings, it seemed that cotinine levels in youth may actually be a good proxy or biomarker of social smoke exposure. However, particularly problematic was the limited range of smoke exposure (both pharmacological and social) in our study. Selective recruitment of participants to ensure greater heterogeneity in smoke exposure is recommended for future research. Exploratory analyses were conducted to consider how this restriction of range may have impacted the findings. After collapsing participants into four categories based

on number of smokers to whom they were exposed, greater second-hand smoke exposure was associated with higher cotinine levels, perceived nicotine dependence, and smoking susceptibility.

Along these same lines, the low number of participants exposed to second-hand smoke limited the power of the study. Notably, however, increasing sample size will not necessarily counter this problem if we were to recruit a large number of never-smokers with similar rates of exposure. A better strategy would be to recruit never-smokers based on their level of exposure to second-hand smoke (children of non-smoking parents, social smoking parents, one parent smoker, both parents smoker). This selective recruitment strategy would likely result in a more heterogeneous sample for both pharmacological and social smoke exposure.

Jaakkola and Jaakkola (1997) reported that cotinine has a half-life of 32-82 hours in the bodily fluids of children, which signifies that cotinine values observed in this study represent short-term exposure to second-hand smoke (i.e., less than four days). Given the sporadic nature of second-hand smoke exposure in never-smokers, this time span may be too short to precisely measure pharmacological smoke exposure in youth. Alternatively, hair nicotine represents a long-term exposure to second-hand smoke of about one month (Al-Delaimy, 2002). It is recommended that future studies use hair nicotine measures (albeit more costly) to evaluate cumulative exposure to second-hand smoke exposure over a longer time interval.

Recently, work on nicotine metabolism, which is processed by enzymes CYP2A6, CYP2B6, and CYP2E1 (Hukkanen, Jacob, & Benowitz, 2005), has shown that smokers who are genetically predisposed to metabolize nicotine slowly, smoke a lower number of cigarettes (Malaiyandi, Sellers, & Tyndale, 2005). This finding provides support for the idea that variation in the metabolism of nicotine into cotinine

is influenced by genes. Of interest are differences in metabolic rates among never-smoking youth who are exposed to second-hand smoke; in fact, these differences are pertinent when using biomarkers. Based on the assumption that slow metabolizers have higher levels of cotinine in their body, high levels of salivary cotinine could reflect slow metabolism of nicotine, while youth who metabolize nicotine rapidly may have similar salivary cotinine levels to youth who are not exposed. Thus, it could be relevant to collect DNA samples in a future study to account for the effects of nicotine metabolism on cotinine levels. Although this study did not support the role of pharmacological exposure on increased smoking risk among never-smoking youth, increasing the number of participants with higher levels of smoke exposure, using hair nicotine as a more stable biomarker, and collecting DNA samples to assess genetic variations in nicotine metabolism represent promising possibilities to better evaluate the presence of a physiological pathway between pharmacological exposure and smoking behaviour.

Strengths

There were several methodological strengths in the present study. First, the measures used in the present study are considered to be psychometrically sound. Self-report measures were largely derived from the Youth Smoking Survey, a national epidemiological survey (representative of the population) for which normative Canadian data are available for comparison. Smoking expectancies were evaluated using the French-Canadian version of the Smoking Expectancy Scale for Adolescents. In a validation study, Racicot and colleagues (2008) replicated the factor structure of the original scale (Hine et al., 2007) using the translated French-Canadian version. Perceived nicotine dependence was measured using a validated scale for adolescents (Nicotine Dependence Scale for Adolescents; Nonnemaker et al., 2004), while

smoking susceptibility was measured using an operational definition commonly reported in the literature (Pierce et al., 1996). Lastly, continine assays are considered highly reliable and valid markers of second-hand smoke exposure.

Second, in order to test pharmacological and social smoke exposure as differential predictors, Structural Equation Modeling (SEM) was used. Unlike ANOVA and regression analyses, SEM analyses allowed for the simultaneous examination of latent variables made up of several manifest variables. In general, the measurement models we derived were largely acceptable in terms of goodness-of-fit. Thus, the latent variables that were created maximized the multiple indices for social smoke exposure (i.e., number of smokers, number of situations, frequency of smoking cigarettes) and smoking risk (i.e., expected benefits and costs, perceived nicotine dependence, susceptibility).

Lastly, recruiting a large sample size (N = 599) of 6-7th graders in public French-speaking schools facilitated recruitment of never-smokers who were more likely to be exposed to second-hand smoke based on results from the Youth Smoking Survey and the Canadian Tobacco Use Monitoring Survey. Despite these methodological strengths, there were limitations which may have impacted our results.

Limitations

One limitation of the present study was the cross-sectional design which precludes the establishment of cause-effect relationships among the predictors (social and pharmacological exposure) and the outcome variable (smoking risk factors); only an experimental design with a temporal component supports causality. However, investigating associations is an important initial step in determining if they should be

tested experimentally in the future (examples of potential experimental studies are described below).

Second, exposure to second-hand smoke among participants in this study was low, with more than 50.0% of participants reporting they were exposed to no smokers (parents, peers, or siblings). The original recruitment aims were to target young youth who were non-smoking. There were no specific aims to ensure a fair distribution of exposure to second-hand smoke. Consequently, reflecting this minimal second-hand smoke exposure, cotinine was asymptotic, and thus, made transformation of the non-normally distributed variable challenging. Transformations make the interpretation of a variable more difficult. Procedures which handle censored or asymptotic data are often preferred, yet quite complex. For example, the SAS Proc LIFEREG is a statistical procedure that handles data which are censored, just like salivary cotinine.

Third, pharmacological and social smoke exposure were not adequately teased apart. In fact, it is possible that in the current study, the cotinine measures likely provide a more accurate measure of social smoke exposure; in other words, they were a biomarker for social smoke exposure. With regard to pharmacological exposure, only one saliva sample and one expired breath sample were collected to derive measures of cotinine, carbon monoxide, and carboxyhemoglobin. While these biomarkers are recognized as good indicators of second-hand smoke exposure, other measures could have been collected to more precisely measure pharmacological exposure. An improved methodological design would include sampling air nicotine with stationary monitors inside the bedroom, home, car, classroom, or neighbourhood of participants. Such a measurement would permit a comparison between the level of exposure to second-hand smoke (measured with stationary monitors) with the amount

of nicotine absorbed into the body (measured with salivary cotinine). However, such a measurement is more methodologically complex when sample sizes are large.

Finally, this study was based on student self-report data exclusively. A multi-modal study, with multiple informants (parents, siblings, peers, classmates, teachers, principals) would be a stronger methodological design. Additionally, given that classrooms of participants were selected, this data is also amenable to multilevel modeling to evaluate differences between classes within schools or across different schools.

Future Research

Although an experiment examining pharmacological smoke exposure would pose considerable ethical challenges, methodologically it would elucidate whether nicotine intake via second-hand smoke uniquely contributes to predicting smoking risk, and thus, suggests a possible physiological pathway. One possible experimental design would be to compare social smoke exposure (watching monitor with parent smoking vs. reading), cue exposure (scent of smoke, with no active chemicals vs. citrus scent), and pharmacological exposure (nicotine patch/gum vs. placebo) and evaluate how these influence youth's smoking risk (endorsement of expected benefits/costs, smoking susceptibility, etc.). Alternatively, participants could be grouped based on objective second-hand smoke exposure in home, car, and neighbourhood (using stationary air sampling devices) and then examine their levels of pharmacological and social smoke exposure and smoking risks. This would permit adequately capturing cases with high pharmacological exposure and low social smoke exposure (i.e., only one parent smokes in very few situations, but smokes very heavily) as compared to cases with low pharmacological exposure and high social smoke exposure (i.e., both parents are social smokers in a variety of settings, but

smoke very infrequently). Thus, these proposed experiments have the potential to permit the evaluation of the relative contribution of social and pharmacological smoke exposure.

In addition to these experimental design strategies, suggested methodological improvements for future research include recruiting participants with a larger range of smoke exposure, using hair samples to detect longer exposure to nicotine, and collecting DNA samples to adequately account for differences in nicotine metabolism. Finally, using a longitudinal design with multiple time points would more convincingly support the role of a physiological pathway of risk for smoking. Similar to the Becklake et al. study (2005), the addition of a temporal component would permit the evaluation of baseline predictors that contribute to subsequent smoking risk factors in never-smokers. As such, data collection with multiple time points facilitates understanding of the sequence in which these relationships are established.

Conclusion

In conclusion, the results of the current study did not support that pharmacological and social smoke exposure are differential predictors of smoking risk factors (including smoking expectancies, perceived nicotine dependence, smoking susceptibility). These results do not provide evidence of a physiological pathway of smoking risk. Methodological limitations including a sample with a restricted range of second-hand smoke exposure (both pharmacologically and socially) likely affected these results. Future researchers are encouraged to consider recruiting youth with greater exposed to second-hand smoke, to use monitors to assess air quality, and to more precisely measure pharmacological exposure (hair nicotine, DNA encoding for CYP2A6, CYP2B6, and CYP2E1 enzymes which metabolize nicotine.)

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