Where there is smoke, there is risk: Social and pharmacological exposure to smoking increase risk for smoking behavior during adolescence

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Abstract

Where there is smoke, there is risk: Social and pharmacological exposure to smoking increase risk for smoking behavior during adolescence

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Social exposure to smoking, or observing smokers, is a robust predictor of adolescent smoking. Recently, an emerging line of research posited that nicotine exposure from secondhand smoke could predict nicotine dependence symptoms and smoking initiation among never-smokers, given that nicotine is a psychoactive substance present in secondhand smoke. The objective of the present research program was to evaluate social exposure to smoking and pharmacological exposure to nicotine from SHS as differential predictors of smoking precursors, such as smoking expectancies, smoking susceptibility, or nicotine dependence symptoms. Adolescent never-smokers constitute an ideal population to study, provided that nicotine exposure from SHS is not confounded by active smoking in that population. This dissertation includes three original quantitative studies.

Using longitudinal data from the NDIT study, Study 1 identified exposure to peer smoking as a significant predictor of nicotine dependence symptoms among never-smokers, which provided convincing evidence that adolescents do not mistakenly endorse such symptoms. Using cross-sectional data from the AdoQuest Study, Study 2 developed the Social Smoking Situations (S^3) Scale, an enhanced psychometric instrument measuring the situational contexts in which social exposure to smoking occurs. Compared with existing measures of social exposure (e.g., "who is smoking"), the S^3 Scale was a stronger predictor of smoking behavior and smoking expectancies. Finally,

Study 3 investigated the differential relations of social and pharmacological exposure to smoking with smoking precursors, using cross-sectional data from the AdoQuest study. This study is the first to demonstrate significant effects of pharmacological exposure to nicotine on smoking expectancies and nicotine dependence symptoms in a sample of adolescent never-smokers.

Overall, this research program provided scientific evidence that exposure to nicotine is a risk factor for smoking among adolescent never-smokers. Its distinctive feature pertains to its emphasis on dismantling the major components of smoke exposure (social vs. pharmacological), and examining their relative consequences on increasing risk for adolescent smoking. It is recommended that future studies use longitudinal data to investigate the unique effects of social and pharmacological exposure on smoking initiation. Finally, current findings could be used to promote complete smoking bans in adolescents' homes and cars.

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DEDICATION

To my beloved grandmother, Lorraine Pigeon-Desbiens (1918-2013)

CONTRIBUTION OF AUTHORS

Study 1

Study 1 was conducted as part of the larger Nicotine Dependence in Teens (NDIT) study, a longitudinal cohort of adolescents designed to investigate the onset and development of cigarette smoking and nicotine dependence. Simon Racicot developed the research question, conducted the literature review, undertook the statistical analyses, interpreted the results, and wrote and revised the manuscript. As Simon Racicot's research supervisor, Dr. Jennifer J. McGrath supervised the literature review, the statistical analyses, and revised the manuscript. Dr. Igor Karp designed the analytic plan and revised the manuscript. Dr. Jennifer O'Loughlin designed the study, contributed to the protocol development, and revised the manuscript.

Study 2 and Study 3

Study 2 and Study 3 were conducted as part of the larger AdoQuest study, an ongoing prospective investigation evaluating the natural course of the development of smoking behavior among French-speaking youth in Montréal, QC. For both Study 2 and Study 3, Simon Racicot developed the research question, conducted the literature review, designed the analytic plan, undertook the statistical analyses, interpreted the results, and wrote and revised the manuscript. As Simon Racicot's research supervisor, Dr. Jennifer J. McGrath co-developed the research question, supervised the statistical analyses, and revised the manuscript.

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LIST OF ABREVIATIONS

APA ..... American Psychiatric Association

CDC ..... Centers for Disease Control and Prevention

CTUMS ..... Canadian Tobacco Use Monitoring Survey

DANDY ..... Development and Assessment of Nicotine Dependence in Youth

ICC ..... Intraclass Correlation

nAChRs ..... Nicotinic Acetylcholine Receptors

ND ..... Nicotine Dependence

NDIT ..... Nicotine Dependence in Teens

OR ..... Odds Ratio

S^3 ..... Social Smoking Situations

SHS ..... Secondhand Smoke

USDHHS ..... United States Department of Health and Human Resources

WHO ..... World Health Organization

YSS ..... Youth Smoking Survey

GENERAL INTRODUCTION

Prevalence of Smoking during Adolescence

Public health organizations around the world have labeled tobacco smoking as the most preventable source of disease, chronic disability, and death (U.S. Department of Health and Human Services [USDHHS], 2004; World Health Organization, 2013), and have reported that tobacco dependence is as powerful a dependency as that of illegal drugs, such as cocaine or heroin (USDHHS, 1988). In spite of multiple prevention efforts aiming to decrease the prevalence rates of smoking, the number of individuals who continue to smoke remains high, and children and adolescents are no exception to this trend. In fact, tobacco smoking typically begins during adolescence and continues well into adulthood (Chassin, Presson, Pitts, & Sherman, 2000). The most recent Canadian Youth Smoking Survey (2010-11 YSS; Health Canada, 2012) revealed that on average, youth smoke their first cigarette at age 13. Youth who smoke daily in grades 6-9 (1%) and grades 10-12 (5%) smoke approximately nine cigarettes per day. Further, this survey showed that 15.5% of Canadian youth in grades 6-9 and 40% in grades 10-12 have tried smoking. These rates were higher in the province of Québec where 24.4% of youth in grades 6-9 and 44% in grades 10-12 have ever tried smoking (Health Canada, 2012).

Secondhand Smoke Exposure: Nature and Prevalence

Although research convincingly shows that active smoking is an unhealthy lifestyle behavior choice, passive smoking or exposure to secondhand smoke (SHS) represents another considerable health concern. SHS can be detected in ambient air when a tobacco product burns (i.e., sidestream smoke) and when a smoker exhales tobacco smoke (i.e., mainstream smoke). Importantly, it has been associated with increasing rates

of pediatric conditions, including respiratory illness and sudden infant death syndrome (USDHHS, 2006), and behavior problems, such as attention deficit and hyperactivity disorder (Kabir, Connolly, & Alpert, 2011). The influential 2006 report of the U.S. Surgeon General concluded unequivocally that there is no such thing as a *risk-free level* of SHS exposure (USDHHS, 2006). In fact, Health Canada (2011) reported that SHS is comprised of more than 4000 chemicals of which 70 are carcinogenic substances; nicotine, tar, carbon monoxide, benzene, hydrogen cyanide, and formaldehyde are some of its principal components. According to the latest Canadian Tobacco Use Monitoring Survey (CTUMS 2012; Health Canada, 2013), 3.3% of children aged 0-11 years and 6.5% of adolescents aged 12-17 years were exposed to SHS at home; prevalence rates were twice as high in Québec (7.2% for ages 0-11; 12.2% for ages 12-17; Health Canada, 2013). Other North American statistics showed that overall, 53% of children are exposed to SHS (Centers for Disease Control and Prevention [CDC], 2010).

In addition to acknowledging that SHS exposure contributes to negative health outcomes, an emerging line of research has posited that SHS exposure could also lead to behavioral sequelae, given that greater SHS exposure is linked to greater nicotine exposure. Due to the psychotropic effects of nicotine, repeated exposure to nicotine from SHS is increasingly recognized as a plausible risk factor for smoking initiation during adolescence (Becklake Ghezzo, & Ernst, 2005; Okoli, Kelly, & Hahn, 2007). In fact, some researchers have suggested the possibility of a "physiological pathway" between nicotine exposure from SHS and prospective smoking behavior (Anthonisen & Murray, 2005), although the presence and nature of such a pathway remains to be identified empirically.

Physiological Effects of Nicotine Administration

The current state of knowledge is clear: nicotine is indisputably the psychoactive substance responsible for tobacco dependence in humans (Henningfield & Heishman, 1995; Stolerman & Jarvis, 1995) and sensitization in animals (Vezina, McGehee, & Green, 2007). After inhalation, nicotine rapidly reaches the brain within about 10 seconds (Benowitz, 1988), and acts as an agonist on the nicotinic cholinergic receptors (nAChRs). Acetylcholine is the neurotransmitter that naturally binds to nAChRs (Benowitz, 2008; Wonnacott, Sidhpura, & Balfour, 2005) that comprise α-subtypes (α2-α10) and β-subtypes (β2-β4; Dani & De Biasi, 2001; Mineur & Piciotto, 2008). Receptors with the α4β2 subtype are the most prevalent high-affinity binding sites for nicotine (Flores, Rogers, Pabreza, Wolfe, & Kellar, 1992). Tobacco use has been associated with the upregulation of nAChRs (Buisson & Bertrand, 2001; Littleton, 2001), which signifies there is a significant increase in the number of receptors on the cells. Upon nicotine binding in the ventral tegmental area of the brain, dopamine is released in the nucleus accumbens (Benowitz, 2010; Nestler, 2005). Dopamine is a neurotransmitter that is highly implicated in reward-seeking behaviors (Balfour, Wright, Benwell, & Birrell, 2000; Benowitz, 2008; Di Chiara, 2000). The specific pathway between nicotine binding in the ventral tegmental area and dopamine release in the nucleus accumbens has been associated with nicotine dependence (ND) (Laviolette & van der Kooy, 2004).

The effects of nicotine use on human neuroanatomy have been previously investigated, using autopsied brains. Compared to nonsmokers, smokers have greater density of nAChRs binding in different areas of the brain, including the hippocampal formation and neocortex, cerebellar cortex, gyrus rectus, and median raphe nuclei

(Benwell, Balfour, & Anderson, 1988), as well as the prefrontal and temporal cortices (Perry, Davila-Garcia, Stockmeier, & Kellar, 1999). The adolescent brain has been shown to be particularly vulnerable to the effects of nicotine (Slotkin, 2002), as nicotine interferes with normal neuronal activity in different brain regions, including the cortex and midbrain (Slotkin, Pinkerton, & Seidler, 2006). Nicotine exposure in adolescent animals has been associated with upregulation of nAChRs for more than a month after the last dose of nicotine (Abreu-Villaça et al., 2003; Trauth, Seidler, McCook, & Slotkin, 1999). Moreover, adolescents appear to be more sensitive to the reinforcing effects of nicotine and less sensitive to its aversive effects (Torres, Tejeda, Natividad, & O'Dell, 2008; Shram, Funk, Li, & Le, 2006). Taken together, research demonstrates that nicotine is a psychoactive substance associated with direct changes in the central nervous system. However, the question remains whether nicotine exposure from SHS is similarly capable of changing the brain, and whether such changes have an influence on smoking behavior. Further research is warranted to address these questions, especially in adolescent populations.

Measurement of Nicotine Exposure from SHS

In their seminal review article, Jaakkola and Jaakkola (1997) indicated that nicotine exposure through SHS can be measured with biomarkers or passive nicotine monitors. Other methods, such as questionnaires, interviews, or monitors measuring inhalable suspended particles are unable to provide estimates specific to nicotine. Okoli, Kelly, and Hahn (2007) reported in their review article that biomarkers can be collected from blood, saliva, urine, hair, or nails, and are affected by bodily processes like metabolism and elimination (U.S. Environmental Protection Agency, 1992). Thus,

biomarkers are a valid measure of one's pharmacological dose of nicotine. Specifically, nicotine biomarkers of SHS exposure measure either the amount of nicotine or its principal metabolite, cotinine. After nicotine intake, more than 80% of nicotine is converted into cotinine by the hepatic enzyme CYP2A6 (Benowitz, 1996). Jaakkola and Jaakkola (1997) indicated that the half-life of cotinine ranges from 32-82 hours in children, reflecting a window of exposure of 2-4 days. Relatedly, the passive nicotine monitor allows for an estimation of the total concentration of nicotine to which individuals are exposed in their environment (Hammond & Leaderer, 1987; Leaderer & Hammond, 1991). The monitor can be worn on oneself or left in a particular place to capture the total concentration of airborne nicotine. Unlike the biomarkers of SHS exposure, the passive nicotine monitor is unaffected by bodily functioning. Taken together, it appears that nicotine exposure is best measured by a combination of biomarkers and passive nicotine monitors, which permits measurement by triangulation.

Nonsmokers and Nicotine Exposure from SHS

Epidemiological data have revealed that nicotine exposure among nonsmokers can yield biological concentrations of nicotine comparable to those found in smokers. When examining nonsmoking employees exposed to SHS at work, researchers have found that the amount of nicotine in their hair was slightly lower than that of smokers (6.69 vs. 7.92 ng/mg: Al-Delaimy, Fraser, & Woodward, 2001; 1.03 vs 1.19 ng/mg: Dimich-Ward, Gee, Brauer, & Leung, 1997). Moreover, experimental data indicate that when exposed to the same concentration of airborne nicotine, nonsmoking children displayed higher biological doses of nicotine than nonsmoking adults, after adjusting for weight (2.3 mg/kg vs. 1.7mg/kg; Willers, Skarping, Dalene, & Skerfving, 1995). Nicotine

doses were also higher in children (27%) than adults (16%) seven days after the exposure, due to a smaller lung volume. Thus, nonsmoking youth appear to be more vulnerable to nicotine exposure than nonsmoking adults. Using blood samples, Pacifici and colleagues (1995) reported blood concentrations of nicotine in nonsmokers that, interestingly, were shown to have psychoactive effects in smokers (discriminative cue: Perkins, Fonte, Sanders, Meeker, & Wilson, 2001). Taken together, these studies convincingly demonstrate that never-smokers exposed to nicotine from SHS are not nicotine-naïve, as they are pharmacologically exposed to nicotine present in SHS.

SHS Exposure as a Risk Factor for Smoking Behavior

Within the past decade, researchers have begun to consider that nicotine exposure from SHS exposure in itself may be a risk factor for smoking behavior. Relying on an objective measure of pharmacological smoke exposure in nonsmoking children, Becklake et al. (2005) reported that salivary cotinine predicted smoking initiation four years later, even after adjusting for the number of smokers at home, which is considered a well-established social predictor of smoking initiation. This longitudinal study was the first to reveal the plausibility of behavioral sequelae related to nicotine exposure from SHS. Next, Okoli, Rayens, and Hahn (2007) also used an objective measure of pharmacological smoke exposure and demonstrated that nonsmoking adults with higher hair nicotine values were more likely to endorse ND symptoms. Thereafter, Bélanger and colleagues (2008) found that a small proportion of never-smoking children endorsing SHS exposure inside an automotive vehicle (4.6%) endorsed ND symptoms, even after ruling out the influence of sibling and peer smoking. Although these researchers did not measure SHS exposure objectively, their intriguing findings sparked curiosity regarding a

plausible link between nicotine exposure and ND symptoms among never-smokers.

Of growing importance is the possibility that endorsing ND prior to smoking initiation represents a risk factor for eventual smoking behavior. Using longitudinal data from the *Nicotine Dependence in Teens* (NDIT) study, O'Loughlin, Karp, Koulis, Paradis, and DiFranza (2009) found that endorsing ND symptoms predicted first puff smoking and onset of daily smoking. Subsequently, Racicot, McGrath, and O'Loughlin (2011a) measured the differential effects of pharmacological smoke exposure and social exposure to smoking on smoking precursors among never-smoking adolescents, using cross-sectional data. To methodologically tease apart pharmacological smoke exposure from social exposure to smoking, Racicot and colleagues (2011a) used both salivary cotinine and self-report questions (i.e., number of smokers, number of contextual situations of smoke exposure). Number of smokers among parents, siblings, and peers independently predicted key smoking precursors, namely, ND symptoms, smoking susceptibility, and expected benefits of smoking; however, salivary cotinine did not predict any precursors, most likely due to the low prevalence of SHS exposure observed in their sample. This latter study was the first step in trying to document a relation between nicotine exposure from SHS and smoking precursors, given that influential sources of social exposure to smoking had been controlled for statistically. The authors recommended that their findings be replicated and extended using different types of biomarkers and using a wider range of smoking precursors. Altogether, these studies provided preliminary yet credible support to the hypothesis that pharmacological exposure to nicotine represents a unique risk factor for smoking initiation and progression during adolescence. However, the number of studies remains scarce and further research

endeavors are warranted.

Smoking Milestones and Early Reports of ND Symptoms

The natural course of the development of smoking behavior and ND during adolescence has been examined. In contrast to the widely held assumption that ND develops from daily use over a long period of time, prospective cohort-based studies have shown that ND symptoms can emerge soon after initiation. The NDIT study revealed that after smoking their first puff of cigarette (0 months), adolescents reported having cravings (4.5 months), feeling physically addicted to cigarettes (5.4 months), and having withdrawal symptoms (11.0 months), two years *prior to* meeting ICD-10 criteria for ND dependence (40.6 months; Gervais, O'Loughlin, Meshefedjian, Bancej, & Tremblay, 2006). In the *Development and Assessment of Nicotine Dependence in Youth* (DANDY) study, DiFranza et al. (2007) observed that ND symptoms could develop as soon as only two days after inhaling smoke from a first cigarette. Further, DiFranza and colleagues (2007) found that adolescents who felt relaxed when inhaling smoke from their first cigarette were more likely to report ND symptoms. Scragg, Wellman, Laugesen, and DiFranza (2008) later reported that 25% of adolescents who endorsed ND symptoms had smoked only one cigarette in their lifetime. Taken together, these findings provide interesting evidence supporting the idea that ND may develop rapidly after the onset of active smoking during adolescence. Considering that: 1) nonsmokers exposed to SHS can absorb quantities of nicotine similar to those observed in smokers (Al-Delaimy et al., 2001; Dimich-Ward et al., 1997); 2) such quantities in nonsmokers have been linked to psychoactive effects in smokers (Pacifici et al., 1995; Perkins et al., 2001); and 3) adolescent smokers can progress rapidly to ND soon after active smoking is initiated

(Gervais et al., 2006; DiFranza et al., 2007), the question remains whether nicotine exposure from SHS among never-smokers leads to neuroadaptations that would, in turn, confer greater risk for smoking initiation during adolescence.

Social Exposure to Smoking as a Risk Factor for Smoking Behavior

One of the most well-established, robust mechanisms for smoking behavior, especially initiation, is social exposure to smoking in one's environment (e.g., social learning; USDHHS, 2012). During adolescence, smoking by parents, siblings, and friends represent the main sources of social exposure to smoking (Avenevoli & Merikangas, 2003; Hoffman, Sussman, Unger, & Valente, 2006). According to the Social Learning Theory (Bandura, 1977), adolescents observe and imitate the behavior of smokers whom they consider role models. Traditionally, social learning theorists posited that adolescents whose role models include parents, siblings, or peers who smoke are more likely to take up smoking than those whose role models do not smoke. However, such theorists omitted to recognize that observing smokers does not confer only greater social influence, but also greater pharmacological exposure to SHS and its components, such as nicotine (Okoli, Kelly, & Hahn, 2007; Racicot et al., 2011a). Consequently, further research needs to examine the potential effects of pharmacological exposure to nicotine from SHS on adolescent smoking.

Objectives of the Present Research Program

To convincingly contend that pharmacological exposure to nicotine has the potential to predict smoking behavior uniquely, researchers ought to use biomarkers and/or passive nicotine monitors while statistically controlling for social exposure to smoking. Previously reported studies (Becklake et al., 2005; Okoli, Rayens, & Hahn,

2007) provide compelling, preliminary evidence that pharmacological exposure to nicotine may confer greater risk for smoking behavior due to the psychoactive effects of nicotine. Thus, it could be plausible that both pharmacological exposure to SHS and social exposure to smoking contribute to smoking initiation among adolescents via their differential effects on smoking precursors.

Adolescent never-smokers constitute an ideal population to study when considering pharmacological exposure to nicotine through SHS as a risk factor for smoking initiation. First, estimates of nicotine exposure are not biased by active smoking, given that they reflect nicotine exposure through passive smoking exclusively. Second, investigating risk for smoking initiation enables researchers to identify which adolescent never-smokers are more likely to initiate smoking and which adolescent never-smokers are less likely to initiate. Importantly, there is a consistent literature which agrees upon variables that are precursors to smoking during adolescence. Examples of such precursors include distinct theoretical concepts, such as smoking expectancies (Hine, Summers, Tilleczek, & Lewko, 1997), smoking susceptibility (Pierce, Choi, Gilpin, Parkas, & Merritt, 1996), and ND symptoms (Bélanger et al., 2008; O'Loughlin et al., 2009). Third, researchers can evaluate whether pharmacological exposure and social exposure to smoking differentially affect the precursors.

Study 1 Aim. The literature pertaining to ND symptoms has reported the unexpected finding that never-smoking adolescents self-report ND symptoms (e.g., "feeling mentally addicted to cigarettes", "feeling like one really needs a cigarette"); this finding has been found across independent samples (e.g., Bélanger et al., 2008; DiFranza et al., 2000; Okoli, Richardson, Ratner, & Johnson, 2009; Prokhorov, Hudmon,

Cinciripini, Marani, 2005; Racicot et al., 2011a). Importantly, endorsing ND symptoms has been associated prospectively with smoking a first puff of cigarette and daily smoking (O'Loughlin et al., 2009), indicating that ND is a risk factor for smoking behavior during adolescence. However, there is a dearth of research investigating the reasons why adolescents who have never smoked any cigarettes endorse items pertaining to ND. Specifically, this scientific discovery is highly incompatible with current conceptualization of ND, which postulates that nicotine use must occur daily (APA, 2000). Exposure to smoking has been hypothesized as one of the mechanisms by which never-smoking adolescents develop ND symptoms. To bridge gaps in scientific research, the aim of Study 1 was to identify potential predictors of ND symptoms in a sample of never-smoking adolescents, using longitudinal data.

STUDY 1:

Predictors of nicotine dependence symptoms among never-smoking adolescents: A longitudinal analysis from the Nicotine Dependence in Teens Study

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ABSTRACT

Background: Recent cross-sectional studies suggest some adolescents who have never smoked cigarettes experience nicotine dependence (ND) symptoms and that exposure to second-hand smoke, social exposure to smoking, and alcohol use are plausible correlates. The aim of this study was to replicate and extend these findings by investigating possible predictors of ND symptoms longitudinally.

Method: Participants included 847 secondary school students who had never smoked cigarettes enrolled in the Nicotine Dependence in Teens Study. Adolescents completed self-report questionnaires measuring smoking status, ND symptoms, and risk factors for ND in smokers (i.e., socio-demographic indicators, social exposure to smoking, psychosocial indicators, and substance use) in 20 survey cycles from 7-11th grade. Generalized estimating equations, which account for repeated measures within individuals, were used to test the predictors of ND symptoms.

Results: Consistent with previous research, 7.88% of never-smokers across all cycles endorsed at least one ND symptom. Younger age ($p \leq .001$), country of birth ($p \leq .05$), peer smoking ($p \leq .001$), teacher smoking ($p \leq .05$), depression ($p \leq .05$), stress ($p \leq .001$), lower self-esteem ($p \leq .05$), impulsivity ($p \leq .05$), and alcohol use ($p \leq .001$) predicted greater ND symptoms in multivariable modeling.

Conclusions: Replicating previous cross-sectional findings, peer smoking and alcohol use predicted ND symptoms among never-smoking adolescents. Extending these findings, previous predictors only observed among ever-smokers, including socio-demographic and psychosocial indicators, also predicted ND symptoms. This longitudinal investigation demonstrated the temporal relation of the predictors preceding

ND symptoms. Future research should consider longer prospective studies with younger children to capture early onset of ND symptoms and with longer follow-up to detect eventual smoking uptake.

1. Introduction

Nicotine dependence (ND) is defined by symptoms of withdrawal, tolerance, and difficulty controlling tobacco use during a 12-month period (American Psychiatric Association & American Psychiatric Association Task Force on DSM-IV [APA], 2000). According to this clinical conceptualization, daily smoking is a requisite criterion for its diagnosis. However, this notion has been challenged by research suggesting that ND can be reported not only soon after smoking initiation, but also *before* initiation. The aim of the present study was to identify predictors of ND symptoms in a longitudinal sample of adolescent never-smokers.

Early reports of ND symptoms have been observed among ever-smokers. DiFranza et al. (2000) found that 22% of adolescents experienced ND symptoms within the first month following consumption of at least one cigarette, with 6% reporting at least one symptom in the first two weeks. Surprisingly, a small percentage of never-smokers endorsed "really needing a cigarette" (2.5%) and "having strong cravings to smoke" (1.7%). Following smoking initiation (0 mos), 20% of adolescent smokers reported mental addiction (2.5 mos), cravings (4.5 mos), physical addiction (5.4 mos), withdrawal symptoms (11.0 mos) and tolerance (13.0 mos), well before the onset of weekly smoking (19.4 mos) and the development of ICD-10 dependence (40.6 mos; Gervais, O'Loughlin, Meshefedjian, Bancej, & Tremblay, 2006). Such findings provide convincing evidence that ND symptoms can be reported early in the course of smoking. Subsequent research examined risk factors during adolescence associated with early reports of ND among smokers.

1.1. Predictors of ND Symptoms during Adolescence in Smoking Populations

Social exposure to smoking by significant others during adolescence is associated not only with smoking behavior (O'Loughlin, Karp, Koulis, Paradis, & Difranza, 2009), but also with ND. Parental smoking (Brook, Saar, Zhang, & Brook, 2009; Kleinjan et al., 2012) and parental ND (Hu, Griesler, Schaffran, & Kandel, 2011) during adolescence were found to predict ND in adolescent and adult smokers. Further, sibling smoking and peer smoking predicted ND in adolescent smokers (Audrain-McGovern et al., 2007; de Leeuw, Engels, Vermulst, & Scholte, 2009; Hu et al., 2011; Wileyto et al., 2009).

Psychosocial indicators have also been identified as risk factors for ND among smoking adolescents. Depressed mood and novelty-seeking predicted ICD-10 ND and loss of autonomy over tobacco use (DiFranza et al., 2007; Karp, O'Loughlin, Hanley, Tyndale, & Paradis, 2006). Moreover, externalizing behavior problems (Hu et al., 2011; Storr, 2008) have been identified as risk factors, whereas impulsiveness was inversely associated with ICD-10 ND (DiFranza et al., 2007). With respect to substance use, alcohol has been found to predict ND among smoking adolescents (Storr, 2008; Wileyto et al., 2009).

In addition to identifying risk factors for ND, growth-modeling studies demonstrate that ND emerges through longitudinal trajectories during adolescence (Hu, Muthen, Schaffran, Griesler, & Kandel, 2008; Kleinjan et al., 2010; Kleinjan et al., 2012). Trajectories are based on distinct profiles (e.g., severity, timing, symptoms). Factors predicting trajectory membership include conduct disorder, parental ND, novelty-seeking (Hu et al., 2008), parental and peer smoking, and depression (Kleinjan et al., 2010). Taken together, social exposure to smoking, psychosocial risk factors, and

substance use have been found to predict ND and trajectory membership in adolescent smokers.

1.2. ND Symptoms during Adolescence in Tobacco-Naïve Populations

Extant literature demonstrates ND symptoms can be reported not only by smokers, but also by never-smokers. It is plausible that second-hand smoke exposure (SHS) explains this unexpected and intriguing observation. Prokhorov, Hudmon, Cinciripini, and Marani (2005) found that the prevalence of 5 of 7 withdrawal symptoms was similar in never- and former smokers. Bélanger et al. (2008) reported 4.6% of never-smoking 5th graders endorsed at least one ND symptom, and SHS exposure in cars (*Hedges g*=.09), sibling smoking (*g*=.14), and peer smoking (*g*=.10) were associated with ND symptoms; parental smoking was not associated (*g*=.06). Racicot, McGrath, and O'Loughlin (2011a) found that the number of smokers among parents, siblings, and peers (*g*=.16) predicted ND symptoms in adolescent never-smokers. Moreover, Racicot, McGrath, and O'Loughlin (2011b) found 6.2% of never-smokers endorsed at least one ND symptom at baseline. Alcohol use (*g*=.11) and peer smoking (*g*=.07) were associated with ND symptoms; parental (*g*=.02) and sibling smoking (*g*=.02) were not associated. Relatedly, never-smoking adolescents reporting ND symptoms have an increased likelihood of smoking susceptibility (Okoli, Richardson, Ratner, & Johnson, 2009) and smoking initiation (O'Loughlin et al., 2009). Taken together, there is emerging evidence that never-smokers endorse ND symptoms, that smoke exposure itself predicts which never-smokers will endorse these symptoms, and that ND symptoms are a risk factor for eventual initiation.

To date, cross-sectional data indicate ND has been observed in never-smoking populations, and social exposure to smoking and substance use are correlates of ND symptoms. Given that ND predicts smoking susceptibility and initiation, identifying predictors of ND symptoms among never-smokers warrants further investigation. The current objective was to identify predictors of ND symptoms in a school-based, longitudinal sample of adolescents who had never smoked. Potential predictors were selected based on previously demonstrated associations with ND in adolescent smokers and included socio-demographic indicators, social exposure to smoking, psychosocial indicators, and substance use.

2. Method

2.1. Procedure and Participants

Nicotine Dependence in Teens (NDIT) is a longitudinal cohort of 1293 7th grade students, aged 12-13 years at baseline, designed to investigate the onset and development of cigarette smoking and ND. Students were recruited in a convenience sample of 10 public schools in Montréal (Québec, Canada) selected in partnership with school boards and principals. To maximize representativeness, schools were purposely selected from urban, suburban, and rural settings, as well as low, moderate, and high socioeconomic districts. Data were collected in 20 survey cycles from 1999 to 2005 (4 per school year from grade 7 to 11). Self-report questionnaires were administered at school in the language of instruction (i.e., English or French). All participants provided assent; informed parental consent was obtained in signed consent forms. NDIT received ethics approval from the *Centre de recherche du CHUM* (#ND06.087).

2.2. Measures

2.2.1. Smoking Status. Smoking status was assessed at each survey cycle using two items: "Have you ever in your life smoked a cigarette, even just a few puffs?" (*No* to *Yes, more than 10 times*) and "Check the one box that describes you best…" (*I have never smoked a cigarette, even just a few puffs* to *I smoke cigarettes every day*). Never-smoking was defined conservatively as having never smoked a cigarette, not even a few puffs.

2.2.2. ND Symptoms. ND symptoms were assessed with items adapted from an ND/craving symptom indicator (O'Loughlin, DiFranza, et al., 2002). Adolescents rated four items on a 4-point scale: "When you see other kids your age smoking cigarettes, how easy is it for you not to smoke?" (*Very easy* to *Very difficult*); "How often have you felt like you really need a cigarette?" (*Never* to *Often*); "How physically addicted to smoking cigarettes are you?" (*Not at all* to *Very*); and "How mentally addicted to smoking cigarettes are you?" (*Not at all* to *Very*). The original ND/craving symptom indicator was based on a sample of smoking adolescents and evidenced excellent internal reliability (Cronbach's α=.94), test-retest reliability (ICC=.91), and convergent validity with the Hooked on Nicotine Checklist (r=.91) and ICD-10 (r=.82). The adapted items were those four answered by never-smokers; principal components analysis revealed the original component structure was retained (i.e., all items loaded on one component; all loadings >.6). Consistent with previous scoring schemes (Bélanger et al., 2008; Racicot et al., 2011b), items were summed to yield a composite score (range 0-12). Prevalence data are estimated for those who endorse at least one ND symptom (i.e., non-zero score).

2.2.3. Socio-demographics. Socio-demographic data included age, sex, language spoken at home, country of birth, parental education, and perceived family income.

2.2.4. Social Exposure to Smoking

2.2.4.1. Adult Smoking. Adolescents indicated whether adults residing in their household smoked cigarettes, based on a list of 10 family members (e.g., mother, father, aunt). The response categories were summed to yield the total number of smoking adults (range 0-10).

2.2.4.2. Sibling Smoking. Adolescents reported how many siblings, including step- or half-siblings, smoked. The response categories were summed to yield the total number of smoking siblings.

2.2.4.3. Peer Smoking. Adolescents answered, "How many of the friends whom you usually hang out with smoke cigarettes?" using a 5-point scale (*None* to *Most or all*).

2.2.4.4. Schoolmate and School Personnel Smoking. Adolescents answered, "I see students smoke near the school" using a 3-point scale (*Not at all true* to *Very true*). A second question was asked about teachers/school staff.

2.2.5. Psychosocial Indicators

2.2.5.1. Depression. Depression was measured with the six-item Mellinger Depressive Symptoms Scale (Kandel & Davies, 1982). Adolescents rated items over the past 3 months using a 4-point scale (*Never* to *Often*). Items are summed to create a total score (range 0-18); higher scores indicate greater depression. This measure evidences good internal consistency (α=.89, Chaiton, Cohen, O'Loughlin, & Rehm, 2010; α=.85, present study).

2.2.5.2. Stress. Stress was measured using a list of stressful life events typically encountered during adolescence (e.g., breaking up with girlfriend, parental divorce; Deschenes, 1997). Adolescents rated whether they were worried or stressed about 15

items over the past 3 months on a 4-point scale (*Not at all* to *A whole lot*). Items are summed to create a total score (range 0-45); higher scores indicate greater stress. This scale has good internal consistency (α=.83-.89, Deschenes, 1997; α=.79, present study).

2.2.5.3. Perceived Academic Performance. Adolescents rated their academic performance in response to the question, "I'm not doing well at school" on a 3-point scale (*Not at all true* to *Very true*).

2.2.5.4. Self-Esteem. Self-esteem was measured using Rosenberg's Self-Esteem Scale (1965; Vallières & Vallerand, 1990). Adolescents rated nine items over the past 3 months on a 3-point scale (*Not at all true* to *Very true*). Items are summed to create a total score (range 0-18); higher scores indicate higher self-esteem. This scale displays good test-retest reliability (*r*=.84) and internal consistency (α=.88, Vallières & Vallerand, 1990; α=.80, present study).

2.2.5.5. Novelty-Seeking. Novelty-seeking was assessed using nine items based on Cloninger's Tridimensional Personality Questionnaire (e.g., "When nothing new is happening, I usually start looking for something that is exciting"; Cloninger, 1987) rated on a 5-point scale (*Not at all true* to *Very true*). Items are summed to yield a total score (range 0-36); higher scores indicate greater novelty-seeking. This scale has good internal consistency (α=.77, Wills, Windle, & Cleary, 1998; α=.81, present study).

2.2.5.6. Impulsivity. Impulsivity was assessed with an abbreviated version of the Eysenck Impulsivity Scale (Eysenck & Eysenck, 1977), which has been previously validated with adolescents (Wills et al., 1998). Adolescents rated seven items on a 5-point scale (*Not at all true* to *Very true*). Items are summed to yield a total score (range 0-28); higher scores

indicate greater impulsivity. This measure evidences good internal consistency (α=.87, Wills et al., 1998; α=.87, present study).

2.2.6. Substance Use. Frequency of other tobacco products and alcohol use in the past three months was assessed separately with two items ("smoke cigar or cigarillo", "drink beer, wine or hard liquor") using a 5-point scale (*Never* to *Usually every day*).

2.3. Data Analysis

The initial sample was comprised of never-smokers at cycle 1. At each subsequent survey cycle, smoking status was ascertained to verify whether participants still met the never-smoking inclusion criterion. Participants were excluded at the given cycle that they reported ever-smoking and every cycle thereafter. For example, a participant reporting smoking for the first time at cycle 5 would be included from cycles 1-4, but excluded from cycles 5-20. Participants whose smoking status was unknown at any follow-up cycle were similarly excluded. This conservative criterion was used to ensure the sample was exclusively never-smokers.

Given the design of NDIT, some questions were asked at every cycle, while others were measured less frequently (detailed study design provided in O'Loughlin et al., 2009). Missing values on questions that were asked once or 2-3 times were substituted in two steps. First, for questions measured only once, the same value was imputed for all cycles. For variables measured 2-3 times, the value from the last available observation was carried backwards. Second, multiple imputation was used for all the remaining missing observations, which included missing values on questions asked at each cycle, questions asked once or 2-3 times, and cycles that had been skipped. Missing values were imputed 20 times in Amelia II (Honaker, King, & Blackwell, 2012).

Generalized estimating equations (GEE), with the independent correlation structure, were employed to account for repeated measures within participants. Given that ND symptoms were coded as a continuous score, analyses were conducted with linear regression modeling. Using an analytic strategy similar to that in earlier NDIT analyses (O'Loughlin et al., 2009), potential predictors at each cycle were used to predict ND symptoms at the subsequent cycle (i.e., T1 variables to predict ND at T2, etc.). This approach was utilized so predictors closest in time (3-4 months prior) would predict ND symptoms. Data for all participants and cycles were pooled, in accordance with the pooling of repeated observations method (e.g., Cupples, D'Agostino, Anderson, & Kannel, 1988; D'Agostino et al., 1990; Karp, Abrahamowicz, Bartlett, & Pilote, 2004).

Univariate and multivariable regression analyses were conducted separately for each potential predictor to evaluate its association with ND symptoms at the next cycle. All multivariable analyses were age- and sex-adjusted; additional covariates were included in the model if the correlation coefficient between the covariate and the potential predictor was ≥.20 (Hosmer & Lemeshow, 2000). Unstandardized beta coefficients, which are less affected by arbitrary features of the study design and population (Greenland, Schlesselman, & Criqui, 1986), were derived with their estimates of standard error and the corresponding Wald χ^2 test statistic.

3. Results

3.1. Descriptive Statistics

Among the entire NDIT cohort, adolescents participated in 16.3 survey cycles (*SD*=5.91). Of these, 847 participants were never-smokers at baseline (Table 1). Compared to ever-smokers (*n*=446; M_{age}=12.99 yrs, *SD*=.73), never-smokers were

younger, t=9.59, p<.001, more likely to be born outside Canada, χ^2=6.76, p<.01, attended an English-language school, χ^2=75.88, p<.001, and spoke English at home, χ^2=104.34, p<.001. There were no sex differences, χ^2=3.04, p=.08. A total of 405 participants reported ever smoking and were excluded at that corresponding cycle. The prevalence of reporting at least one ND symptom among never-smokers, across all cycles, was 7.88% (*SD*=1.98; range 4.8-13.0%). The attrition rate did not differ between never-smokers and ever-smokers, 25% over 20 cycles; t=1.13, p=.81.

3.2. Predictors of ND Symptoms

Univariate and multivariable regression models are presented in Table 2. In multivariable modeling, among the socio-demographic variables, younger age and being born in Canada were associated with greater ND symptoms, while controlling for covariates. Other socio-demographic variables including sex, language spoken at home, family income, and parental education were not associated in the multivariable models. Among the social smoke exposure indicators, observing peer and teacher smoking were associated with greater ND symptoms, after controlling for covariates. Living with adult smokers, having siblings who smoke, and being exposed to schoolmate smoking were not associated in the multivariable models. Among the psychosocial indicators, higher self-reported depression, stress, and impulsivity, as well as lower self-esteem were associated with greater ND symptoms, while controlling for covariates. Novelty-seeking and perceived academic performance were not associated in the multivariable models. Finally, among the substance use indicators, more frequent alcohol use was associated with greater ND symptoms, while controlling for covariates. Cigar/cigarillo use was not associated.

4. Discussion

Accumulating evidence suggests ND symptoms can occur soon after smoking initiation (e.g., DiFranza et al., 2007; Gervais et al., 2006). Intriguingly, cross-sectional studies evidence that 4-6% of never-smoking adolescents endorse items measuring ND (e.g., Bélanger et al., 2008; Racicot et al., 2011b). The objective of this study was to corroborate and extend previous cross-sectional findings by identifying predictors of ND symptoms longitudinally among adolescents who had never smoked a cigarette, not even a few puffs. In the present study, the prevalence of never-smokers endorsing ND symptoms (7.8%) was similar to past reports. Consistent with findings in adolescent smokers (e.g., Audrain-McGovern et al., 2007; de Leeuw et al., 2009; Hu et al., 2011), socio-demographic indictors, social exposure to smoking, psychosocial indicators, and substance use predicted ND symptoms in never-smokers.

Age and country of birth were the socio-demographic indicators associated with ND symptoms. Age was inversely associated to ND. Higher ND symptoms were observed in the early survey cycles when adolescents were younger. Relatedly, adolescents who were more likely to endorse ND symptoms commenced smoking earlier, and consequently, were excluded at a younger age. This phenomenon is referred to as the "depletion of susceptibles" (Garbe & Suissa, 2005; Karp et al., 2006) whereby in a longitudinal cohort of adolescent never-smokers, those who initiate smoking are excluded from the sample. Among participants who began smoking during follow-up, a higher proportion was censored in earlier survey cycles compared to later cycles. Of note, the prevalence of adolescents who tried smoking is consistent with national estimates of smoking initiation among youth attending 10-12th grades (47.8% vs. 48.2%, respectively;

Health Canada, 2008). Previous research has demonstrated that ND symptoms predict smoking uptake in adolescents (O'Loughlin et al., 2009). Further, attrition does not account for the observed age finding; never- and ever-smokers in the entire NDIT cohort were followed for the same number of survey cycles. The other socio-demographic indicator, country of birth, was also associated. Foreign-born participants obtained lower ND symptoms scores, regardless of language spoken at home. It is possible that Canadian-born participants were raised in a more "pro-smoking" culture.

Social exposure to smoking is a risk factor for ND symptoms in adolescents (e.g., Bélanger et al., 2008; Brook et al., 2009; de Leeuw et al., 2009). Current findings corroborated this association as smoking by friends (g=.19) predicted ND symptoms. Parental (g=.03) and sibling (g=.01) smoking were not associated. This result is consistent with previous studies showing that the relative influence of family members and friends varies by age, with family members having a greater influence in childhood and friends having a greater influence in adolescence (e.g., Vitaro, Wanner, Brendgen, Gosselin, & Gendreau, 2004). Social contagion theory (Rowe, Chassin, Presson, Edwards, & Sherman, 1992) may explain the observation that never-smokers come to believe that they (should) experience cravings when friends talk openly about their cravings. To our knowledge, smoking by school personnel has never been evaluated as a risk factor for ND among never-smokers. Implementing and enforcing smoke-free policies in schools should be emphasized as a strategy to prevent the development of risk factors for smoking initiation (Barnett et al., 2007).

Among psychosocial indicators, depression, stress, self-esteem, and impulsivity predicted ND symptoms. Affect control, boredom reduction, and greater social benefits

are consequences that never-smoking adolescents expect from smoking cigarettes (Hine, Honan, Marks, & Brettschneider, 2007). Negative affect may predispose adolescents to perceive ND symptoms and expect smoking will help curb unpleasant emotions. Foster, Racicot, McGrath (2012) found never-smoking adolescents with clinical levels of impulsivity were more likely to believe smoking controls affect and reduces boredom. These adolescents are at increased risk of initiating smoking. In adolescent smokers, smoking expectancies have been found to predict eventual ND (Heinz, Kassel, Berbaum, & Mermelstein, 2010).

When examining psychoactive substances other than cigarettes, alcohol consumption predicted ND symptoms (g=.12), which coincides with past findings (Racicot et al., 2011b). This association was probable, given that smoking and alcohol use frequently co-occur (Jackson, Sher, & Wood, 2000). Alcohol use confers greater risk for ND in recent onset adolescent smokers (Dierker, Rose, Donny, & Tiffany, 2011). Relatedly, alcohol use has been found to increase tolerance to nicotine in mice (Collins, Burch, de Fiebre, & Marks, 1988).

4.1. Limitations

There were three limitations of the current study. First, data were self-reported. Although misclassification is possible, self-report is systematically used to measure adolescent smoking behavior and it is reliable (Eppel, O'Loughlin, Paradis, & Platt, 2006). Future studies should use biomarkers to cross-validate smoking status. Second, schools were selected via convenience sampling, which may limit the generalizability of the results. However, school selection was stratified by population density (i.e., rural, suburban, urban) and socioeconomic status (i.e., low, medium, high), as reported by

school boards, to maximize external validity. Third, the non-experimental design of NDIT makes causal inference challenging; however, using prospective data permitted the evaluation of temporal relationships between ND symptoms and potential predictors.

4.2. Strengths

Strengths of this study also merit consideration. First, the analytical sample (*N*=847) included a large number of adolescents recruited in secondary schools. Given that adolescence is the developmental stage when individuals are most likely to experiment with tobacco (Chassin, Presson, Pitts, & Sherman, 2000), this period was optimal for evaluating risk factors for ND. Second, the measurement definitions of ND and never-smoking status were rigorous. Items measuring ND have been shown to possess strong psychometric properties (Bélanger et al., 2008; O'Loughlin, DiFranza, et al., 2002). Never-smoking status was defined conservatively to ensure that participants had never smoked cigarettes, not even a puff. Third, ND predictors were derived from validated, standardized scales, which facilitates comparisons across studies. The wide range of predictors were classified into four categories and investigated to depict a multifaceted analysis of ND symptoms.

4.3. Conclusions

The unexpected phenomenon of ND symptoms among never-smokers has been repeatedly observed (cf., Bélanger et al., 2008; DiFranza et al., 2000; O'Loughlin et al., 2009; Okoli et al., 2009; Prokhorov et al., 2005; Racicot et al., 2011a, 2011b). This longitudinal study replicated previous cross-sectional findings, as peer smoking and alcohol use predicted ND symptoms among never-smoking adolescents. Extending these findings, additional predictors of ND, previously only observed among ever-smokers,

were identified including socio-demographic indicators (i.e., age, country of birth) and psychosocial indicators (i.e., depression, stress, self-esteem, impulsivity). Further, this longitudinal investigation established temporality of exposure whereby indicators preceded endorsement of ND symptoms at subsequent time-points. These findings contribute new knowledge regarding the emergence of ND symptoms in never-smoking adolescents. The question remains whether adolescents correctly understand the content of these items; however, qualitative work suggests that adolescents conceptually distinguish physical addiction ("like an empty spot in the chest") from mental addiction ("I think I can only feel it in my head"; O'Loughlin, Kishchuk, DiFranza, Tremblay, & Paradis, 2002, p. 205). Further psychometric development of the assessment of ND symptoms in young never-smokers should consider whether individual items are differentially weighted, using an item-response theory framework (e.g., MacPherson, Strong, & Myers, 2008), across childhood and adolescence.

Converging evidence with animal data provide additional support for the hypothesis that active smoking is not required to experience ND symptoms and suggest neuroadaptations account for somatic withdrawal effects observed in nicotine-naïve rats exposed to SHS (Yamada et al., 2010). Based on the Sensitization-Homeostasis model (DiFranza & Wellman, 2005), nicotine exposure through SHS could prime the addiction pathway and lead to experiencing ND symptoms (Bélanger et al., 2008). Recent findings support this notion of pharmacological priming. Brody and colleagues (2011) showed that SHS exposure leads to $\alpha 4\beta 2^*$ nicotinic cholinergic receptor occupancy in non-smokers, which parallels findings of withdrawal sensations observed in tobacco-naive rats exposed to SHS (Small et al., 2010). Relatedly, it is unclear whether

pharmacological exposure to nicotine may partly explain established findings with social modeling. Biomarkers of nicotine and SHS exposure could be used to empirically evaluate pharmacological priming as a predictor of ND symptoms.

Future research should investigate why some participants reporting ND symptoms convert to smoking, whereas others may be resilient to smoking uptake despite endorsing ND symptoms. Longer prospective studies are strongly recommended to capture both early endorsement of ND symptoms and late smoking onset. In the present sample, 30% who converted to ever-smoking status started smoking within the first three cycles. While the current longitudinal study started in 7th grade, it may be prudent to begin investigating the emergence of ND symptoms in elementary school. Some adolescents may endorse ND symptoms and resist smoking uptake due to protective factors (e.g., extracurricular activities, good relationship with parent; DiFranza et al., 2007), genetic differences in nicotine metabolism (Malaiyandi, Sellers, & Tyndale, 2005), or possibly perceived risk of SHS exposure (Song, Glantz, & Halpern-Felsher, 2009). Previous research has suggested there may be a class of "late bloomers" who endorse ND symptoms, but take a longer period of time before trying smoking (Karp, O'Loughlin, Paradis, Hanley, & Difranza, 2005). Thus, follow-up periods extending into young adulthood may be warranted to accurately detect eventual smoking uptake. Prospective studies including additional covariates and employing other analytic strategies (e.g., latent growth curve modeling) could help further address this question. Prevention programming should consider ND a novel predictor for adolescent smoking behavior, given that ND predicts smoking susceptibility (Okoli et al., 2009) and smoking initiation (O'Loughlin et al., 2009).

Table 1
Descriptive Statistics of ND Symptom Predictors at Survey Cycles 1, 10, and 19

Predictor	Cycle 1 M (SD) or % *N* = 847	Cycle 10 M (SD) or % *N* = 476	Cycle 19 M (SD) or % *N* = 325
Socio-demographics			
- Age (years)	12.7 (.5)	14.8 (.4)	17.0 (.4)
- Female sex (%)	49.8%	46.6%	46.5%
- Language spoken at home (%)			
English	58.2%	59.2%	58.8%
French	22.4%	21.6%	20.3%
English and French	10.4%	9.0%	9.2%
Other	9.0%	10.1%	11.7%
- Born in Canada (%)	91.0%	92.0%	91.7%
- Family income (% better)	10.8%	18.7%	19.7%
- Parental education (years)	14.5 (1.4)	14.6 (1.7)	14.6 (2.0)
Social Smoke Exposure			
- Adult smoking score (0 – 10)	.5 (.8)	.3 (.6)	.3 (.5)
- Sibling smokers (number)	.2 (.5)	.2 (.6)	.2 (.5)
- Peer smokers (% none)	79.5%	43.3%	32.6%
- Schoolmates seen smoking (% very true)	89.9%	83.6%	82.5%
- Teachers seen smoking (% very true)	18.3%	20.4%	18.8%
Psychosocial Indicators			
- Depression score (0 – 18)	5.9 (3.3)	5.1 (4.3)	6.2 (4.7)
- Stress score (0 – 45)	4.4 (3.9)	4.0 (4.6)	5.0 (5.1)
- Poor academic performance (% very true)	59.9%	63.5%	64.9%
- Self-esteem score (0 – 18)	14.4 (2.5)	14.6 (3.2)	14.9 (3.2)
- Novelty-seeking score (0 – 36)	15.8 (5.2)	15.8 (6.7)	15.5 (6.7)
- Impulsivity score (0 – 28)	7.8 (4.2)	7.7 (5.3)	7.7 (5.7)
Substance Use			
- Cigar or cigarillo use (% never)	99.4%	99.2%	96.9%
- Alcohol use (% never)	67.1%	56.3%	42.8%

Note. M = mean. SD = standard deviation.

Table 2
GEE Models Predicting ND Symptoms in Adolescent Never-smokers

	Univariate			Multivariable			
Predictor	b	SE	Wald χ^2	b	SE	Wald χ^2	Covariates
Socio-demographics							
- Age	-.02	.01	12.31***	-.04	.01	40.84***	Peer smoking, alcohol
- Sex							Depression, stress
Female	REF			REF			
Male	.08	.03	8.35**	.01	.02	0.11	
- Language at Home			1.47			1.80	Country of birth,
English	REF			REF			parental education
French	-.02	.03	.49	-.01	.03	.05	
English and French	-.03	.03	1.04	-.02	.03	.39	
Other	.02	.04	.12	.07	.07	1.11	
- Country of Birth							Language at home
Canada	REF			REF			
Other	-.08	.03	7.71**	-.11	.04	6.04*	
- Family income	.04	.04	.91	-.03	.04	.48	Stress, self-esteem
- Parental education	.01	.01	.41	.01	.01	.57	Language at home, adult smoking
Social Smoke Exposure							
- Adult smoking	.02	.02	.88	.02	.02	1.25	Parental education
- Sibling smoking	.01	.02	.13	.01	.02	.09	
- Peer smoking	.09	.02	24.55***	.09	.02	31.10***	Alcohol
- Schoolmate smoking	.00	.04	.00	-.02	.04	.43	Teacher smoking
- Teacher smoking	.03	.02	2.12	.03	.02	3.73*	Schoolmate smoking
Psychosocial Indicators							
- Depression	.02	.00	36.77***	.01	.00	5.15*	Stress, self-esteem
- Stress	.03	.00	29.41***	.02	.01	13.19***	Depression, self-esteem, impulsivity, family income
- Perceived Academic Performance	-.05	.02	6.41*	-.01	.02	.27	Self-esteem
- Self-esteem	-.02	.01	16.71***	-.02	.01	6.22*	Academic performance, family income, stress, depression
- Novelty-seeking	.01	.00	16.06***	.00	.00	.03	Impulsivity
- Impulsivity	.01	.00	19.59***	.01	.00	5.28*	Novelty-seeking, stress
Substance Use							
- Cigar or cigarillo	.11	.08	1.82	.10	.09	1.30	Alcohol
- Alcohol	.05	.01	12.77***	.04	.01	11.29***	Peer smoking

Note. All multivariable models also included age and sex as covariates.
b = unstandardized beta coefficient. SE = standard error. REF = reference category.
* $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

TRANSITION TO STUDY 2

The purpose of Study 1 was to further our understanding of the development of ND symptoms among never-smokers, which is, by definition, inconsistent with the current conceptualization of ND (APA, 2000). Informed by the determinants of ND among smokers, we tested socio-demographic indicators, social exposure to smoking, psychosocial indicators, and substance use as longitudinal predictors of ND symptoms in a sample of never-smoking adolescents. Peer smoking, a measure of social exposure to smoking, emerged as one of the strongest significant predictors of ND symptoms; this is congruent with previously published cross-sectional studies demonstrating that peer smoking (Bélanger et al., 2008) and number of smokers among family members and friends (Racicot et al., 2011a) predict ND symptoms. Despite these data providing support for the link between social exposure to smoking and endorsement of ND symptoms, the pathway by which they are associated is not fully understood.

Other researchers have debated the relation between social exposure to smoking and ND symptoms, contending that social exposure should not be associated with ND among never-smokers, as the latter phenomenon is impossible conceptually. As such, they hypothesized that our measures of social exposure raised the possibility that adolescents learned to believe they were dependent on nicotine by observing smokers, without being "truly" dependent. Relatedly, they recommended that our measures be improved to evaluate the relationship between social exposure and ND symptoms more validly.

To test the underlying contribution of social exposure to ND symptoms, we evaluated the content of the variables measuring parental, sibling, and peer smoking. This

critical analysis led us to the conclusion that our measures of social exposure to smoking focused exclusively on the number of smokers among adult family members, siblings, and friends. Other measures of social exposure to smoking found in the literature typically only assess the prevalence of other smokers (e.g., yes/no for mother/father, siblings, peers); the relative frequency of their smoking behavior (e.g., not at all, occasionally, regularly, very often; Vitaro et al., 2004); or the ordinal proportion of smokers among parents, siblings, or peers (e.g., all, more than half, less than half, hardly anybody; de Vries, Engels, Kremers, Wetzels, & Mudde, 2003). Thus, these existing measures help determine the identity and the number of smokers in adolescents' social environment without providing additional information about the situational contexts in which these smokers use cigarettes. This distinction has potential implications for adequately investigating the relation between social exposure to smoking and ND symptoms among never-smokers. As an example, one may question whether it is the number of friends who smoke that essentially leads to the development of ND symptoms. Alternatively, one may question whether observing friends smoking under specific circumstance (e.g., smoking at school when anxious and talking openly about cravings) is, in fact, what better explains the onset of ND symptoms in a population of never-smokers. Extending this line of reasoning, could it be possible that reports of ND symptoms are situation-specific, whereby never-smoking adolescents experience ND symptoms only in the same situations where they observed their friends smoke, but not in other situations? Traditional measures of social exposure to smoking, which largely focus on smokers' identity ("who"), would be unable to address that research question.

Social Learning Theory (Bandura, 1977) has often been cited as the predominant theoretical framework to explain why observation of smokers in one's environment is a predictor of smoking. Specifically, it is posited that adolescents are more likely to imitate the behavior of individuals whom they view as role models, like their parents, siblings, or friends. Family members and friends who smoke typically consume cigarettes in a variety of contexts, including specific places (e.g. living room, car), under specific circumstances (e.g., with others, following meals), and in specific mood states (e.g., relaxed, bored; Van Gucht, Van der Bergh, & Vansteenwegen, 2010). However, the majority of research has focused almost exclusively on who the smokers are, but has neglected to consider the contextual situations in which adolescents observe smokers use cigarettes, that is, the "where" and "when". The cue-reactivity literature (cf. Carter & Tiffany, 1999) has evidenced that environmental cues (i.e., contextual situations) are associated with smoking behavior among smokers (Conklin, 2006). The repercussions of environmental cues in the context of social exposure to smoking remain largely unknown, yet such cues could increase the risks associated with smoking initiation. The development of a new measure permitting assessment of the contextual situations in which youth observe their parents, siblings, and peers smoke would be highly informative.

Thus, measuring social exposure comprehensively is of paramount importance for better understanding the effects of social exposure on smoking precursors and smoking behavior. To reach this goal, the objectives of Study 2 were to a) develop an enhanced measure of social exposure and examine its psychometric properties, and b) compare the predictive ability of this new scale with that of existing measures of social exposure to smoking**.**

STUDY 2

Development and psychometric properties of the Social Smoking Situations (S^3) Scale: An enhanced measure of social exposure to smoking during adolescence

Racicot, S., & McGrath J.J. (Under Review)

ABSTRACT

Existing measures of exposure to smoking, such as number of parents, siblings, or friends who smoke, fail to capture the contexts in which the exposure occurs. This study developed the Social Smoking Situations (S^3) Scale to more precisely measure contextual exposure to smoking during adolescence. Informed by the cue-reactivity literature and using focus groups, items of contextual exposure to smoking were generated for three categories of smokers: parents, siblings, and peers. Participants (N = 761; M_{age} = 15.6, SD = 1.3; 61.4% female) were recruited as part of the AdoQuest Study in Montreal, QC. Principal components analysis was used to identify the component structure of the parental, sibling, and peer versions of the S^3 Scale. S^3 scores were computed subsequently to test their association with smoking behavior and smoking expectancies. Further, S^3 scores were compared with existing measures (i.e., number of smokers) to determine which would emerge as a stronger predictor of smoking behavior and smoking expectancies. Overall, S^3 scores were stronger predictors than existing measures; this finding was consistent across the parent (OR_{avg}: 2.59 vs. 1.36), sibling (OR_{avg}: 3.44 vs. 1.59), and peer (OR_{avg}: 3.89 vs. 1.38) versions. The S^3 Scale is a new psychometrically sound instrument that may provide a more robust measurement of social exposure to smoking during adolescence. Importantly, it has the potential to strengthen prevention programming and intervention efforts aimed at adolescents, as it could depict a more precise portrait of the individual and contextual sources of social exposure to smoking.

INTRODUCTION

Observing smokers in one's social environment is a robust predictor of smoking during adolescence (Brandon, Herzog, Irvin, & Gwaltney, 2004; Collins & Ellickson, 2004; U.S. Department of Health and Human Services, 2012). Observing smoking by parents, siblings, and peers constitute the predominant sources of exposure to smoking, and their influence has been attributed largely to social modeling (cf. Avenevoli & Merikangas, 2003; Hoffman, Sussman, Unger, & Valente, 2006; Kobus, 2003; Simons-Morton & Farhat, 2010; Tyas & Pederson, 1998). Children and adolescents are 2.7 times more likely to initiate smoking if parents smoke, 2.3 times more likely if siblings smoke (Leonardi-Bee, Jere, & Britton, 2011), and 3.3 times more likely if peers smoke (O'Loughlin, Karp, Koulis, Paradis, & DiFranza, 2009).

Social Learning Theory (Bandura, 1977) posits that adolescents initiate smoking by observing and imitating the smoking behavior of role models. In fact, observing smoking by others occurs naturally in the social environment under varied circumstances (Van Gucht, Van der Bergh, Beckers, & Vansteenwegen, 2010). Thus, exposure to smoking constitutes a broader context of observing others and the moments or settings in which they smoke. The majority of research that examines social modeling focuses predominantly on the role models who smoke (i.e., the "who"), but typically fails to consider important contextual situations of exposure to smoking (i.e., the "where" and "when"). For example, two adolescents have exactly the same friends who smoke: the first sees their friends smoking sporadically at parties, whereas the second sees them smoking frequently at school and parties. Existing measures of exposure to smoking would code both adolescents as having the same number of friends who smoke, but

would overlook the different contextual aspects of exposure to peer smoking. Existing measures of exposure to smoking do not adequately capture the contextual situations in which role models smoke, which may themselves contribute to the social modeling phenomenon. Interestingly, another literature has considered contextual situations and how they influence smoking.

The cue-reactivity literature among smokers (cf. Carter & Tiffany, 1999) has identified several contextual situations in which adolescent and adult smokers crave or use cigarettes. Common types of smoking situations studied include: locations (e.g., home, car, bar/restaurant); activities (e.g., doing chores, being "on the go", watching television, eating, drinking alcohol); social contexts (e.g., smoking with others, at a party, with friends); and mood states (e.g., angry, relaxed, bored; cf. Cronk & Piasecki, 2010; Dunbar, Scharf, Kirchner, & Shiffman, 2010; Shiffman et al., 2002; Tagmat, Wolff, Schumann, John, & Thyrian, 2011; Van Gucht et al., 2010). The cue-reactivity framework underscores the pertinence of considering smoking situations when studying tobacco use. Tagmat and colleagues (2011) found that adolescents who experienced stronger urges to smoke in social situations, negative mood states, and routine situations (e.g., waiting for the bus) reported greater daily smoking. Among adult smokers, contextual situations including locations, activities, social contexts, and mood states have been associated with more cravings (Dunbar et al., 2010) and cigarette smoking (Cronk & Piasecki, 2010; Shiffman et al., 2002). Further, Van Gucht and colleagues (2010) reported that adult smokers who had greater nicotine dependence consumed cigarettes in more locations and during more varied activities. Taken together, the effects of smoking situations on smokers have been well established within the cue-reactivity framework.

Interestingly, contextual situations may play a parallel role during the emergence of adolescents' smoking behavior, depending on where and when youth observe family members and peers smoke. More precise measures of exposure to smoking, by whom, where, and in what context may improve our ability to predict adolescent smoking uptake.

The construct of social exposure to smoking encompasses both observing others smoking and the contextual situations in which others smoke. However, existing measures of smoke exposure are limited as they narrowly conceptualize exposure to only the source of exposure ("who"); they fail to capture the richer topography of social exposure to smoking via contextual situations (i.e., “where” and “when”). Thus, there is a need for development of a refined psychometric instrument to more comprehensively assess the construct of social exposure to smoking. It is posited that enhanced assessment of broader contextual situations in which adolescents observe others smoking will yield a more precise measure of social exposure to smoking.

The objective of the present study was to develop the Social Smoking Situations (S^3) Scale to more accurately measure social exposure to smoking among adolescents. The approach was twofold. First, guided by classical measurement theory, the S^3 Scale and its psychometric properties were developed. Specifically, principal components analysis was used in constructing the S^3 Scale, while psychometric properties were evaluated by testing aspects of convergent and concurrent validity. Second, the S^3 Scale was tested against existing measures of exposure to smoking (i.e., prevalence of others smoking) to predict smoking behavior and smoking expectancies. It was hypothesized

that compared to existing measures, the S^3 Scale would more strongly predict smoking behavior and smoking expectancies among adolescents.

METHOD

Development of Item Pool

Contextual situations of social exposure to smoking were defined as "when" and "where" adolescents observe smokers use cigarettes, and "what" the smokers are doing at the time of exposure. The item pool was developed in three stages. First, the cue-reactivity literature was reviewed to empirically inform item development (e.g., Tagmat et al., 2011; Van Gucht et al., 2010). Second, separate focus groups were conducted with adolescents and young adults whereby they identified any situations in which adolescents may observe smoking by their parents, siblings, and peers. Third, independent tobacco experts reviewed the items. The number of unique situations identified was 35 for parents, 43 for siblings, and 40 for peers; thus, we maintained separate versions for parents, siblings, and peers.

Procedure and Participants

Adolescents were recruited in 7^{th}, 9^{th}, and 11^{th} grade as part of the AdoQuest Study, an ongoing prospective investigation evaluating the natural course of the development of smoking behavior in youth (for more information on AdoQuest, please see Bélanger et al., (2008); Racicot, McGrath, & O'Loughlin, (2011a)). AdoQuest received approval from the institutional review boards of McGill University, Concordia University, and *Centre de recherche du CHUM*. Adolescents who received written consent from their parents completed self-report questionnaires, including the parental, sibling, and peer versions of the S^3 Scale. For each contextual situation item, adolescents

were instructed to rate their current level of exposure to smoking using a three-point scale (e.g., "my parents smoke when eating dinner"; 0 = not true, 1 = somewhat true, 2 = very true).

Study 1: Construction of the S^3 Scale

Initial Component Structure and Reduction of the Item Pool

An initial Principal Components Analysis (PCA), using varimax rotation and Kaiser's (1960) criterion (eigenvalue $\geq$ 1), was applied to all items. This approach permitted identification of separate components and examination of internal consistency within each component. Specifically, PCA yielded a four-component solution on the parental version, and five-component solutions for both the sibling and peer versions. To maximize internal consistency within each component, the item pool on each version was reduced to retain the most salient items that would make the scale most parsimonious and practical. Items within each component displaying the highest component loadings and highest Cronbach's alpha were retained. This process led to the retention of 12 parental items, 14 sibling items, and 14 peer items, which comprise the final version of the S^3 Scale.

S^3 Scale: Final Version

PCA with varimax rotation was applied to the final S^3 Scale, which revealed that the component structure was identical to that obtained with the original, unreduced item pool. For the parental version (Table 1), components were labeled: meals (variance = 21.64%; Cronbach's α = .90), social activities (20.49%; α = .87), moods (20.29%; α = .92), and unpleasant states (19.40%; α = .85); internal consistency was excellent (α = .93). For the sibling version, components were labeled: quiet activities (20.11%; α =

.92), social activities (18.68%; α = .89), meals (17.56%; α = .92), moods (17.38%; α = .95), and belongingness (14.01%; α = .90); internal consistency was excellent (α = .92). For the peer version, components were labeled: quiet activities (18.71%; α = .90), moods (18.30%; α = .93), social activities (17.22%; α = .86), at school (16.36%; α = .87), and belongingness (12.88%; α = .83); internal consistency was excellent (α = .93).

Study 2: Psychometric Properties of the S^3 Scale

Measures

S^3 Scores. Items within each component of the parent, sibling, and peer versions were averaged to compute S^3 scores. These scores represent a mean ranging from 0 to 2, whereby higher scores indicate greater contextual exposure to smoking. S^3 scores were also computed using factor scores; results were largely identical (not depicted) and averages were preferred to factor scores for practical reasons.

Existing Measures of Exposure to Smoking. Number of parents who smoked and lived in the same household as the adolescents was assessed with four separate questions: "Does your biological mother / biological father / step-mother / step-father currently smoke cigarettes?" Smoking by stepparents was included, as it influences adolescent smoking (Fidler, West, van Jaarsveld, Jarvis, & Wardle, 2008). Scores range from 0 to 4 (highest number of parents who smoked). Further, participants were asked to write in the exact number of siblings, half-siblings, and stepsiblings who smoked and lived with them. Similarly, they were asked to write in the exact number of close friends who smoked. These questions are consistent with those asked in nationally representative surveys, such

as the Canadian Youth Smoking Survey (YSS; Health Canada, 2007) and the U.S. National Youth Tobacco Survey (Center for Disease Control and Prevention, 2013).

Smoking Expectancies. The French-language version (Racicot, McGrath, Hine, O'Loughlin, & Guyon, 2008) of the Smoking Expectancy Scale for Adolescents (Hine, Honan, Marks, & Brettschneider, 2007) was used to measure the expected benefits and expected costs of smoking. Using a 10-point scale (0 = *Completely unlikely* to 9 = *Completely likely*), adolescents rated the likelihood that each expected cost and each expected benefit would happen to them. Mean scores were calculated for expected costs and benefits. This scale has excellent internal consistency (costs, α = .94; benefits, α = .92; Racicot et al., 2008). Smoking by parents and friends has been found to predict smoking expectancies (Hine et al., 2007; Racicot, McGrath, & O'Loughlin, 2011a), which in turn, have been associated with prospective smoking (Hine, McKenzie-Richer, Lewko, Tilleczek, & Perreault, 2002).

Smoking Behavior. Smoking behavior included questions drawn from the YSS and the Canadian Tobacco Use Monitoring Survey (Mills, Stephens, & Wilkins, 1994). Specifically, adolescents answered six questions: ever tried a cigarette, even just a few puffs (yes/no); ever inhaled cigarette smoke into your lungs (yes/no); ever smoked one whole cigarette (yes/no); ever smoked 100 or more whole cigarettes (yes/no); smoking frequency (i.e., number of days over the last month where smoking occurred); and smoking intensity (i.e., number of cigarettes smoked per day).

Analytic Plan

Data analysis comprised four steps. First, the association between S^3 scores and existing measures was evaluated to determine the degree of overlap between these measures. Second, S^3 scores on the parental, sibling, and peer versions were tested as predictors of smoking behavior and smoking expectancies. Third, existing measures (i.e., number of parents, siblings, and peers who smoked) were also tested as distinct predictors of smoking behavior and smoking expectancies, for comparison with the predictability of S^3 scores. Fourth, S^3 scores were directly compared with existing measures to determine whether our novel S^3 measure was more strongly associated with smoking behavior and smoking expectancies.

RESULTS

Descriptive Statistics

A total of 761 adolescents completed the S^3 Scale. On average, participants were aged 15.6 years (*SD* = 1.3), and the majority was female (61.4%). About half of the adolescents reported ever smoking a cigarette even just a few puffs (48.6%), and more than a third of the sample endorsed ever inhaling smoke into their lungs (37.4%). A third of the adolescents reported ever smoking a whole cigarette (33.3%), while only 15% of adolescents endorsed ever smoking more than 100 cigarettes. Participants smoked on average 3.55 days (*SD* = 9.03) over the last 30 days, and smoked 1.16 cigarettes per day (*SD* = 3.38). Adolescents were less likely to endorse benefits of smoking (*M* = 2.49, *SD* = 1.86) and expected the costs of smoking were more likely to occur to them (*M* = 5.61, *SD* = 2.01).

Descriptive statistics performed on the S^3 scores indicated few adolescents identified having no exposure to parental smoking (15.4%, *M* = .82, *SD* = .60), sibling smoking (20.5%, *M* = .61, *SD* = .50), or peer smoking (9.2%, *M* = .83, *SD* = .51), irrespective of the contextual situation. This indicates that the vast majority of participants endorsed exposure in at least one situation on the S^3 scale. According to existing measures, adolescents averaged 1.15 (*SD* = .82) smoking parents, 1.25 (*SD* = .90) smoking siblings, and 2.52 (*SD* = 2.69) smoking peers.

Convergent Validity between Existing Measures and S^3 Scale

Table 2 displays zero-order correlations between S^3 scores and existing measures. Existing measures were moderately correlated with the S^3 total score on the parental version ($r = .43$, $p \leq .001$), sibling version ($r = .43$, $p \leq .001$), and peer version ($r = .40$, $p \leq .001$). Existing measures were also significantly associated with the S^3 component scores for the parental version ($r_{avg} = .36$), sibling version ($r_{avg} = .31$), and peer version ($r_{avg} = .29$).

Concurrent Validity of S^3 Scale

S^3 total and component scores were significantly associated with ever smoking a cigarette; ever inhaling smoke into one's lungs; ever smoking one whole cigarette; and ever smoking more than 100 cigarettes on all three versions (parent, sibling, peer), although the component "belongingness" generally was not significantly associated with smoking behavior (Table 3). Moreover, the S^3 total and component scores (with the exception of "belongingness") were significantly associated with smoking frequency and smoking intensity on all three versions (Table 4). Further, with respect to smoking expectancies, the S^3 scores (total and components) were significantly associated with

expected benefits on the parental and peer versions, but not on the sibling version. The component "social activities" was consistently associated with expected costs.

Concurrent Validity of Existing Measures of Exposure to Smoking

Number of parents who smoked was significantly associated with smoking behavior, but not smoking expectancies (Tables 3-4), while number of siblings who smoked was significantly associated only with a few indicators of smoking behavior. Number of friends who smoked was significantly associated with smoking behavior and smoking expectancies.

Comparison of S^3 Scale with Existing Measures of Exposure to Smoking

S^3 scores generated greater effect size estimates than existing measures when predicting smoking behavior on the parental version (Table 3; total S^3: OR_{avg} = 2.59; components S^3: OR_{avg} = 1.88; existing: OR_{avg} = 1.35); the sibling version (total S^3: OR_{avg} = 3.44; components S^3: OR_{avg} = 2.16; existing: OR_{avg} = 1.59); and the peer version (total S^3: OR_{avg} = 3.89; components S^3: OR_{avg} = 2.98; existing: OR_{avg} = 1.38). The total S^3 score generally exhibited larger standardized beta coefficients than existing measures, except for the peer version (Table 4). However, some components on the peer version, such as "moods" or "quiet activities", generated greater effect size estimates than existing measures.

DISCUSSION

Researchers are learning increasingly about the importance of considering contextual situations when measuring social exposure to smoking and predicting smoking initiation among adolescents. In fact, existing measures fail to capture the contextual situations in which adolescents are exposed to smoking in their social environment.

Consistent with findings from the cue-reactivity framework, several of the components identified on the S^3 Scale were largely similar to the situations in which smokers report cravings or using cigarettes. Examples of such situations include social activities, mood states, meals, or quiet activities, suggesting that our scale has the ability to capture the situations in which smokers themselves reported smoking in previous studies. Further, analyses of convergent validity showed that the strength of association between S^3 scores and existing measures was moderately high. This implies that S^3 scores and number of smokers both measure a theoretically related construct, namely, social exposure to smoking. The fact that their strength of association was not excessively high, however, suggests that S^3 scores and existing measures capture unique aspects of social exposure to smoking. As such, present findings provide support for our conceptualization such that existing measures are mostly smoker-centric (i.e., "who is smoking"), whereas the S^3 Scale broadens the construct to include contextual situations of exposure (i.e., "where" and "when" are adolescents observing their parents, siblings, and friends smoke).

Analyses of concurrent validity showed that globally, adolescents exposed to smoking in more situations were more likely to be smokers, smoked more frequently and with greater intensity, and held more positive beliefs about smoking. Most importantly, S^3 scores were generally more strongly associated with smoking behavior and smoking expectancies than existing measures on all three versions of the S^3 Scale, which suggests the latter represents a potent alternative when measuring social exposure to smoking. This study corroborates and extends previous findings that situations of smoke exposure

predict greater smoking susceptibility in adolescent never-smokers (Racicot et al., 2011a).

Present findings provide support to the hypothesis that the S^3 Scale is a novel, psychometrically sound instrument designed to more precisely assess contextual exposure to smoking. Okoli, Browning, Rayens, and Hahn (2008) contend that the relation between social exposure to smoking and smoking behavior can be explained by two theoretical frameworks: social modeling (e.g., Krohn, Skinner, Masey, & Akers, 1985) and cue-reactivity (e.g., Caggiula et al., 2001). Given that adolescents complete the S^3 Scale for targeted role models, the scale encompasses elements of social modeling, such as observational learning of said role models. Moreover, the S^3 Scale captures elements of the cue-reactivity framework, given that contexts are environmental cues (Conklin, 2006). Observing one's father smoke after he has eaten a meal provides different contextual cues from when he is smoking while he is stressed, which may have different repercussions on the observing adolescent. In fact, this study highlighted that not all types of situations were equal. As an example, "social activities" emerged as a much stronger component than "belongingness".

Study Limitations

First, the cross-sectional nature of this study does not permit assessment of temporality. Second, S^3 items do not measure the intensity or duration of exposure. Nicotine monitors (Hammond & Leaderer, 1987) could be used in future studies to objectively obtain these data. Third, the S^3 measures contextual situations in which adolescents are exposed to cigarette smoking only. Although enquiring about other tobacco products would have been informative, cigarettes remain by far the most popular

(Health Canada, 2007). Fourth, this study used a full-sample design to validate the S^3 Scale; future studies should replicate the current findings with a larger sample, including a holdout sample. Fifth, the S^3 Scale is limited to social exposure to parental, sibling, and peer smoking. Additional versions could be developed to more comprehensively assess contextual exposure to smoking, including other family members, strangers in public places, or social media.

Implications and Recommendations for Future Research

From a methodological perspective, the S^3 Scale may be used as an alternative to existing measures to more precisely capture contextual exposure to smoking. Researchers may opt to examine the effects of the total S^3 scores or look at the effects of each individual component on smoking behavior during adolescence. Longitudinal research should test whether S^3 scores predict key smoking behavior milestones. Moreover, momentary ecological assessment (Shiffman, 2009) could be used to explore the degree of concordance between situations in which adolescents report exposure to smoking and situations in which parents, siblings, and friends report smoking.

From a public health perspective, this new, enhanced scale has the potential to strengthen prevention efforts targeting youth, as it could depict a more precise portrait of social exposure to smoking. Specifically, situations of smoke exposure known to be strongly associated with smoking could be depicted purposefully in media campaigns. Classroom interventions could address the situations in which adolescents observe smokers and emphasize the negative consequences of smoke exposure on smoking-related beliefs and eventual smoking. Relatedly, future research should examine the situations in which adolescents initiate cigarette smoking themselves. If these situations

are consistent with the ones in which they previously observed smokers, prevention programs could target specific situations of smoke exposure for better prevention of smoking uptake.

Conclusions

The development of the S^3 Scale and its psychometric properties was conducted to gain greater insight about the nature and repercussions of contextual situations of youth's exposure to smoking. The parental, sibling, and peer versions displayed excellent internal consistency and were significantly associated with smoking behavior and expected benefits. Compared with existing measures, S^3 scores were typically more strongly associated with smoking behavior and expected benefits, suggesting they are better predictors. Present findings suggest that the S^3 Scale is a brief, psychometrically sound instrument that could be used as an alternative to existing measures when assessing contextual exposure to smoking and statistically modeling adolescent smoking behavior. Thus, prevention programming should not only target who is smoking around adolescents, but also in which situations.

Table 1
Component Loadings on the Parental, Sibling, and Peer Versions of the S^3 Scale

	Component loadings				
Parental Items: They smoke (when)…	Social activities	Moods	Meals	Unpleasant states	-
at a party	**.85**	.24	.18	.15	-
in a group	**.79**	.33	.19	.22	-
having guests	**.74**	.28	.15	.35	-
in a calm situation	.31	**.81**	.24	.30	-
in a happy situation	.35	**.76**	.21	.32	-
in a boring situation	.35	**.76**	.22	.31	-
eating dinner	.17	.18	**.89**	.10	-
eating lunch	.20	.14	**.87**	.19	-
eating breakfast	.10	.19	**.84**	.20	-
doing housework	.29	.13	.20	**.77**	-
sick	.17	.38	.17	**.77**	-
having physical pain	.21	.39	.20	**.75**	-
Sibling Items: They smoke (when)…	Social activities	Moods	Meals	Quiet activities	Belonging-ness
at a party	**.91**	.17	.16	.10	.09
drinking alcohol	**.82**	.25	.19	.20	.06
in a group	**.78**	.30	.13	.06	.29
in a calm situation	.26	**.82**	.28	.30	.10
in a happy situation	.33	**.78**	.29	.28	.16
in a boring situation	.37	**.77**	.31	.21	.16
eating lunch	.22	.24	**.84**	.21	.20
eating dinner	.21	.35	**.82**	.24	.04
eating breakfast	.14	.21	**.74**	.44	.21
studying	.10	.18	.16	**.87**	.18
reading	.13	.17	.26	**.86**	.05
doing homework	.14	.25	.25	**.84**	.15
wanting to be part of a group	.13	.10	.14	.15	**.92**
wanting to impress others	.17	.14	.13	.13	**.91**
Peer Items: They smoke (when)…	Social activities	Moods	At school	Quiet activities	Belonging-ness
in a group	**.78**	.19	.32	.14	.27
using drugs	**.77**	.23	.15	.14	.26
at a party	**.77**	.27	.30	.10	.14
in a sad situation	.19	**.84**	.26	.28	.12
in a stressful situation	.32	**.81**	.25	.26	.08
in an infuriating situation	.25	**.79**	.30	.29	.07
during recess	.24	.24	**.79**	.21	.09
after school	.44	.26	**.75**	.17	.06
before going to school	.20	.32	**.74**	.31	.06
on the computer	.12	.20	.14	**.90**	.12
watching TV	.10	.19	.20	**.87**	.06
talking on the phone	.14	.33	.23	**.78**	.13
wanting to impress others	.22	.07	.04	.08	**.89**
wanting to be part of a group	.20	.08	.09	.14	**.88**

Table 2
Zero-order Correlations of S^3 Scores and Existing Measures of Exposure to Smoking

Parental version	1	2	3	4	5	6	
1. Smokers (Parents) [a]	-						
2. S^3 Score (Total)	.43 **	-					
3. Social activities	.43**	.84**	-				
4. Moods	.39**	.89**	.71**	-			
5. Meals	.21**	.73**	.44**	.50**	-		
6. Unpleasant activities	.39**	.84**	.61**	.72**	.47**	-	

Sibling version	1	2	3	4	5	6	7
1. Smokers (Sibling) [a]	-						
2. S^3 Score (Total)	.43**	-					
3. Social activities	.40**	.78**	-				
4. Moods	.39**	.88**	.64**	-			
5. Meals	.30**	.84**	.48**	.68**	-		
6. Belongingness	.25**	.59**	.37**	.38**	.38**	-	
7. Quiet activities	.22*	.74**	.36**	.57**	.61**	.35**	-

Peer version	1	2	3	4	5	6	7
1. Smokers (Friends) [a]	-						
2. S^3 Score (Total)	.40**	-					
3. Social activities	.32**	.82**	-				
4. Moods	.32**	.80**	.60**	-			
5. At school	.30**	.78**	.65**	.68**	-		
6. Belongingness	.15**	.57**	.51**	.28**	.26**	-	
7. Quiet activities	.34**	.72**	.41**	.60**	.54**	.29**	-

Note. a = Existing measures of exposure to smoking
* $p < .05$; ** $p < .01$

Table 3
Binary Logistic Regression Estimates for Smoking Behavior

	Ever tried a cigarette		Ever inhaled smoke into your lungs		Ever smoked one whole cigarette		Ever smoked 100 or more cigarettes	
Predictor variable	*OR*	(95%, CI)	*OR*	(95%, CI)	*OR*	(95%, CI)	*OR*	(95%, CI)
Parental version								
S^3 score (Total)	2.35	(1.77, 3.12)***	2.37	(1.76, 3.18)***	2.32	(1.71, 3.14)***	3.32	(2.13, 5.17)***
Social activities	1.82	(1.46, 2.29)***	1.76	(1.39, 2.23)***	1.83	(1.43, 2.35)***	2.45	(1.66, 3.61)***
Moods	1.84	(1.48, 2.30)***	1.85	(1.47, 2.33)***	1.86	(1.46, 2.37)***	2.42	(1.70, 3.46)***
Meals	1.72	(1.37, 2.17)***	1.83	(1.45, 2.31)***	1.76	(1.39, 2.23)***	2.11	(1.53, 2.91)***
Unpleasant activities	1.69	(1.33, 2.15)***	1.69	(1.32, 2.14)***	1.57	(1.23, 2.01)***	1.90	(1.35, 2.66)***
Smokers (parents) [a]	1.23	(1.01, 1.50)*	1.28	(1.04, 1.57)*	1.36	(1.09, 1.68)**	1.54	(1.16, 2.06)**
Sibling version								
S^3 score (Total)	3.31	(1.91, 5.72)***	4.10	(2.37, 7.09)***	2.64	(1.57, 4.44)***	3.69	(1.95, 6.97)***
Social activities	2.07	(1.48, 2.90)***	2.20	(1.54, 3.13)***	1.82	(1.28, 2.59)***	2.80	(1.63, 4.81)***
Moods	2.29	(1.59, 3.29)***	2.72	(1.88, 3.93)***	2.02	(1.41, 2.88)***	2.54	(1.61, 4.01)***
Meals	1.73	(1.20, 2.50)**	2.13	(1.48, 3.06)***	1.59	(1.12, 2.26)**	1.83	(1.20, 2.80)**
Quiet activities	2.44	(1.42, 4.20)***	2.65	(1.62, 4.35)***	1.85	(1.18, 2.90)**	1.93	(1.17, 3.18)**
Belongingness	1.06	(.63, 1.79)	1.01	(.58, 1.76)	1.37	(.79, 2.38)	2.15	(1.11, 4.19)*
Smokers (siblings) [a]	1.68	(1.07, 2.62)*	1.53	(1.00, 2.33)*	1.55	(1.00, 2.39)*	1.47	(.88, 2.47)
Peer version								
S^3 score (Total)	3.18	(2.23, 4.54)***	3.71	(2.60, 5.29)***	3.29	(2.32, 4.66)***	5.37	(3.47, 8.30)***
Social activities	2.12	(1.53, 2.94)***	2.29	(1.62, 3.26)***	2.39	(1.66, 3.43)***	3.79	(2.01, 7.13)***
Moods	2.19	(1.75, 2.74)***	2.49	(1.99, 3.13)***	2.26	(1.81, 2.83)***	3.84	(2.80, 5.28)***
At school	2.42	(1.90, 3.08)***	2.81	(2.19, 3.61)***	2.47	(1.93, 3.16)***	5.65	(3.72, 8.59)***
Quiet activities	3.10	(2.24, 4.29)***	3.34	(2.48, 4.50)***	2.75	(2.09, 3.62)***	3.68	(2.77, 4.89)***
Belongingness	.94	(.74, 1.18)	.92	(.73, 1.16)	1.04	(.82, 1.31)	.97	(.74, 1.28)
Smokers (friends) [a]	1.39	(1.27, 1.53)***	1.41	(1.29, 1.54)***	1.39	(1.28, 1.51)***	1.31	(1.22, 1.41)***

Note. a = Existing measures of exposure to smoking. OR = Odds ratio; CI = Confidence interval.
*$p \leq .05$; ** $p \leq .01$; ***$p \leq .001$.

Table 4
Linear Regression Estimates for Smoking Behavior and Smoking Expectancies

	Measures of smoking behavior						Smoking expectancies					
	Smoking frequency			Smoking intensity			Expected benefits			Expected costs		
Predictor Variable	β	*t*	η^2	β	*t*	η^2	β	*t*	η^2	β	*t*	η^2
Parental version												
S^3 Score (Total)	.20	5.02***	.04	.19	4.64***	.04	.13	3.08**	.02	.06	1.50	.00
Social activities	.18	4.55***	.03	.17	4.27***	.03	.12	3.04**	.02	.14	3.47***	.02
Moods	.21	5.15***	.04	.19	4.77***	.04	.11	2.81**	.01	.04	.91	.00
Meals	.11	2.62**	.01	.11	2.73**	.01	.09	2.25*	.01	-.01	-.23	.00
Unpleasant activities	.17	4.19***	.03	.14	3.49***	.02	.08	2.01*	.01	.03	.73	.00
Smokers (parents) [a]	.18	4.30***	.03	.16	3.87***	.03	-.01	-.20	.00	.00	.05	.00
Sibling version												
S^3 Score (Total)	.19	3.21***	.04	.21	3.42***	.04	.10	1.64	.01	.09	1.43	.01
Social activities	.13	2.14*	.02	.13	2.20*	.02	.05	.73	.00	.14	2.28*	.02
Moods	.27	4.53***	.07	.27	4.63***	.08	.09	1.47	.01	.10	1.54	.01
Meals	.14	2.26*	.02	.16	2.55**	.02	.08	1.25	.01	.07	1.08	.00
Quiet activities	.12	2.01*	.02	.12	1.99*	.02	.11	1.71	.01	.02	.25	.00
Belongingness	.02	.25	.00	.06	.72	.00	.16	1.95	.02	-.01	-.08	.00
Smokers (siblings) [a]	.11	1.20	.01	.07	.73	.01	.10	1.06	.01	.11	1.18	.01
Peer version												
S^3 Score (Total)	.31	7.85***	.10	.31	7.86***	.10	.21	5.27***	.05	.06	1.40	.00
Social activities	.20	3.67***	.04	.19	3.59***	.04	.22	4.04***	.05	.21	4.01***	.05
Moods	.36	9.23***	.13	.35	8.91***	.12	.23	5.67***	.05	.06	1.34	.00
At school	.36	9.22***	.13	.33	8.45***	.11	.22	5.49***	.05	.07	1.74	.01
Quiet activities	.41	10.86***	.17	.41	10.82***	.17	.20	4.98***	.04	-.03	-.71	.00
Belongingness	-.08	-1.88	.01	-.05	-1.18	.00	.05	1.14	.00	.08	2.02*	.01
Smokers (friends) [a]	.32	8.04***	.10	.34	8.41***	.11	.20	4.75***	.04	-.08	-1.99*	.01

Note. a = Existing measures of exposure to smoking. β = standardized beta coefficient; η^2 = eta squared, estimate of effect size.
*$p \leq .05$; ** $p \leq .01$; ***$p \leq .001$.

TRANSITION TO STUDY 3

The aim of Study 2 was to develop a refined psychometric instrument to measure and evaluate the effects of the situational contexts in which adolescents observe their parents, siblings, and peers smoke. Traditionally, such contexts have been overlooked by existing measures of social exposure to smoking (i.e., who is smoking, how many people are smoking), which largely focus on social modeling as their theoretical approach. Informed by the cue-reactivity framework which investigates the effects of smoking cues (e.g., ashtrays) on smokers, we postulated that the contextual cues in which social exposure to smoking occurs (e.g., witnessing parents smoke after a meal) could, in and of themselves, contribute to greater smoking risk. Consequently, we hypothesized that the S^3 Scale would permit a more comprehensive assessment of social exposure, as it encompasses elements of both social modeling and cue-reactivity frameworks.

As expected, the S^3 Scale was found to be more strongly associated with indicators of smoking behavior and smoking expectancies than the more narrowly focused, existing measures. Consistent with our hypotheses, the S^3 Scale generated larger estimates of effect size. This finding supports the proposition that the S^3 Scale measures the broader context of observing smokers and the many circumstances under which they smoke cigarettes, and that such contextual cues add to social exposure to smoking as a theoretical concept. Consequently, findings from Study 2 strongly suggest the S^3 Scale is an enhanced, more optimal instrument that allows for more precise measurements of social exposure to smoking and its effects on smoking behavior during adolescence.

When adolescents observe significant others smoke cigarettes, they engage not only in social learning within specific situational contexts, but they also inhale doses of

nicotine from SHS exposure. Epidemiological data have shown that SHS exposure can yield concentrations of nicotine in nonsmokers that are similar to those observed in some smokers (Al-Delaimy et al., 2001; Dimich-Ward et al., 1997). Notably, nicotine is known to have psychoactive effects on the central nervous system of smokers (Henningfield & Heishman, 1995), and nonsmokers inhaling SHS have been found to absorb concentrations of nicotine in levels known to have psychotropic responses in actual smokers (Pacifici et al., 1995; Perkins et al., 2001). Interestingly, an increasing number of researchers are beginning to consider that pharmacological exposure to nicotine from SHS (Becklake et al., 2005; Okoli, Rayens, & Hahn, 2007; Racicot et al., 2011a) could constitute a unique risk factor for adolescent smoking. As such, a plausible physiological pathway linking pharmacological exposure to nicotine during childhood and adolescence with subsequent smoking initiation has been proposed (Anthonisen & Murray, 2005). To test this hypothesis, we previously examined the differential effects of pharmacological and social exposure on precursors to smoking in a sample of never-smoking adolescents (Racicot et al., 2011a). Number of smokers among parents, siblings, and friends, but not salivary cotinine, was associated with smoking expectancies, ND symptoms, and smoking susceptibility. We discussed that low levels of pharmacological exposure observed in our sample, and using only one biomarker type (cotinine) could explain that pharmacological exposure did not significantly predict smoking precursors.

In spite of the non-significant finding for pharmacological exposure, we remained curious about the possibility of a physiological pathway between nicotine exposure from SHS and smoking behavior during adolescence, yet we were aware that testing that research question via the experimental approach would have posed serious ethical

challenges. The goal of Study 3 was to evaluate the differential relations of pharmacological and social exposure to smoking with the development of precursors to smoking. We tested our newly developed S^3 Scale (i.e., an enhanced measure of social exposure to smoking) against three objective pharmacological measures of nicotine exposure (i.e., salivary cotinine, hair nicotine, and passive nicotine monitor) to predict six smoking precursors: expected benefits, expected costs, temptations to try smoking, aversion to SHS exposure, smoking susceptibility, and ND symptoms.

STUDY 3

Is nicotine the smoking gun? The effects of pharmacological exposure to nicotine and social smoke exposure on smoking precursors among adolescent never-smokers

Racicot, S., & McGrath, J.J. (Under Review)

ABSTRACT

Social exposure to smoking, or observing smokers in one's environment, is a well-established predictor of smoking behavior during adolescence. Emerging evidence suggests that pharmacological exposure to nicotine from secondhand smoke (SHS) is also associated with smoking behavior. This study investigated whether pharmacological exposure to smoking uniquely predicts greater risk for smoking initiation among adolescent never-smokers. Participants included 338 never-smokers (M_{age}=12.9, SD= 0.4; 53% female) who answered questions about their social exposure to smoking (i.e., situations of SHS exposure derived from the Social Smoking Situations (S^3) Scale), and known smoking precursors (i.e., expected benefits and costs, temptations to try smoking, aversion to SHS, smoking susceptibility, ND symptoms). Saliva and hair samples were collected to derive pharmacological measures of cotinine and nicotine. Participants also wore a monitor to measure airborne nicotine. Greater pharmacological exposure was significantly associated with greater expected benefits (p = .018) and lower expected costs (p = .026). Greater social exposure was significantly associated with greater temptations to try smoking (p = .005), lower aversion to SHS exposure (p = .001), and greater smoking susceptibility ($p \leq .001$). Finally, greater social exposure was significantly associated with greater ND symptoms, but only in the presence of greater pharmacological exposure ($p \leq .001$). This study is the first to reveal that nicotine exposure from SHS poses a risk for developing smoking precursors.

INTRODUCTION

In spite of numerous anti-tobacco campaigns, more than half of North American children (53.6%) continue to be exposed to secondhand smoke (SHS; Centers for Disease Control and Prevention, 2010). SHS has been associated with deleterious health repercussions during childhood, including infections (e.g., ear infections, bronchitis, pneumonia), respiratory problems (e.g., asthma, coughing, sneezing), sudden infant death syndrome (U.S. Department of Health and Social Services [USDHHS], 2006), and neurobehavioral disorders (e.g., ADHD, learning disability; Kabir, Connolly, & Alpert, 2011). However, emerging evidence suggests that SHS exposure is also associated with smoking behavior during adolescence, and this association could be explained by nicotine exposure (Anthonisen & Murray, 2005). In fact, nicotine, the main psychotropic substance in tobacco (USDHHS, 1988), has been associated with nicotine dependence (ND) in humans (Henningfield & Heishman, 1995; Stolerman & Jarvis, 1995), sensitization in animals (Vezina, McGehee, & Green, 2007), and is present in SHS (Okoli, Kelly, & Hahn, 2007).

SHS Exposure in Smokers

Extant literature focusing on smokers indicates that SHS exposure confers greater risk for continued smoking and ND symptoms, over and above that of one's own active smoking behavior. Among adults, SHS exposure at home and in an automotive vehicle has been associated with a lower likelihood of contemplating cessation and attempting to quit, and a greater likelihood of reporting ND symptoms (Okoli, Browning, Rayens, & Hahn, 2008; Wilson-Frederick et al., 2011). Among adolescents, number of days where SHS exposure occurred at home has been associated with a greater likelihood of smoking

upon awakening in the morning and smoking more cigarettes daily, and lower odds of attempting to quit and ceasing smoking (Wang, Ho, Lo, & Lam, 2012, 2013). Specifically, SHS exposure at home partially mediated the relation between smoking by family members with smoking in the morning, daily cigarette consumption (Wang et al., 2012), and smoking cessation (Wang et al., 2013). Such findings indicate that even among smokers, nicotine exposure through SHS seems to represent a distinctive and additive risk factor for continued smoking and ND.

Exposure to SHS and Nicotine Intake in Nonsmoking Populations

In addition to conferring greater risk for smoking, SHS exposure has an impact on nonsmoking populations. Nonsmokers exposed to SHS inhale large amounts of its nicotine, namely, between 60 and 80% (Iwase, Aiba, & Kira, 1991). When comparing smokers with nonsmokers exposed to SHS at work, the concentration of nicotine measured in hair samples was largely comparable (1.2 vs. 1.0 ng/mg: Dimich-Ward, Gee, Brauer, & Leung, 1997; 7.9 vs. 6.7 ng/mg: Al-Delaimy, Fraser, & Woodward, 2001). Nonsmokers living with a nonsmoking spouse were found to have lower hair nicotine values than those whose spouse smoked outside the home only (0.3 vs. 0.5 ng/mg; Yoo et al., 2010), suggesting that even low exposure matters. An experimental study indicated that youth are more affected by SHS exposure than adults. After exposing nonsmoking children and nonsmoking adults to the same amounts of SHS, children exhibited a higher dose of nicotine than adults not only immediately after the exposure, but also one week later (Willers, Skarping, Dalene, & Skerfving, 1995). Importantly, these studies demonstrate that nonsmokers exposed to SHS absorb high doses of nicotine.

Pacifici and colleagues (1995) reported blood nicotine concentrations in nonsmokers that, at the same concentration, produce psychotropic effects in smokers (Perkins, Fonte, Sanders, Meeker, & Wilson, 2001). Recently, neuroimaging findings in young adults have revealed that smokers and nonsmokers exposed to SHS do not differ with respect to the occupancy of α4β2 nicotinic acetylcholine receptors (nAChRs) in the thalamus, brainstem, or cerebellum (Brody et al., 2011). Similarly, SHS exposure leads to greater density of α7 nAChRs in the stratum oriens and CA2/3, and non-α7 nAChRs in the dentate gyrus and thalamus of nicotine-naive rats (Small et al., 2010). Altogether, these findings suggest that nonsmokers can absorb similar quantities of nicotine when compared to smokers, and neuroimaging studies provide convincing evidence that such exposure has neuronal effects. Given that nicotine has deleterious effects on the developing brain of children and adolescents (cf. Slotkin, 2002), they represent a population that is particularly vulnerable to the effects of SHS exposure.

SHS Exposure and Smoking Behavior Milestones

Consistent with the literature showing that nonsmokers absorb non-negligible doses of airborne nicotine, SHS exposure has been identified as a risk factor for the onset of smoking behavior milestones. SHS exposure (i.e., number of days exposed at home, inside a car) has been associated with smoking in the past month and established smoking (Seo, Bodde, & Torabi, 2009), as well as ever smoking (Glover et al. 2011). The latter study showed that after adjusting for self-reported SHS exposure, parental smoking was no longer a significant predictor of ever smoking. Further, Wang, Ho, and Lam (2011) showed that exposure to parental smoking and SHS exposure at home (number of days where exposure occurred) predicted smoking initiation two years later. Follow-up

analyses revealed that SHS exposure partially mediated the relation between parental smoking and smoking initiation. Thus, these studies suggest that SHS exposure can act as a mediator or explain the relation between social exposure to smoking (e.g., parental smoking) and smoking behavior. However, these studies measured self-reported SHS exposure, but not pharmacological exposure to nicotine from SHS.

SHS Exposure in Never-Smokers: Evidence for Precursors to Smoking

SHS exposure has been identified as a risk factor not only for smoking behavior per se, but also for smoking precursors among never-smokers. Precursors to smoking are risk factors that develop prior to smoking initiation and are known to influence initiation, such as smoking susceptibility or smoking expectancies (cf. Racicot, McGrath, & O'Loughlin, 2011a). SHS exposure at home or in a vehicle has been shown to contribute to greater smoking susceptibility among adolescent never-smokers (Seo, Torabi, & Weaver, 2008). Experiencing an "unpleasant or gross" sensation during SHS exposure emerged as a protective factor against smoking susceptibility, whereas liking the smell of smoke was a risk factor for smoking susceptibility among preadolescent never-smokers (Lessov-Schlaggar, Wahlgren, Liles, Ji et al., 2011; Lessov-Schlaggar, Wahlgren, Liles, & Jones et al., 2011). Moreover, Bélanger et al. (2008) showed that SHS exposure predicted endorsement of ND symptoms among never-smoking 5th graders, even after adjusting for smoking by siblings and peers. Similarly, nicotine-naïve rats exposed to SHS exhibited behaviors consistent with withdrawal symptoms in animals (Small et al., 2010; Yamada et al., 2010). Thus, SHS exposure has been shown to contribute to smoking susceptibility and ND symptoms, which are known risk factors for smoking

initiation (Pierce, Choi, Gilpin, Parkas, & Merritt, 1996; O'Loughlin, Karp, Koulis, Paradis, & DiFranza, 2009).

Pharmacological Exposure to Nicotine from SHS and Greater Risk for Smoking

SHS exposure is frequently measured as the number of days (e.g., Wang et al., 2011) or the number of places where the exposure occurs (e.g., home vs. car; Okoli, Rayens, & Hahn, 2007), using self-report questions. Due to improvements in technology permitting greater ease of objective measurement of SHS exposure, researchers are relying increasingly on biomarkers of pharmacological exposure to nicotine through SHS (Jaakkola & Jaakkola, 1997). Upon entering the body, nicotine is mainly metabolized into cotinine, which can be found in bodily fluids like blood, saliva, and urine (Benowitz, 1996). Salivary cotinine is a biomarker that provides a short-term estimate of nicotine exposure over the last 2-4 days in children, given that its half-life ranges from 32 to 82 hours in that population (Jaakkola & Jaakkola, 1997). On the other hand, hair nicotine is a biomarker that provides long-term measurement of nicotine exposure, given that each centimeter of hair represents an estimate of nicotine exposure over the last 30 days (Al-Delaimy, 2002).

An increasing number of researchers are using nicotine biomarkers. Becklake, Ghezzo, and Ernst (2005) found that salivary cotinine measured in nonsmoking children predicted smoking initiation four years later, even after adjusting for the number of smokers at home. Further, Okoli, Rayens, and Hahn (2007) showed that adult nonsmokers with higher hair nicotine values were 2.2 times more likely to endorse four or more DSM-IV nicotine withdrawal symptoms. Thus, these studies provide preliminary, compelling evidence that pharmacological exposure to nicotine may

contribute to smoking behavior. Using salivary cotinine biomarkers, Racicot, McGrath, and O'Loughlin (2011a) studied the effects of pharmacological exposure to nicotine on precursors to smoking initiation among never-smoking adolescents. Salivary cotinine did not significantly predict smoking precursors, which was largely attributable to the low levels of SHS exposure observed in the sample.

Social Exposure to Smoking

Observing smoking by parents, siblings, and peers across different contextual situations in youth's social environment has been referred to as social exposure to smoking (Racicot, Drouin, & McGrath, 2014), and is a robust, consistent predictor of smoking during adolescence (Leonardi-Bee, Jere, & Britton, 2011; O'Loughlin et al., 2009). Social Learning Theory (Bandura, 1977) posits that adolescents observe and imitate the smoking behavior of role models, and its effects have been documented for the past few decades (e.g., Krohn, Skinner, Massey, & Akers, 1985). Recently, researchers have begun to acknowledge that observing smokers may lead not only to greater social exposure to smoking (i.e., observing who is smoking, where, and when), but also greater pharmacological exposure to nicotine through SHS (i.e., inhaling and absorbing airborne nicotine). According to this conceptualization, it is imperative to simultaneously measure social exposure to smoking and pharmacological exposure to nicotine through SHS to evaluate their independent effects on smoking precursors.

The Present Study

Extending our earlier work (Racicot et al., 2011a), this study evaluated the differential effects of social exposure to smoking (i.e., contextual situations of parental, sibling, and peer smoking) and pharmacological smoke exposure (i.e., salivary cotinine,

hair nicotine, and nicotine monitor) on smoking precursors in a sample of adolescent never-smokers. Smoking precursors were selected purposefully due to their established relations with smoking behavior. Specifically, smoking expectancies develop prior to smoking initiation (Foster, Racicot, & McGrath, 2012) and contribute to prospective smoking (Hine, McKenzie-Richer, Lewko, Tilleczek, & Perreault, 2002). Aversion to SHS exposure has been associated with a greater likelihood of having a home smoking ban (Martinez-Donate et al., 2007), which in turn, has been associated with a lower likelihood of smoking initiation (Emory, Saquib, Gilpin, & Pierce, 2010). ND symptoms among adolescent never-smokers (Racicot, McGrath, Karp, & O'Loughlin, 2013) predicted first cigarette puff and onset of daily smoking in a 5-year longitudinal study (O'Loughlin et al., 2009) Smoking susceptibility is a well-established predictor of smoking initiation among never-smoking adolescents (Pierce et al., 1996). Further, smoking susceptibility has been associated with social exposure to smoking (Racicot et al., 2011a), and sensitivity to SHS exposure (Lessov-Schlaggar, Wahlgren, Liles, Ji et al., 2011) among never-smoking youth. The present study tested two specific hypotheses. First, it was hypothesized that after adjusting for social exposure to smoking, greater pharmacological exposure to nicotine from SHS would remain the only significant predictor associated with greater smoking precursors. Second, it was hypothesized that greater social exposure to smoking would predict greater smoking precursors, but only in the context of greater pharmacological exposure.

METHOD

Participants

Adolescents (N = 406; 52.5% females; M_{age} = 13.0 years, SD = 0.5) attending secondary schools within the greater Montreal area participated in AdoQuest, a longitudinal cohort study designed to examine the development of smoking behavior and ND during adolescence. Adolescents were recruited in seventh grade, as there is a higher probability that they never smoked cigarettes compared to higher grades (never smokers: 91.0% 7th grade vs. 80.8-52.3% 8-12th grades; Health Canada, 2012). The Institutional Review Board of Concordia University approved the AdoQuest study (#1000116).

Procedure

After receiving approval from the school boards, school principals and teachers were contacted to obtain their authorization to collect data during class time. Informed consent forms were sent home to parents with the students. Data collection consisted of two visits in each classroom. At the first visit, research personnel provided standardized information about the study objectives and confidentiality. Participants were instructed to complete a self-report questionnaire silently. While adolescents were completing questionnaires, trained research assistants collected a saliva sample (cotinine) and hair sample (nicotine) from each student. At the end of the first visit, passive nicotine monitors were distributed for participants to wear on themselves for the next seven consecutive days. The second visit occurred one week later when research assistants returned to the classrooms to collect the passive nicotine monitors.

Measures

Social Smoking Situations (S^3) Scale. Social exposure to smoking was assessed with the S^3 Scale, which measures contextual situations of smoke exposure to parental, sibling, and peer smoking (Racicot et al., 2014). Adolescents rated their current level of smoke exposure in each situation (e.g., "my friends smoke after school"; 0 = not true, 1 = somewhat true, 2 = very true). Items were averaged to compute a S^3 Total score, which represents a mean ranging from 0 to 2. Higher S^3 scores indicate greater contextual exposure to smoking. Similarly, we computed a separate score for each version (parents, siblings, peers), and each of the seven subscales derived from principal components analysis. This scale has excellent internal consistency reliability (Cronbach's α = .92-93; Racicot et al., 2014).

Salivary Cotinine. Salivary cotinine samples were assayed in duplicate by Salimetrics, LLC (State College, PA, USA). Testing was performed using a highly sensitive enzyme immunoassay that requires a volume of 20 µl of saliva for each determination and has a .15 ng/mL limit of sensitivity with an intra-assay coefficient of variation of 4.1% (Salimetrics, 2011). The mean of the two duplicates was used as the salivary cotinine value. In adolescents, a cut-off value of 11.4 ng/ml has been used to differentiate smokers from nonsmokers (Kandel et al., 2006).

Hair Nicotine. Approximately 10-15 strands of hair were collected from each student's scalp, and the centimeter closest to the root was used for analysis. Hair samples were assayed for nicotine using reversed-phase high performance liquid chromatography with

electrochemical detection (HPLC-ECD) by the Laboratory Services of Capital Coast Health Limited (Wellington, New Zealand) with a limit of quantification of .04 ng/mg (Mahoney & Al-Delaimy, 2001). Although HPLC-ECD is not used diagnostically to classify adolescents according to their smoking status (personal communication with Dr. Wael Al-Delaimy, August 14, 2012), hair nicotine provides an excellent dose-response measure of exposure to SHS.

Passive Nicotine Monitors. Passive nicotine monitors measure the concentration of nicotine in ambient air (Hammond & Leaderer, 1987; Leaderer & Hammond, 1991). Specifically, the monitor consists of a windscreen, a filter treated with sodium bisulfate, and a 3.7-cm polystyrene cassette. Adolescents were instructed to wear the monitor continuously for seven days, except for bathing/showering, physical activity (e.g., swimming, martial arts), and sleeping. When not wearing the monitor, they were asked to leave it nearby in their proximal environment (e.g., bedside table while sleeping at night; desk on school days). Nicotine collected on the sodium bisulfate filters was assayed by gas chromatography with nitrogen selective detection at the School of Public Health, University of California – Berkeley. Passive nicotine monitors have a limit of detection of 0.01μg. Nicotine concentration was calculated by dividing the quantity of nicotine found on the sodium bisulfate filters by the estimated total volume of air sampled over seven days (Hammond & Leaderer, 1987).

Smoking Expectancies. Participants completed the French-language version (Racicot, McGrath, Hine, O'Loughlin, & Guyon, 2008) of the Smoking Expectancy Scale for

Adolescents (Hine, Honan, Marks, & Brettschneider, 2007), which measures two principal factors: expected costs (e.g., "get lung cancer") and expected benefits (e.g., "feel less stressed"). Using a 10-point scale (0 = *completely unlikely* to 9 = *completely likely*), participants rated the probability that each cost or benefit would occur if they were smokers. An average score was calculated for each factor (0 – 9). This scale has excellent internal consistency (Cronbach's alpha: expected costs = 0.94, expected benefits = 0.92, Racicot et al., 2008).

Temptations to Try Smoking. Participants rated the extent to which they were tempted to try smoking in 15 situations (e.g., "With friends at a party", "When I'm embarrassed to be a nonsmoker), using a 5-point scale (*not at all tempted* to *extremely tempted*). An average score was calculated (0 – 4). This scale has excellent internal consistency among adolescents (Cronbach's alpha = 0.94; Hudmon, Prokhorov, Koehly, DiClemente, & Gritz, 1997).

Aversion to SHS Exposure. Using a 3-point scale (*strongly agree* to *do not agree at all*), participants rated the extent to which they dislike SHS exposure (e.g., "I feel bothered when someone smokes around me"), prefer smoke-free places (e.g., "I prefer to study in smoke-free environments), and support laws banning smoking inside specific public places (e.g., restaurant, schools, hospitals; Martinez-Donate et al., 2007). Scores represent an average ranging from 0 to 2, where higher scores represent greater aversion to SHS exposure. This scale has good internal consistency (Cronbach's alpha = .83, Martinez-Donate et al., 2007).

Smoking Susceptibility. Smoking susceptibility was measured using two items from the pan-Canadian Youth Smoking Survey (Health Canada, 2007; e.g., "Have you ever been curious about smoking a cigarette?), and three items from the study by Pierce and colleagues (1996; e.g., "If one of your best friends were to offer you a cigarette, would you smoke it?) A composite score was created by summing the items (0 – 11) and computing the mean, where higher scores represent greater susceptibility to smoking. This coding system has been used previously, and smoking susceptibility was associated with social exposure to smoking (ß = .27; Racicot et al., 2011a).

ND Symptoms. Participants answered seven items measuring ND symptoms among adolescents. Specifically, six items were derived from an "ND/craving indicator" (O'Loughlin, DiFranza et al., 2002; e.g., "How often do you have cravings to smoke cigarettes?") and one item was derived from the Nicotine Dependence Scale for Adolescents (Nonnemaker et al., 2004; e.g., "I sometimes have strong cravings where it feels like I'm in the grip of a force that I cannot control"). Items were summed and divided by the number of items to compute average ND scores, which were log-transformed to correct for positive skewness. ND symptoms have been associated with SHS exposure in a car (*OR* = 1.2), sibling smoking (*OR* = 1.8), and peer smoking (*OR* = 2.2; Bélanger et al., 2008).

Analytic Plan

Linear regression was used to test social exposure to smoking (S^3 Total score) and pharmacological exposure to nicotine (i.e., salivary cotinine, hair nicotine, nicotine badge monitors) as predictors of smoking precursors (i.e., expected benefits, expected costs,

temptations to try smoking, aversion to SHS, smoking susceptibility, ND symptoms). First, each predictor was tested in univariate modeling. Second, S^3 score and each of the three pharmacological measures were tested in paired multivariable modeling. Third, S^3 score, each of the three pharmacological measures, and their interaction were tested in full multivariable modeling.

RESULTS

Descriptive Statistics

A total of 406 adolescents completed questionnaires and provided biomarkers (52.5% female; M_{age} = 13.0 years, SD = 0.5). Of these, the analytic sample included 338 adolescents (83.25%) who endorsed the inclusion criterion that they had "never smoked a cigarette, not even a few puffs" (53.0% female; M_{age} = 12.9 years, SD = 0.4). On average, participants reported that the benefits of smoking were "very unlikely" to happen to them, while the costs of smoking were "somewhat likely" to happen to them (See Table 1). Overall, participants reported they were "not at all" tempted to smoke and they endorsed high levels of aversion to SHS exposure. The mean score for ND was low; however, a subset of never-smoking adolescents endorsed at least one ND symptom (19.8%). This percentage endorsing ND symptoms is higher than that reported in another study of never smoking youth using identical items (5%; Bélanger et al., 2008), which is likely attributable to the younger age of their sample (5^{th} grade). Mean score for smoking susceptibility was low (M = .20, SD = .31), although 41.1% of the sample endorsed at least one item positively, and thus, would be classified as susceptible to smoking (Pierce et al., 1996).

An examination of the S^3 scores indicated that a total of 39.6% of adolescents endorsed social exposure to smoking. Specifically, 33.7% endorsed social exposure to parental smoking, 14.2% to sibling smoking, and 14.5% to peer smoking. Similarly, adolescents reported experiencing social exposure during the following contextual situations: social activities (34.3%), mood states (25.7%), meals (28.8%), belongingness (13.3%), quiet activities (11.2%), unpleasant activities (21.6%), and at school (13.9%). With respect to pharmacological exposure, values for the biomarkers were below the cut-off used to distinguish smokers from nonsmokers, suggesting that adolescents accurately reported they were never-smokers. Mean salivary cotinine (M = .48 ng/ml, SD = 1.21) was lower than the established cut-off value for categorizing adolescents as smokers (11.4 ng/ml; Kandel et al., 2006). Mean hair nicotine value (M = .38 ng/mg, SD = 1.40) and mean passive nicotine monitor value (M = .59 μg, SD = 2.05) were also consistent with values expected from nonsmokers. Further, mean values for all three measures of pharmacological exposure were above the lower limit of sensitivity (salivary cotinine = .15 ng/ml, Salimetrics, 2011; hair nicotine = 0.04 ng/mg, Mahoney & Al-Delaimy, 2001; nicotine monitor = .01μg, Hammond & Leaderer, 1987), suggesting that adolescents were exposed to nicotine from SHS.

Social and Pharmacological Smoke Exposure to Predict Smoking Precursors

Linear regression modeling was used to test social exposure and pharmacological exposure as differential predictors of smoking precursors. First, when comparing the univariate, paired, and full models, pharmacological exposure best predicted smoking expectancies in univariate modeling (i.e., univariate were best fitting models). Specifically, higher nicotine exposure from ambient air, measured with the passive

nicotine monitor, was significantly associated with greater likelihood of expected benefits $F(1,337) = 5.64$, $R^2 = .02$, $p = .018$, and lower likelihood of expected costs $F(1,337) = 5.00$, $R^2 = .02$, $p = .026$ (See Table 2). Neither social exposure, nor their interaction was significantly associated with smoking expectancies.

Second, social exposure best predicted temptations to try smoking, aversion to SHS exposure, and smoking susceptibility in univariate modeling. That is, higher S^3 total score was significantly associated with more temptations to try smoking $F(1,337) = 8.10$, $R^2 = .02$, $p = .005$, lower aversion to SHS exposure $F(1,337) = 11.99$, $R^2 = .04$, $p = .001$, and greater smoking susceptibility $F(1,337) = 20.73$, $R^2 = .06$, $p \leq .001$. Neither pharmacological exposure, nor their interaction was significantly associated with these precursors.

Finally, the interaction between pharmacological exposure and social exposure best predicted ND symptoms. Specifically, the full model including cotinine ($\beta = -.19$, $t = -2.15$, $p = .032$), S^3 Total score ($\beta = .19$, $t = 3.19$, $p = .002$), and their interaction ($\beta = .26$, $t = 2.71$, $p = .007$) was significantly associated with ND symptoms $F(3,337 = 10.38$, $R^2 = .09$, $p \leq .001$. Similarly, the full model including the nicotine monitor ($\beta = -.20$, $t = -1.87$, $p = .062$), S^3 Total score ($\beta = .21$, $t = 3.44$, $p = .001$), and their interaction ($\beta = .25$, $t = 2.21$, $p = .028$) was significantly associated with ND symptoms $F(3,337) = 9.50$, $R^2 = .08$, $p \leq .001$. Interpretation of the interaction effects indicated greater social exposure was significantly associated with greater ND symptoms, but only in the presence of greater pharmacological exposure (see Figure 1). In other words, when adolescents were exposed to higher nicotine, greater social exposure was associated with endorsing more ND symptoms.

Post-hoc Exploratory Analyses

Exploratory analyses were also conducted to evaluate the relation between smoking precursors and S^3 subscales. S^3 subscales were derived from principal components analysis (Racicot, et al., 2014) and provide information about the source of exposure (who: parents, siblings, peers) and the situational context of exposure (when/ where: social activities, moods, meals, quiet activities, belongingness, unpleasant activities, at school). Table 2 includes the univariate associations between these S^3 subscales and each of the six smoking precursors. When examining the source of exposure, S^3 Peer was more strongly associated with temptations to try smoking and smoking susceptibility, whereas S^3 Sibling was more strongly associated with aversion to SHS. When examining the situational contexts of exposure, S^3 Social Activity emerged as the subscale that was most consistently associated with temptations to try smoking, aversion to SHS exposure, and smoking susceptibility.

DISCUSSION

A growing body of research provides convincing evidence that pharmacological exposure to nicotine from SHS is a plausible risk factor for smoking initiation during adolescence (cf. Okoli, Kelly, & Hahn, 2007). Social exposure to smoking, however, is a well-established risk factor for adolescent smoking (USDHHS, 2012). While social exposure was not associated with smoking expectancies, it did significantly predict greater temptations to smoke, lower aversion to SHS exposure, and greater smoking susceptibility, which is consistent with previous research. Conversely, greater pharmacological exposure to nicotine from SHS was associated with greater expected benefits and lower expected costs. Importantly, this study is the first to document a

relation between pharmacological exposure and smoking expectancies. Finally, greater social exposure was associated with greater ND symptoms, but only in the context of greater pharmacological exposure. Taken together, this study highlights a dose-response relation between smoke exposure (social and pharmacological) and the development of precursors to smoking among never-smoking adolescents. This will, in turn, confer greater risk for smoking initiation.

Current findings corroborate previously reported research demonstrating a link between greater social exposure and greater smoking susceptibility (Azagba & Asbridge, 2013; Leatherdale, Brown, Cameron, & McDonald, 2005; Schuck, Otten, Engels, & Kleinjan, 2012). These results extend earlier findings of an association between greater social exposure and greater expected benefits and lower expected costs (Hine et al., 2007; Racicot et al., 2011a); greater social exposure and greater ND symptoms (Bélanger et al., 2008; Racicot et al., 2011a, 2011b, 2013); and greater pharmacological exposure and greater ND symptoms (Okoli, Rayens, & Hahn, 2007). Taken together, the present study replicated and extended findings from an emerging research area postulating that pharmacological exposure to nicotine from SHS is a unique risk factor for smoking. As such, current results are consistent with previous studies showing that pharmacological exposure to nicotine increases the risk of prospective smoking initiation during adolescence (Becklake et al., 2005); predicts withdrawal signs among nicotine-naïve strain rats (Small et al., 2010); and leads to nAChRs occupancy in the brains of nonsmoking adults (Brody et al., 2011).

Pharmacological Exposure to Nicotine as a Unique Risk Factor for Smoking

Our results indicate the nicotine monitor, a measure of the total concentration of airborne nicotine over seven days, best captured smoking expectancies that include a comprehensive list of expected benefits (i.e., affect control, social benefits, boredom reduction, weight control) and expected costs (i.e., addiction, appearance costs, social costs, health costs) in relation to cigarette smoking (Hine et al., 2007). Unlike salivary cotinine and hair nicotine, the monitor may represent the overall intensity of nicotine exposure through SHS, and is not affected by bodily functions like metabolism. Contrary to expectations, hair nicotine did not emerge as a significant predictor of smoking precursors. Studies investigating genetic differences in rates of nicotine metabolism suggest that individuals who metabolize nicotine rapidly could have lower biomarker values that similarly exposed individuals who metabolism nicotine slowly (Chenoweth, O'Loughlin, Sylvestre, Tyndale, 2013; Malaiyandi, Sellers, & Tyndale, 2005). However, genetic differences should have affected salivary cotinine values similarly. It is possible that extraneous factors (youth dying their hair or applying other chemicals) may explain this non-significant result. Future studies are needed to address that question.

As posited by Anthonisen and Murray (2005), our results support the plausibility of a physiological pathway between nicotine exposure from SHS and smoking behavior, irrespective of social modeling. Although the nature of such a pathway remains to be elucidated, the neuronal effects of nicotine in the brains of never-smokers could conceivably resemble those observed in smokers. Benowitz (2010) reported that the pathway from the ventral tegmental area to the nucleus accumbens in the mesolimbic dopaminergic system has been linked to dependence in smokers; thus, this pathway could

be a potential candidate. According to the Sensitization-Homeostasis Model, neuroadaptations can be observed soon after administration of low nicotine doses (DiFranza, Huang, & King, 2012), which poses the question as to whether nicotine intake from SHS exposure could suffice in triggering such neuroadaptations. Given that nonsmokers exposed to SHS can absorb concentrations of nicotine related to nicotine discrimination in smokers (Pacifici et al., 1995; Perkins et al., 2001), the present study lends support to the possibility of neuroadaptations induced by nicotine exposure through SHS.

Okoli, Kelly, and Hahn (2007) raised the hypothesis that repeated nicotine absorption from SHS may contribute to greater tolerance of its aversive sensations, which could possibly make initial experiences with active smoking less aversive and, consequently, more rewarding. Positive responses when smoking one's first cigarette (e.g., relaxation) have been linked to greater ND (Ursprung, Savageau, & DiFranza, 2011), while experiencing positive experiences during SHS exposure has been associated prospectively with greater smoking susceptibility (Lessov-Schlagger, Wahlgren, Liles, Jones et al., 2011). In the context of the present findings, one could contend that greater pharmacological exposure leads to the development of positive beliefs (e.g., "smoking helps calm down") and unawareness of negative consequences (e.g., "smoking damages health"), which could plausibly explain why adolescents continue to smoke following an initial pleasant experience. Overall, nicotine intake from SHS is a probable, unique risk factor for smoking, regardless of social exposure to smoking. This suggests that exposure to nicotine, even in the absence of smokers (i.e., third-hand smoke), could confer risk.

Social Exposure to Smoking as a Unique Risk Factor for Smoking

Adolescents who spend time with smokers engage in social learning while being simultaneously exposed to environmental cues across various contextual situations. Current findings showed social exposure best predicted precursors which appear to be based on principles of social modeling and conditioning. As an example, greater temptations to try smoking in different situations (e.g., feeling tempted to smoke when at a party with friends) imply observational learning of peer smoking within a specific contextual situation (i.e., a party). Lower aversion to SHS exposure (e.g., being more unfavorable to smoking bans in various settings, such as public transit) could be linked to witnessing smokers discuss their disapproval of a smoking ban while in that specific setting. Greater smoking susceptibility (e.g., greater likelihood of accepting a cigarette offer from a friend) could also be linked to elements of social modeling, such as wanting to imitate friends' smoking behavior and thus, being less likely to decline a cigarette offer. Given that these precursors appear to be more psychosocial than biological in nature, this may explain why pharmacological exposure did not predict these specific precursors.

The influence of parental, sibling, and peer smoking has been documented extensively (Leonardi-Bee et al., 2011; Hoffman, Sussman, Unger, & Valente, 2006). Findings from the current study are consistent with the principles of social modeling (Bandura, 1977) across varied contextual situations (Van Gucht, Van der Bergh, Beckers, & Vansteenwegen, 2010). Most recently, the number of smokers in the social environment of never-smoking youth predicted desire to smoke in the presence of environmental cues (e.g., sight and smell of smoke; Schuck, Kleinjan, Otten, Engels, &

DiFranza, 2013), using the cue-triggered scale of the Autonomy Over Smoking Scale (DiFranza, Wellman, Ursprung, & Sabiston, 2009), thereby supporting a theoretical connection between social modeling and cue exposure among never-smokers. Overall, this study supports previously reported findings, whereby social exposure to smoking, even in the absence of direct nicotine exposure (e.g., observing parents smoke outside from inside; observing smoking in movies or on television) still constitutes a considerable risk factor for smoking.

Social Exposure in the Presence of Greater Pharmacological Exposure

The current study indicated that adolescents with greater social exposure to smoking reported more ND symptoms, but only in the presence of greater pharmacological exposure, as evidenced by higher values for salivary cotinine and the nicotine monitor. In other words, adolescents with greater social exposure did not report more ND symptoms when nicotine levels derived from salivary cotinine and the nicotine monitor were lower. ND is a multifaceted phenomenon involving biological processes (e.g., being physically addicted to cigarettes), social modeling and cue exposure (e.g., wanting to smoke when observing peers smoking; wanting to smoke in forbidden places). This could plausibly explain why social exposure best predicted ND symptoms only in the context of higher pharmacological exposure. Consistent with notions of conditioning, a possible mechanism would imply that adolescents inhale nicotine from SHS as they are observing smokers describe their ND symptoms in different situations (e.g., being with peers who are smoking in the schoolyard while talking about their cravings). Thus, the present findings support the idea of three interconnected mechanisms facilitating an understanding of the relation between smoke exposure and smoking behavior: social

modeling, conditioning (i.e., exposure to environmental cues), and pharmacological exposure to nicotine from SHS (Okoli et al., 2008). Further, this finding provides crude, preliminary support to the animal literature reporting complex interactions between nicotine and non-pharmacological cues (cf. Chaudhri et al., 2006). Overall, higher social exposure paired with higher pharmacological was most predictive of reporting ND symptoms.

Limitations

Three methodological limitations require consideration. First, the cross-sectional, correlational nature of the study precludes establishment of causal relations between nicotine exposure from SHS and smoking precursors. In alignment with animal studies (Small et al., 2010; Yamada et al., 2010), experiments could be conducted on humans to manipulate pharmacological and social exposure across different conditions (e.g., observing smokers in the same room vs. behind a mirror; inhaling nicotine from an electronic cigarette vs. inhaling water vapor from an electronic cigarette, etc.) and to assess their consequences on temptations to try smoking, ND symptoms, or susceptibility. Consistent with previous imaging research (Brody et al., 2011), future neuroimaging studies could consider whether nicotine exposure induces neural changes during adolescence by comparing the brains of never-smokers frequently exposed to nicotine to those of never-smokers with no or minimal exposure.

Second, we did not examine whether adolescents were compliant with monitor instructions. Nevertheless, we relied on three distinct indicators of pharmacological exposure for triangulation of nicotine exposure. In addition to utilizing the monitor, which provides an estimate of the total concentration of airborne nicotine in youth's

immediate environment (Hammond & Leaderer, 1987), we used a short-term (cotinine, 2-4 days; Jaakkola & Jaakkola, 1997) and a long-term (hair nicotine, 30-31 days; Al-Delaimy, 2002) estimate of nicotine exposure. The moderately high amount of overlap between the three pharmacological indicators (r_{avg} = .52) strongly suggests that adolescents wore the monitor as instructed. Further, we used the S^3 Scale to measure social exposure to smoking, which focuses on contextual situations of exposure and represent an enhanced psychometric instrument compared to traditional measures of social exposure (Racicot et al., 2014).

Third, the present study focused on smoking precursors rather than smoking initiation per se. It is recommended that future research test the longitudinal relation between pharmacological exposure and key smoking behavior milestones, including initiation and daily smoking. However, investigating the risk factors that set never-smokers at risk of initiating smoking from those not at risk is important. Such results among never-smokers could permit researchers to prospectively evaluate the effects of smoke exposure on age of onset or the rate of progression through the different smoking stages.

Conclusions

This study is the first to report that nicotine exposure from SHS poses a risk for developing precursors to smoking. The psychoactive effects of nicotine have been hypothesized as a plausible mechanism explaining the association of greater exposure to nicotine from SHS with greater expected benefits and lower expected costs of smoking. Moreover, this study demonstrated that greater social exposure is related to greater ND symptoms, but only in the presence of greater pharmacological exposure. This suggests

that social exposure is necessary, but not sufficient in explaining ND symptoms among never-smokers. Public health implications include that smoking bans should be implemented in homes and cars where youth spend time, given that it is not just watching smokers that matters, but also being exposed to nicotine in their absence. From a smoking cessation perspective, this study could inform current debates pertaining to the efficacy and safety of nicotine delivered via electronic cigarettes.

Table 1

Descriptive Statistics

Variables	*M*	(*SD*)
Expected benefits (0 – 9)	2.46	(1.66)
Expected costs (0 – 9)	5.83	(2.17)
Temptations to try smoking (0 – 4)	.26	(.43)
Aversion to SHS exposure (0 – 2)	1.75	(.41)
Nicotine Dependence Symptoms (0 – 3)	.08	(.20)
Smoking Susceptibility (0 – 2)	.20	(.31)
S3 Total Score	.07	(.15)
S3 SCORES (VERSION)		
Parents (0 – 2)	.17	(.35)
Siblings (0 – 2)	.02	(.13)
Peers (0 – 2)	.03	(.16)
S3 SCORES (SUBSCALES)		
Social Activities (0 – 2)	.11	(.21)
Moods (0 – 2)	.07	(.18)
Meals (0 – 2)	.07	(.19)
Belongingness (0 – 2)	.02	(.12)
Quiet Activities (0 – 2)	.01	(.07)
Unpleasant Activities (0 – 2)	.12	(.34)
At School (0 – 2)	.04	(.20)
Salivary cotinine (ng/ml)	.48	(1.21)
Hair nicotine (ng/mg)	.38	(1.40)
Passive nicotine monitor ($\mu g/m^3$)	.59	(2.05)

Table 2

Univariate Regression Models Predicting Risk Factors for Smoking Initiation

	Smoking Expectancies											
	Expected Benefits		Expected Costs		Temptation		Aversion		Susceptibility		Nicotine Dependence	
Predictor Variable	β	*t*	β	*t*	β	*t*	β	*t*	β	*t*	β	*t*
Age	.06	1.19	-.01	-.12	-.01	-.17	-.03	-.55	-.03	-.56	.08	1.48
Sex	.05	.93	.01	.23	.00	.02	.07	1.32	.06	1.07	-.03	-.57
Social Exposure												
S^3 Total Score	.08	1.43	-.02	-.38	.15	2.85**	-.19	-3.46**	.24	4.55**	.26	4.84**
Parent[a]	.03	.61	-.02	-.27	.07	1.20	-.11	-2.00*	.15	2.75**	.16	2.96**
Sibling[a]	.06	1.18	.01	.11	.06	1.18	-.26	-4.88**	.16	2.89**	.16	3.02**
Peers[a]	.09	1.60	-.03	-.53	.22	4.21**	-.04	-.81	.21	3.94**	.22	2.12**
Social Activities[a]	.08	1.44	-.02	-.29	.17	3.12**	-.20	-3.72**	.26	4.98**	.25	4.74**
Moods[a]	.06	1.17	-.02	-.39	.12	2.13*	-.13	-2.38*	.21	2.01**	.18	3.40**
Meals[a]	.05	.94	-.02	-.41	.04	.68	-.12	-2.16*	.04	.73	.16	3.03**
Belonging[a]	.08	1.54	.02	.32	.16	3.05**	-.17	-3.12**	.20	3.73**	.19	3.58**
Quiet Activities[a]	.07	1.30	-.01	-.16	.11	1.99*	-.22	-4.05**	.17	3 .09**	.22	4.16**
Unpleasant[a]	.05	.87	.01	.14	.06	1.07	-.10	-1.76	.15	2.86*	.17	3.17**
School[a]	.08	1.54	-.06	-1.02	.23	4.26**	-.09	-1.58	.23	4.34**	.23	4.24**
Pharmacological Exposure												
Cotinine	.09	1.58	-.04	-.76	-.04	-.65	-.07	-1.26	.01	.24	.09	1.64
Hair	.05	.96	-.06	-1.10	.00	.08	-.15	-2.82**	.10	1.87	.09	1.56
Monitor	.13	2.38*	-.12	-2.24*	.00	.05	-.05	-.94	.02	.37	.10	1.85

Note. a = S^3 subscales; variables investigated in exploratory analyses only. β = standardized beta coefficient.

*$p \leq .05$; ** $p \leq .01$

Figure 1: Interaction between S^3 Scores and cotinine to predict nicotine dependence

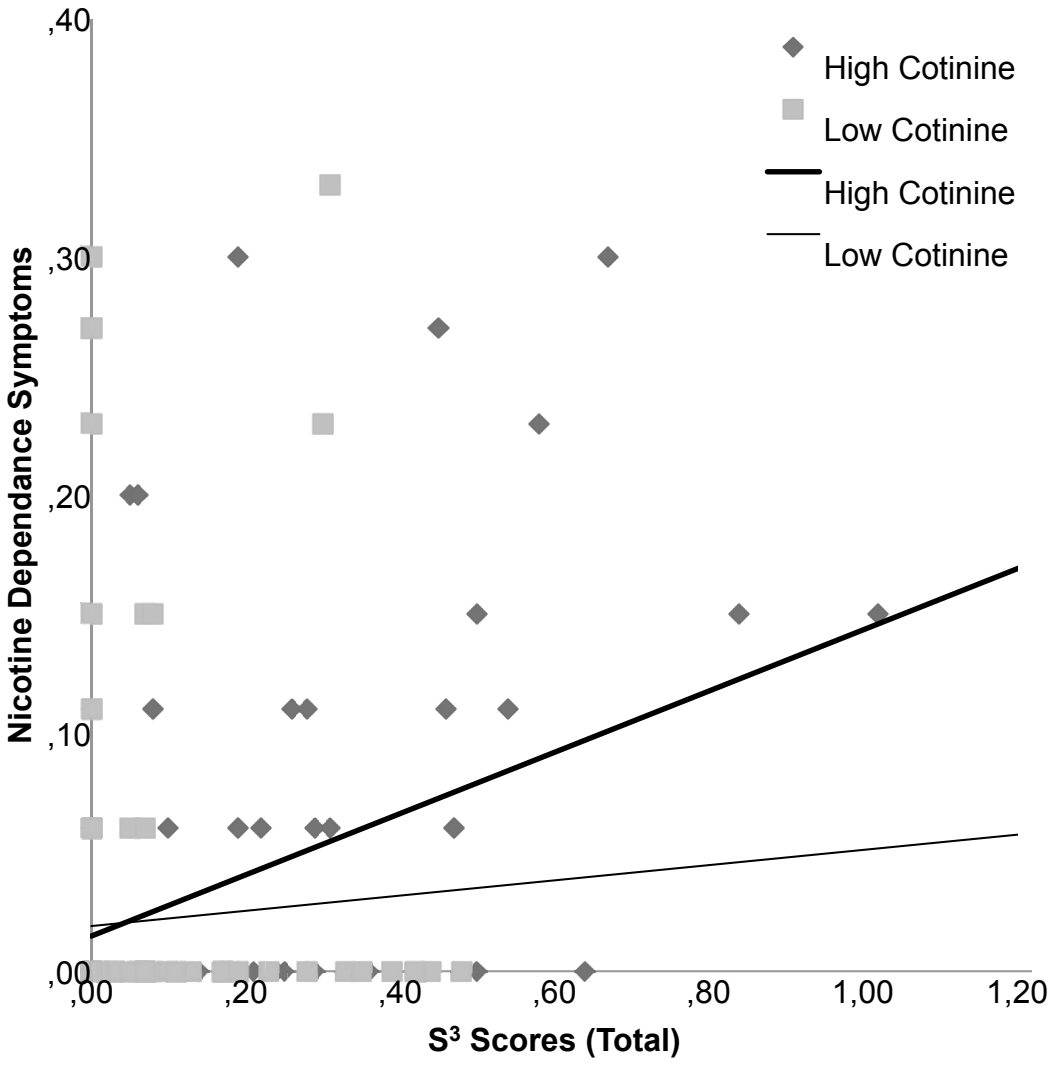

Figure 1: Cotinine was median split to facilitate depiction of the significant interaction; continuous variable retained in statistical modeling reported in text.

GENERAL DISCUSSION

In spite of well-intentioned prevention programs and punchy media campaigns, the prevalence of smoking remains too high (e.g., 44% of Québec adolescents in grades 10-12 try smoking; Health Canada, 2012). Considering that smoking typically begins during adolescence and tracks into adulthood (Chassin et al., 2000), identifying and understanding the factors that explain why some never-smoking adolescents will eventually turn into smokers is still a public health priority. Extant literature has convincingly concluded that one of the putative predictors of smoking initiation is exposure to others' smoking. Researchers have become interested in investigating the different effects of two major components of smoke exposure on smoking behavior during adolescence: 1) social exposure to smoking and 2) pharmacological exposure to nicotine from SHS exposure.

The first major component, social exposure to smoking, has been investigated extensively by social scientists. Informed by Social Learning Theory (Bandura, 1977), past research has largely conceptualized social exposure to smoking as the influence of smoking by role models, such as parents, siblings, or friends (e.g., Collins & Ellickson, 2004). While social modeling remains a robust mechanistic explanation for the underlying association between smoke exposure and smoking initiation, other mechanisms have been hypothesized as additional, likely contributors to adolescent smoking behavior. In fact, social exposure to smoking implies that adolescents observe their parents, siblings, and friends smoke cigarettes (i.e., social modeling), yet few had considered that such exposure takes places across a variety of situational contexts that could act as environmental cues (e.g., diverse settings, moments, circumstances; Conklin,

2006; Van Gucht et al., 2010). Thus, one question that remains is whether contextual situations of social exposure to smoking could enable researchers to better measure social exposure and, consequently, better predict smoking than existing indicators, which typically focus on "who smokes" or "how many individuals smoke" in youth's social environment.

The second major component, pharmacological exposure to nicotine from SHS exposure, has gained more scientific attention recently (cf. Okoli, Kelly, & Hahn, 2007). In effect, researchers reported that nonsmokers exposed to high levels of nicotine from SHS display biological doses of nicotine as high as those observed in some smokers (Al-Delaimy et al., 2001; Dimich-Ward et al., 1997). Others indicated that nonsmokers exposed to nicotine from SHS absorb quantities of nicotine higher than those required by smokers to discriminate the effects of nicotine (Pacifici et al., 1995; Perkins et al., 2001). Taken together, such data convincingly demonstrate that nonsmokers exposed to nicotine from SHS are not nicotine-naïve, although studies relying on social modeling as their paradigm have traditionally overlooked that passive nicotine intake occurs when youth observe smokers consume cigarettes. This raises another question as to whether nicotine exposure from SHS represents a unique risk factor for smoking onset, regardless of the effects of social exposure to smoking.

The publication of unexpected, intriguing research findings significantly informed the development of the present dissertation. Some researchers reported that pharmacological exposure to nicotine from SHS predicted smoking initiation during adolescence (Becklake et al., 2005), and ND symptoms among nonsmoking adults working in bars and restaurants (Okoli, Rayens, & Hahn, 2007). Similarly, children self-

reporting SHS exposure in a vehicle also endorsed ND symptoms, after adjusting for social exposure (Bélanger et al., 2008). Relatedly, the study by O'Loughlin and colleagues (2009) showed that endorsing ND symptoms predicted smoking initiation and daily smoking. Based on these previously reported findings, we conducted a study in which we found that social exposure was linked to smoking expectancies, ND symptoms, and smoking susceptibility (Racicot et al., 2011a). Although these findings suggested that SHS exposure is a likely contributing factor to smoking precursors, the relative contribution of pharmacological and social exposure remained largely unknown. Never-smokers represent an important population to study, given that pharmacological exposure to nicotine from SHS can be measured validly without the confounding effects of active smoking. Further, understanding the risk factors present in never-smokers prior to smoking uptake could strengthen prevention programming.

To address these research gaps, the objective of this dissertation was threefold. In Study 1, we tested the longitudinal predictors of ND symptoms in a sample of never-smokers, which is, by definition, inconsistent with current conceptualization of ND (APA, 2000). Measures of social exposure were investigated as potential predictors of ND symptoms. In Study 2, we developed an enhanced measure of social exposure to smoking – the S^3 Scale – to more accurately measure the influence of smoking parents, siblings, and peers on adolescents. Specifically, we were interested in determining whether contextual situations of smoke exposure (i.e., where, when) would better explain adolescent smoking than traditional measures which typically focus on the absence or presence (e.g., "does your mother smoke: yes/no"), or the number (e.g., "two of my closest friends smoke") of smokers. In Study 3, we evaluated the differential effects of

social exposure to smoking and pharmacological exposure to nicotine from SHS on smoking precursors to determine whether both exposure routes were significant predictors. Altogether, the ultimate goal of this dissertation was to evaluate the two major components of smoke exposure (social and pharmacological) as potential mechanisms explaining the development of precursors to smoking, which have been involved in the transition from never smoking to ever smoking.

Using longitudinal data from the NDIT study, Study 1 identified peer smoking, stress, and alcohol use as significant predictors of ND symptoms. By demonstrating that specific predictors could be used to statistically model reports of ND symptom in a sample of never-smokers, we provided further evidence that adolescents do not mistakenly or randomly endorse ND symptoms. Since the NDIT study did not include biomarkers of nicotine exposure, it was impossible to test pharmacological smoke exposure as a potential predictor of ND symptoms. Nevertheless, Study 1 was the first to demonstrate a longitudinal relation between social exposure and reports of ND symptoms by never-smokers, which represents a meaningful contribution to the literature. According to our findings, reports of ND symptoms by never-smokers should not be considered spurious; instead, they should be viewed as another target for the prevention of smoking. However, some researchers have raised criticism, arguing that measurement error is the key reason linking social exposure to ND symptoms in nonsmokers.

In Study 2, we hypothesized that social exposure to smoking, as a theoretical construct, encompasses both observing smokers and the contextual situations in which the exposure takes place. Accordingly, we developed the S^3 Scale to assess the contextual situations in which adolescents witness their parents, siblings, and peers smoke. Using

cross-sectional data from the AdoQuest Study, we found the S^3 Scale more strongly predicted smoking behavior and smoking expectancies than existing measures of social smoke exposure (e.g., "who is smoking"). We concluded that the S^3 Scale represents an enhanced psychometric instrument which permits more comprehensive assessment of the effects of social exposure to smoking. Researchers could use the S^3 Scale as an alternative to traditional measures to study temporal relations between contextual situations of exposure to smoking and eventual smoking uptake. This holds promise for assisting researchers in pinpointing high-risk situations for smoking behavior more efficiently. Further, using the S^3 Scale represents a judicious methodological decision to help researchers distinguish the effects of social exposure from those of pharmacological exposure more precisely.

In Study 3, we investigated the differential relations of social and pharmacological exposure to smoking with smoking precursors, using cross-sectional data from the AdoQuest study. This study is the first to demonstrate an association of greater pharmacological exposure with greater expected benefits and lower expected costs. Further, this is the first study to document the finding that greater social exposure is associated with more ND symptoms, but only in the context of greater pharmacological exposure. Congruent with previous findings, greater social exposure was associated with greater temptations to try smoking, greater smoking susceptibility, and lower aversion to SHS exposure. Overall, Study 3 enabled us to support the hypothesis that pharmacological exposure to nicotine from SHS is a plausible, unique risk factor for smoking behavior during adolescence.

Taken together, this research program provides additional, stronger evidence that exposure to nicotine via SHS is a formidable risk factor for smoking precursors and, ultimately, smoking behavior. The distinctive feature of this research program pertains to its emphasis on dismantling the major components of smoke exposure (social vs. pharmacological), and examining their relative consequences on increasing risk of adolescent smoking. Developing the S^3 Scale and testing it against three objective measures of nicotine exposure from SHS is an original contribution that bridges gaps in scientific knowledge.

Present Findings in Relation to Current State of Knowledge

In this dissertation, we highlighted the pertinence of considering contextual situations when measuring social exposure to smoking. While the cue-reactivity framework largely focuses on cues associated with actual smoking (Carter & Tiffany, 1999), it has placed less emphasis on environmental cues in the context of social exposure to smoking. Thus, this dissertation extends cue-reactivity findings in animals (Caggiula et al., 2001) and smokers (Conklin, 2006; Cronk & Piasecki, 2010; Dunbar et al., 2010; Shiffman, et al., 2002; Tagmat et al., 2011; Van Gucht et al., 2010). Consistent with the position taken by Okoli et al. (2008), we posit that social exposure to smoking encompasses elements of both *social learning* (i.e., observing and imitating role models) and c*ue-reactivity* (i.e., observing role models smoke cigarettes across different contextual situations). The importance of non-nicotinic factors in explaining smoking behavior is a known phenomenon. In fact, nicotine has been shown to enhance the reinforcing effects of non-pharmacological cues paired with nicotine use, as well as the reinforcing effects of non-nicotine cues that are not paired with nicotine (Caggiulia et al.,

2009; Chaudhri, et al., 2006). DiFranza and Wellman (2005) have also proposed a role for non-nicotinic cues in their Sensitization-Homeostasis Model.

Furthermore, conclusions from the present dissertation corroborate and extend findings from different frameworks, ranging from physiological studies in animals to biopsychosocial studies in humans. From a neurobiological perspective, current conclusions are consistent with animal studies showing that nicotine-naïve rats exposed to nicotine exhibit nicotine withdrawal signs (Small et al., 2010; Yamada et al., 2010), and with neuroimaging studies in humans showing that nAChRs occupancy in smokers is not statistically different from that in nonsmokers in the aftermath of nicotine exposure through SHS (Brody et al., 2011). The Sensitization-Homeostasis Model developed by DiFranza and Wellman (2005) posits that irregular nicotine self-administration in experimental smokers may help to explain the development of sensitization, which has been considered a plausible mechanism related to smoking progression (Vezina et al., 2007). The Sensitization-Homeostasis Model was not developed with respect to SHS exposure; nonetheless, it could possibly be extended to nonsmokers who, just like experimental smokers, can be irregularly exposed to nicotine via SHS. Further, our findings are consistent with a new line of research reporting that experiencing positive sensations when exposed to nicotine from SHS (e.g., feeling relaxed) increases the likelihood of susceptibility (Lessov-Schlaggar, Wahlgren, Liles, Ji et al., 2011; Lessov-Schlaggar, Wahlgren, Liles, & Jones, 2011), whereas those who perceive SHS exposure as unhealthy are less likely to initiate smoking (Song et al., 2009).

General Methodological Limitations

Two key limitations of this research program merit consideration. First, participants completed self-report questionnaires, and no other informants (e.g., first-degree relatives, friends) provided information. Although home visits could be conducted to gather additional data about youth's exposure to smoking, previous reports have established that youth reliably report smoking-related information (Eppel et al., 2006; Harakesh, Engels, de Vries et al., 2006). Importantly, participants provided salivary cotinine and hair nicotine samples, and wore the passive nicotine monitor for a week, which suggests that collecting collateral data would not necessarily improve measurement accuracy.

Second, all three studies used a correlational design and Study 2 and Study 3 relied exclusively on cross-sectional data, which precludes establishment of temporal and causal associations. As an example, this dissertation did not allow for an evaluation of the long-term effects of past pharmacological exposure to nicotine, although hair nicotine provides an estimate of nicotine exposure over the last 30 days (Mahoney & Al-Delaimy, 2001). Moreover, this dissertation did not investigate genetics as a contributing factor to smoking precursors. While adolescents whose family members smoke are more at risk of smoking themselves because of a shared genetic vulnerability (Avenevoli & Merikingas, 2003), genes moderate the rate of nicotine metabolism (slow vs. fast metabolism) due to their effects on the hepatic enzyme CYP2A6 (Malaiyandi et al., 2005). Recent research suggests that adolescents who metabolize nicotine slowly smoke more cigarettes and are more dependent on nicotine (Rubinstein, et al., 2013), while others have found that slow metabolism increases likelihood of smoking cessation among adolescents (Chenoweth et

al., 2013). Genetic differences between individuals could affect biomarker values (Avila-Tang et al., 2013; Jaakkola & Jaakkola, 1997), such that higher pharmacological exposure could be attenuated by faster nicotine metabolism when examining biomarker values. In fact, greater pharmacological exposure paired with slower nicotine metabolism could lead to higher biomarker values than greater pharmacological exposure paired with faster metabolism. Prospective studies should test whether nicotine metabolism moderates the relation between pharmacological exposure and outcomes for smoking.

Recommendations for Future Research

Conclusions from the present dissertation motivated us to think about the next questions that other researchers or we should address. First, it is recommended that studies use longitudinal data to investigate the unique effects of social and pharmacological exposure on smoking initiation, and to evaluate the possibility that the association of social exposure with smoking initiation is moderated or mediated by pharmacological exposure (e.g., Wang et al., 2011). Moreover, future research should explore the complex relations among smoking precursors, using path analysis or structural equation modeling. For example, Okoli and colleagues (2009) found that ND symptoms predicted smoking susceptibility, while Bélanger et al. (2008) found that smoking susceptibility predicted ND symptoms. Ursprung, DiFranza, Costa, and DiFranza (2009) reported no relation between ND symptoms and smoking expectancies. Unraveling these intricate relations requires further research. Second, studies relying on the animal model have the potential to experimentally isolate the effects of nicotine exposure from SHS among nonsmokers. Recently, Cohen and George (2013) developed an animal model in which rodents are exposed to nicotine vapors in a non-contingent

fashion, which simulates the reality that nonsmoking humans are intermittently exposed to nicotine from SHS. Further, they contend that this line of work could plausibly help determine whether electronic cigarettes represent an effective cessation technique, although the safety of electronic cigarettes, which only deliver doses of nicotine, has not been established yet (Chen, 2012; Odum, O'Dell, & Schepers, 2012).

Public Health Implications

Over the last few years, public health organizations have deployed considerable effort to reduce youth's exposure to SHS. Smokefree policies in homes, cars, school settings, and multi-unit housing have been recommended (e.g., Leatherdale & Ahmed, 2009; Lee et al., 2012; Pizacani, Maher, Rohde, Drach, & Stark, 2012). Exposure to high concentrations of nicotine from SHS in automobiles, for example, is very preoccupying from a public health perspective (Jones, Navas-Acien, Yuan, & Breysse, 2009). Based on the present dissertation, we strongly support the necessity of implementing and enforcing smokefree laws to protect youth from SHS exposure. In addition to recommending that smokers abstain from smoking in the presence of youth, we suggest that smokers abstain from smoking in places where youth spend time, even in their absence. As an example, parents should avoid smoking inside the family car, even when they are alone, given that their children will be exposed eventually to nicotine from the residual smoke (thirdhand smoke). Moreover, we suggest that researchers further investigate emissions of nicotine from electronic cigarettes. Given that they deliver doses of nicotine, the question remains as to whether or not electronic cigarettes represent a dangerous source of nicotine exposure.

Conclusions

Altogether, these dissertation findings generate new knowledge about the differential effects of social and pharmacological exposure on smoking risk during adolescence. Evidence supports the possibility of a physiological pathway between nicotine exposure from SHS and eventual smoking, irrespective of the effects of social exposure to smoking. However, future research is required to confirm and identify the nature of such a pathway. From a health prevention viewpoint, our findings could be used to further inform public health policy-making, thereby encouraging complete smoking bans in environments where youth spend time, including households, cars, multi-unit housing, and school premises.

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