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An examination of the relationships between causal attributions for smoking and smokers' treatment seeking and quit intentions: A structural equation modeling approach

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AN EXAMINATION OF THE RELATIONSHIPS BETWEEN CAUSAL

ATTRIBUTIONS FOR SMOKING AND SMOKERS' TREATMENT SEEKING AND

QUIT INTENTIONS: A STRUCTURAL EQUATION MODELING APPROACH

by

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Smokers' Treatment Seeking and Quit Intentions: A Structural Equation Modeling

Approach

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ABSTRACT

With increasing knowledge of the role that genetics play in the development and treatment of nicotine dependence, it is expected that in the future smoking cessation treatment will be able to be tailored to a smoker's genetic profile. Despite anticipated benefits such as improved quit rates, concerns have been raised about the impact of genetic testing results on perceived control over smoking, motivation to quit, and treatment seeking behaviour. One potential mediator of such outcomes are causal attributions, the causal explanations people form for behaviours and events, which evidence suggests can be altered by genetic testing feedback. The purpose of the current study was to perform a comprehensive assessment of causal attributions for current smoking and to examine the associations between these attributions and variables expected to predict future smoking cessation behaviour. Two structural equation models were tested that represented a series of hypotheses regarding how causal attributions influence intentions to quit smoking and intentions to seek smoking cessation treatment, via beliefs about perceived control over smoking and perceived effectiveness of treatment. Causal attributions were represented by causal types (biological, psychological, social, and stress) in one model and by causal dimensions (locus of causality, stability, internal control, and external control) in a second model; both models were otherwise identical.

Participants were 418 current daily smokers in Ontario, Canada, that had previously participated in the Ontario Health Study. Overall, participants most frequently attributed their smoking to habit, addiction, and/or stress, while attributions to genetics were among the least frequent. Additionally, knowledge that genetics play a role in

determining level of addiction to nicotine was not pervasive. Study findings supported

the hypothesized model in which causal dimensions directly predicted level of perceived

control over smoking (personal or via treatment), which in turn predicted perceived

effectiveness of pharmacological and psychosocial smoking cessation treatments,

intentions to quit smoking, and intentions to seek cessation treatment. Results failed to

find similar associations with causal types. Current findings can be applied to future

research on the effects of providing genetic testing feedback to smokers in clinical

settings, and may have wider applicability to other health threats.

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CHAPTER I

INTRODUCTION

Despite the fact that comprehensive tobacco control efforts have resulted in a substantial decrease in tobacco consumption over the past several decades, smoking is still the leading cause of preventable death in North America (Centers for Disease Control and Prevention, 2012; World Health Organization, 2002). In Canada, 62% of smokers report that they are seriously considering making a quit attempt in the next six months (Reid & Hammond, 2011). Unfortunately, only 3-5% of those who attempt to quit on their own will achieve sustained abstinence after a given quit attempt (Hughes, Keely, & Naud, 2004). Several smoking cessation treatments have been shown to help smokers improve their odds of long-term quit success (Fiore et al., 2008; Lemmens, Oenema, Knut, & Brug, 2008), yet most quit attempts are made without their aid (Cokkinides, Ward, Jemal, & Thun, 2005; Shiffman, Brockwell, Pillitteri, & Gitchell, 2008). In fact, it has been remarked that "it is difficult to identify any other condition that presents such a mix of lethality, prevalence, and neglect, despite effective and readily available interventions" (Fiore et al., 2008, p. 1).

Researchers investigating nicotine and tobacco use and dependence continue to uncover the role that genetics play in the ability to quit smoking, including evidence of individual variation in the effectiveness of smoking cessation medications (Lerman, Schnoll & Munafò, 2007). It is expected that with increased knowledge regarding the impact of individual genetic variants on treatment efficacy, healthcare providers may eventually be able to tailor treatment based on a smoker's genetic profile in order to maximize quit success (Lerman et al., 2007: Meiser, Mitchell, McGirr, Van Herten, &

Schofield, 2005). Using genetic information to optimize treatment will require providing patients with feedback regarding genetic test findings. As a result, questions and concerns have been raised about what psychosocial and behavioural effects might follow from providing this information to patients. Some primary concerns raised are whether genetic feedback leads to decreased perceived control over health threats given that one's genetic makeup is fixed; whether genetic feedback will have any impact, either beneficial or detrimental, on motivation to change health behaviour and actual behaviour performance; and whether genetic feedback will prompt changes in the perceived effectiveness and utilization of different types of interventions, particularly a preference for biologically based treatment (e.g., taking medication) over psychosocial treatment (e.g., counselling). Based on the research to date, however, it has been recognized that "whether and how genetic information might add positively to existing interventions or produce counterproductive responses is still largely unknown" (McBride, Bowen et al., 2010, p.561).

Determining whether genetic information improves the adoption of healthy behaviour was identified as a top research priority by an interdisciplinary group of scientists convened by the National Human Genome Research Institute in 2008 (McBride, Bowen et al., 2010). The group further recommended that attention be paid to identifying the mechanisms through which genetic communication might influence health behaviours, and that social and psychological theory could be used to frame these research questions. The current study focuses on one such factor that may act as a mediator and/or moderator of behavioural outcomes following genetic testing, *causal*

attributions, the implicit and explicit causal explanations people form to answer questions of "why?" (Kelley, 1973).

Causal beliefs for a particular health condition have been found to differ based on genetic test feedback (Marteau et al., 2004) and to change following genetic counselling (Albada et al., 2013). Theory predicts that a change in causal beliefs will subsequently lead to changes in motivation and behaviour (Leventhal et al., 1980; Weiner, 1985). Prior to examining whether causal attributions function as an intermediary variable between genetic feedback and psychological or behavioural outcomes, a better understanding of how causal attributions impact smoking behaviour is beneficial. Thus, the purpose of the current study is to examine whether causal attributions for smoking influence smokers' intentions to quit smoking and to seek smoking cessation treatment, with an emphasis on perceived control and perceived treatment effectiveness as mediating variables. In order to examine this research question, the current study will test a causal model incorporating these variables among a general population of smokers in Ontario, Canada.

CHAPTER II

LITERATURE REVIEW

The following literature review will provide background on treatment seeking for smoking cessation and the future potential for application of genetic testing to smoking cessation treatment, causal attributions as a potential mediator between genetic testing and various outcomes, the relevance of attributions for health-related outcomes, measurement of causal attributions, and relevant theoretical frameworks that guided study design and interpretation of findings. Following this review, the purpose of the study and hypotheses will be presented. See Appendix A for a glossary of important terms.

Smoking: Prevalence, Consequences, and Treatment

Prevalence. Following initial empirical evidence documenting a link between smoking and lung cancer in the 1950s and 1960s (Doll & Hill, 1954; U.S. Department of Health, Education, and Welfare, 1964), a growing awareness of the health risks associated with smoking and resulting enactment of tobacco control policies, such as increased taxation on cigarettes (e.g., Hu, Sung, & Keeler, 1995), indoor smoking bans in public spaces (e.g., Fichtenberg & Glantz, 2002), and restrictions on tobacco advertising (e.g., Quentin, Neubauer, Leidl, & König, 2007), have resulted in a considerable decrease in the prevalence of smoking in North America (Centers for Disease Control and Prevention, 1999). However, the decline in smoking prevalence has slowed and rates have remained relatively stable over the past several years (Centers for Disease Control and Prevention, 2011b; Health Canada, 2011). Estimates indicate that roughly 1 in 5 Americans (19%) and 1 in 6 Canadians (17%) still smoke, representing a combined total

of approximately 50 million current smokers in North America (Centers for Disease Control and Prevention, 2011b; Health Canada, 2011). Furthermore, the prevalence of smoking is increasing in the developing world due to population growth and tobacco industry targeting, creating what is now being referred to as a "global tobacco epidemic" (World Health Organization, 2011). Nearly one billion people in the world smoke today and without implementation of comprehensive tobacco control strategies that number is expected to continue to rise (World Health Organization, 2008).

Health consequences. More than fifty years of extensive research has provided decisive evidence of a link between smoking and a multitude of detrimental health effects leading to morbidity and early mortality, including several forms of cancer, cardiovascular and respiratory disease, reduced fertility and other reproductive effects, and blindness (U.S. Department of Health and Human Services, 2010). Tobacco-related illness is estimated to account for more than 37,000 deaths each year in Canada (Baliunas et al., 2007) and 440,000 deaths in the United States (Centers for Disease Control and Prevention, 2011b), and is also estimated to be responsible for a total of $210 billion annually in direct and indirect costs to the Canadian and American economies (Centers for Disease Control and Prevention, 2011b; Rehm et al., 2007).

Smoking cessation treatment. Epidemiological data indicate that 69% of current smokers in the United States would like to stop smoking completely (Centers for Disease Control and Prevention, 2011a), while 62% of smokers in Canada report that they are seriously considering making a quit attempt in the next six months (Reid & Hammond, 2011). The good news is that a former smoker can begin to experience health benefits almost immediately upon quitting smoking. Within 20 minutes of stopping smoking heart

rate decreases and between two weeks to three months after quitting an ex-smoker's heart attack risk begins to decline. Within 12 months of quitting one's risk of coronary heart disease is cut by half, and by 15 years risk of stroke and coronary heart disease is the same as a non-smoker (U.S. Department of Health and Human Services, 2004). Unfortunately, only 3-5% of those who attempt to quit without assistance achieve prolonged abstinence for at least 6-12 months after a given quit attempt (Hughes et al., 2004). Several effective tobacco dependence treatments that can improve the odds of long-term cessation are widely available (Fiore et al., 2008; Lemmens et al., 2008). For example, using the nicotine patch to aid with cessation almost doubles the probability of quitting (RR = 1.9; Stead, Perera, Bullen, Mant, & Lancaster, 2008), while the medication varenicline more than doubles the probability of successfully quitting (RR = 2.3; Cahill, Stead, & Lancaster, 2011). A strong dose-response relationship exists between the length of in-person counselling a person receives and long-term abstinence rates. Even minimal counselling (< 3 minutes) is effective compared to no contact (OR = 1.3), although intensive counselling (> 10 minutes per session) more than doubles quit rates (OR = 2.3; Fiore et al., 2008). Combining medication and counselling further improves treatment outcomes compared to either treatment modality alone (Fiore et al., 2008). Extensive evidence also proves the effectiveness of other forms of intervention, including group counselling, tailored self-help materials (e.g., manuals), and telephone quitlines (Fiore et al., 2008).

Application of Genetic Testing to Smoking Cessation Treatment

Completion of the Human Genome Project in 2003 prompted a rapid proliferation of research identifying links between numerous genetic variants and a variety of mental

and physical health conditions (Feero, Guttmacher, & Collins, 2008). Researchers

investigating nicotine and tobacco use and dependence have discovered that in addition to

a host of environmental factors, genetics plays an important role in the initiation of

tobacco use, progression to nicotine dependence, and ability to quit smoking (Lerman et

al., 2007). Based on data from twin, family, and adoption studies, it is estimated that

genetic factors account for approximately 60% of the risk for initiating smoking, 70% of

the variability in progression to nicotine dependence, and approximately 50% of the

variance in achieving abstinence following a given quit attempt (Lerman et al., 2007;

Malaiyandi, Sellers, & Tyndale, 2005).

        In light of evidence for heritability of nicotine dependence, researchers have

focused on identifying the genetic mechanisms involved. For example, some genetic

variants affect the activity of particular enzymes, altering the rate at which nicotine is

metabolized and thereby influencing the levels of nicotine present in the body. The rate at

which nicotine is metabolized in turn modifies smoking behaviour and risk for nicotine

dependence (Malaiyandi et al., 2005). Individuals who metabolize nicotine faster due to

greater enzyme activity have been found to smoke a greater number of cigarettes per day

(Malaiyandi et al., 2006), are more dependent on nicotine (Kubota et al., 2006), report

more severe cravings during nicotine replacement treatment (Lerman et al., 2006),

experience more withdrawal symptoms during abstinence from smoking (Kubota et

al., 2006), and are less likely to quit smoking (Gu, Hinks, Morton, & Day, 2000; Lerman

et al., 2006).

        Research in the field of pharmacogenetics is generating evidence that genetic

factors contribute to individual differences in the effectiveness of medication for smoking

cessation such as nicotine replacement therapy (NRT) and bupropion (Zyban) (Lerman et al., 2007). For example, one study found that smokers with the Asp40 variant of the mu-opioid receptor gene had approximately twice the quit rate with higher dose NRT (transdermal patch) than with lower dose NRT (nasal spray), while smokers with the Asn40 variant were equally likely to quit regardless of dose of NRT (Lerman et al., 2006). Gaining a further understanding of the role of genetic variation on individual outcomes with pharmacotherapeutic cessation aids may eventually allow healthcare providers the ability to tailor treatment type, dose, and duration based on a smoker's genetic profile in order to optimize treatment efficacy and improve quit rates (Lerman et al., 2007: Meiser et al., 2005).

Using genetic information to optimize treatment for nicotine dependence will require providing patients with feedback about testing for genetic variants and the implications for their smoking and treatment. At present, the clinical application of genetic assessment is limited: many genetic markers have not yet been discovered, most known markers make only modest independent contributions to disease risk and much remains to be learned about the contribution of markers that have been identified, including how they interact with environmental factors (Feero et al., 2008). However, as an increased number of genetic markers are identified and their contribution to disease onset and prognosis is further clarified, it is expected that they will be incorporated into clinical practice.

The discovery of genetic markers for disease and the ability to test for them has raised questions and concerns about what psychosocial and behavioural effects might follow from providing this information to patients. There has been a growing interest in

whether communicating the results of such tests encourages adoption of health-promoting behaviours such as early screening and medication adherence, as well as the uptake of risk-reducing behaviours such as smoking cessation (e.g., McBride, Koehly, Sanderson, & Kaphingst, 2010). Learning that one has a genetic predisposition and therefore is at increased risk for a health condition, may increase motivation to change behaviour, providing the behaviour is believed to have a positive impact on the outcome and that the individual has a sufficient degree of self-efficacy regarding their ability to carry out the behaviour (Sanderson & Wardle, 2005). This outcome is consistent with several models of health behaviour change that incorporate perceived susceptibility as a predictor of health behaviour performance (e.g., Health Belief Model; Abraham & Sheeran, 2005). Alternatively, the absence of a genetic vulnerability could also promote health behaviour change if it strengthens the belief that behaviour change would be effective at reducing risk (Sanderson & Wardle, 2005). It is also possible that providing genetic risk information will be detrimental and discourage behaviour change. For example, lack of control over genetic risk may lead to hopelessness, or receiving feedback of a "normal" or nonelevated risk may prompt a sense of false reassurance that discourages behaviour change (Collins, Wright, & Marteau, 2011).

Conducting research to determine whether such genetic tests will motivate lifestyle changes or whether they are associated with any adverse psychological or behavioural outcomes is necessary to determine their clinical utility and determine to what extent they provide any health benefits (Sanderson et al. 2008; Collins, Green, Guttmacher, & Guyer, 2003). Any beneficial or harmful outcomes would have important implications for their use (Sanderson et al., 2008). To date, several studies have examined

the impact of genetic testing on smoking behaviour and related determinants. Of those studies, most examined whether providing smokers with personalized genetic test results associated with lung cancer risk affected subsequent motivation to quit, number of quit attempts and/or cessation rates (Audrain et al., 1997; Carpenter et al., 2007; Hamajima, Atsuta, Goto, & Ito, 2004; Hamajima, Suzuki, Ito, & Kondo, 2006; Ito et al., 2006; Lerman et al., 1997; McBride et al., 2002; Sanderson et al., 2008).

The possibility that knowledge of increased genetic risk may engender *fatalism*, the belief that one has little or no control over a health outcome (e.g., "nothing I do will make a difference"), has been of particular concern. Research has shown that people tend to view health conditions associated with genetic causes as less controllable than those with psychosocial or environmental causes (Senior, Marteau, & Peters, 1999; Shiloh, Rashuk-Rosenthal, & Benyamini, 2002). Given that a greater level of perceived control is a predictor of motivation for and achievement of health behaviour change (Luszczynska & Schwarzer, 2005), there are concerns that conveying or reinforcing a sense that a health outcome is not controllable may adversely affect motivation to change behaviour (Wright, Weinman, & Marteau, 2003; Shiloh, 2006; Marteau et al., 2004; Wright et al., 2007).

A systematic review was recently conducted by Collins and colleagues (2011) in order to examine whether providing personalized genetic risk information has any impact on fatalism, which they operationalized as perceived control over prevention and/or treatment of the disease under study (obesity, heart disease, depression, or diabetes). Meta-analyses of neither the clinical nor analogue studies ($N = 5$) revealed any impact of genetic risk feedback on perceived control in either the short or longer term. Based on

these findings, the authors concluded that there was no evidence to suggest that personalized genetic risk information engenders feelings of fatalism among patients (Collins et al., 2011).

While the review by Collins et al. (2011) focused on studies that provided genetic risk feedback, Wright et al. (2007) chose to examine the impact of genetic causal attributions on perceived control and cessation rates among smokers making an NRT-assisted quit attempt without undergoing genetic testing. Results revealed that smokers who perceived genes as a cause of their smoking reported significantly lower perceived control over smoking compared to smokers who did not (Wright et al., 2007). Perceived control was operationalized in this study as self-efficacy to avoid smoking in tempting situations (e.g., with friends at a party). These same smokers had lower quit rates at all time-points over a one year period; however, the differences in quit rates did not reach statistical significance, but did approach significance at 26- and 52-week follow-up (ps < .10). A path model specifying genetic causal attributions as a predictor of quit success, with perceived control as a mediating variable, demonstrated a similar trend and was very close to significant at 52 weeks ($p = .056$). Attrition was not described and therefore it is unknown to what extent the initial sample and follow-up samples may have differed. However, if findings are valid and replicated in other samples, it suggests that genetic testing will impact perceived control to the extent that it alters underlying causal explanations for the health threat. Lower levels of perceived control in turn may lead to lower long-term quit success.

Smerecnik, Grispen, and Quaak (2011) recently conducted a meta-analysis of five randomized/quasi-randomized controlled trials to investigate whether genetic testing for

susceptibility to smoking-related disease has any impact on smoking cessation rates, as well as two proposed cognitive mediators of behavioural change – risk perception and motivation to quit. Results revealed that smokers who underwent genetic testing for smoking-related disease were significantly more likely to have quit smoking in the short term (less than 6 months) compared to smokers who had not undergone genetic testing, pooled OR = 1.9. However, this effect was no longer present at longer term follow-up (greater than 6 months). Subgroup analyses suggested that this effect was a result of smokers who tested positive (increased susceptibility to smoking-related disease) being marginally more likely to quit smoking than controls who did not undergo testing. Testing negative did not appear to adversely affect smoking cessation, rather a similar positive short-term effect on risk perception and motivation to quit smoking was also observed (Smerecnik et al., 2011). Therefore, it appears that genetic testing for susceptibility to smoking-related disease promotes temporary enhancement in smokers' motivation to quit smoking and short-term improvement in cessation rates, but was not effective in helping smokers to quit for good. Further research is needed to explore the potential to extend these short-lived successes so they are more enduring, and if so, by what means this can be achieved.

Based on a systematic review of the literature, McBride and colleagues concluded that the research to date "suggest[s] that genetic risk feedback will not add value to existing smoking cessation programs when very small increases in risk are conveyed, personalized risk feedback is based on a single genetic variant, or smokers have high pre-existing levels of motivation to quit" (McBride, Koehly et al., 2010). All but one of the four randomized trials reviewed (Ito et al., 2006; Lerman et al., 1997; McBride et al.,

2002; Sanderson et al., 2008) provided feedback on testing for a single gene variant, which generally confer only a minimal increase in risk for smoking-related disease. For example, as part of genetic testing feedback, smokers in the Sanderson et al. (2008) trial were informed that there is only a 3% difference (from approximately 8% to 11%) in absolute risk of lung cancer among participants with higher and lower risk GSTM1 gene variants. It is plausible that the small degree of risk communicated to smokers during testing for single gene variants is not sufficiently motivating, and that this has played a role in the findings to date.

One group of researchers has provided genetic risk feedback for multiple gene variants (Hamajima et al., 2004, 2006). In two separate studies, participants received feedback based on three gene variants. One study found that those with 0-1 higher-risk variants were less likely to have quit smoking compared with those with 2-3 higher-risk variants (4% vs. 17%) at 1-year follow-up (Hamajima et al., 2006), while an earlier study found no association between smoking cessation and number of higher-risk gene variants detected (Hamajima et al., 2004). Additional support that increased risk conveyed during genetic feedback translates into higher levels of motivation for behaviour change, comes from a study that provided smokers with feedback about alpha-1 antitrypsin (AAT) gene variants (Carpenter et al., 2007). While most severely AAT deficient smokers will develop emphysema, carriers for AAT deficiency have only a small increased risk of emphysema compared to the general population. Results revealed that smokers who learned they had severe AAT deficiency were more than three times more likely to report having made a quit attempt during 3-month follow-up compared to smokers who tested normal, while the rate of quit attempts among carriers did not significantly differ.

Smokers with severe AAT deficiency were also more likely than both carriers and noncarriers to seek information on treatment and use pharmacotherapy to assist with smoking cessation. While abstinence rates did not significantly differ at 3-month follow-up, smokers with severe AAT deficiency exhibited significantly greater reductions in their smoking (45%) compared to both carriers (19%) and noncarriers (11%). As an increased number of gene variants are discovered and their collective impact on risk determined, it will be increasingly possible to evaluate the impact of providing smokers with risk information of greater magnitude.

In addition to minimal risk conveyed in current genetic tests, McBride, Koehly, and colleagues (2010) also raised concern that several studies to date drew their samples from populations of smokers with pre-existing high levels of motivation, making the findings more difficult to interpret and less applicable to the general population of smokers. For example, Sanderson et al. (2009) and Lerman et al. (1997) offered genetic feedback within the context of a smoking cessation intervention, and thus those recruited were already motivated to quit smoking. Carpenter et al. (2007) also found that those most motivated to quit were those most likely to choose to undergo genetic testing. Finally, Sanderson et al. (2009) drew their sample from the biological relatives of recently diagnosed lung cancer patients. Under these conditions, providing information of heightened genetic risk may have diminished ability to further motivate cessation. Clearly, additional research is needed to address these limitations before conclusive statements can be made about the impact of genetic testing on motivational and behavioural outcomes.

To date, research examining the impact of genetic feedback on motivation to change smoking behaviour has focused on personalized feedback regarding risk for lung disease. However, smoking treatment tailored according to presence or absence of particular gene variants may be more likely to incorporate feedback regarding risk for nicotine dependence. For example, this may include a discussion of the role genotype plays in determining rate of nicotine metabolism, and the implications this has for risk of dependence, treatment efficacy, and anticipated treatment outcomes. Yet, there is an absence of research examining the impact of genetic feedback for risk of nicotine dependence on various motivational and behavioural outcomes for either smokers or nonsmokers (i.e., prevention). Beyond genetic testing for smoking-related diseases such as lung cancer, research is also needed to investigate the effects of providing genetic risk information regarding genetic predisposition to nicotine dependence.

A recent study on genetic testing for alcohol-related risks suggests that the outcomes of providing smokers with information regarding increased risk for nicotine dependence versus smoking-related disease may differ (Hendershot, Otto, Collins, Liang, & Wall, 2010). Hendershot et al. (2010) examined the impact of a genetic feedback intervention for alcohol-related health risks. The study provided feedback on the presence of the ALDH2*2 allele variant which leads to increased blood levels of acetaldehyde, a carcinogenic substance, following consumption of alcohol. Individuals homozygous for this allele experience strong physiological reactions to alcohol (e.g., flushing) and have minimal risk for alcohol dependence (Luczak, Glatt, & Wall, 2006). Individuals heterozygous for the allele also have decreased risk of alcohol use and dependence, but are at considerably increased risk for alcohol-related cancers (Yang, et al., 2010).

Hendershot et al. (2010) provided college students with web-based feedback regarding alcohol-related cancer risk and alcohol dependence based on ALDH2 genetic testing and conducted a follow-up to determine impact on subsequent drinking behaviour and other theoretically based correlates or determinants of behaviour change. Those who learned of increased risk of alcohol-related cancers and lower risk for alcohol dependence based on their genotype decreased the quantity and frequency of their alcohol consumption from baseline to 30-day follow-up to a greater extent than those randomized to attention-control feedback. The same group also demonstrated increased perceptions of risk and rated positive alcohol expectancies (e.g., "I would act sociable") as being less desirable compared to controls following the intervention. Those who learned of an increased risk for alcohol dependence in the absence of increased risk for alcohol-related cancer demonstrated increased fear arousal and perception of risk, and stronger intentions to change drinking behaviour, but did not modify their drinking behaviour.

Thus, learning of increased cancer risk prompted a change in behaviour, while learning of increased risk for alcohol dependence did not. The authors speculate that genetic risk information relating to cancer was likely perceived as more severe and therefore likely had a greater motivational effect. Cancer may also be perceived as having a greater genetic basis compared to alcohol dependence, and this may also alter the motivational impact of risk information provided. Comparable research is needed to examine whether a similar pattern of findings would emerge for genetic feedback regarding risk for nicotine dependence. If findings are similar, we might expect that providing smokers with information of heightened genetic risk for nicotine dependence

increases motivation for behaviour change; however, additional support may be necessary to translate this into actual quit attempts and success.

Identifying Mediating Factors Between Genetic Testing and Health Behaviour Change

The National Human Genome Research Institute convened an interdisciplinary group of scientists in October 2008 to recommend research priorities in the communication, behavioural, and social sciences in order to help facilitate the translation of genetic discoveries into public health applications (McBride, Bowen et al., 2010). Reporting on the discussion and recommendations of this meeting, McBride, Bowen and colleagues (2010) noted that "whether and how genetic information might add positively to existing interventions or produce counterproductive responses is still largely unknown" (McBride, Bowen et al., 2010, p. 561) and thus recommended that one of the top three research priorities should be to determine whether genetic information improves the adoption of healthy behaviours to a greater extent than current approaches (McBride, Bowen et al., 2010). McBride, Bowen and colleagues further recommended that social and psychological theory could be used to frame these research questions and that:

consideration should be given to the mechanisms through which genetic communication might influence health behaviors. Genetic-risk feedback might have its greatest influence on intermediate factors such as emotional responses (e.g., fear or worry), motivation to seek formal interventions, self-confidence to adhere to an intervention (self-efficacy), or beliefs that interventions will or will not be effective (response efficacy). (p. 561)

One such factor that may act as a mediator and/or moderator of behavioural outcomes following genetic testing is causal attributions, the implicit and explicit causal

explanations for the condition(s) or behaviour(s) involved.

Aside from altering level of perceived risk, genetic testing for a condition may alter the extent to which an individual perceives genetic makeup as a cause for that condition, such as nicotine dependence or smoking-related disease. Interviews with patients have revealed that information communicated in genetic counselling is modified and integrated with their own lay explanations for a condition (Skirton, 2001). Skirton (2001) found that individuals may have an accurate understanding and recall of genetic information presented, yet simultaneously express personal beliefs about the condition that deviate from that knowledge (Skirton, 2001). Thus, while causal beliefs may change, the extent to which they do is likely to vary across individuals based in part on how they fit into their existing lay explanations for a condition, and it cannot be assumed that subsequent causal beliefs are entirely representative of what was communicated when genetic test results were provided.

Any changes to causal attributions that may occur in turn have the potential to affect other cognitive and motivational factors, which may ultimately impact behaviour. To date, causal attributions have received little attention in the literature examining the impact of genetic testing on health-related outcomes and cognitive outcomes. Yet, causal attributions have the potential to act as a mediator and/or moderator of outcomes and may help elucidate the relationship between genetic testing and behavioural and motivational outcomes. For example, an observed decrease in perceived control following genetic testing could be hypothesized to be a function of the degree to which attributions to genetic causes have changed, to the extent that genetic causes are seen as uncontrollable. If at baseline an individual has a pre-existing strong belief in genetic causes for a

condition, providing genetic test results is not as likely to impact causal attributions to the same degree as for someone who has focused their causal beliefs on behavioural (or other) factors. This may account in part for the fact that some studies have found that genetic testing per se had a motivational impact (at least in the short term) on health outcomes, including smoking, rather than whether someone tested positive or negative for a particular gene variant (McBride et al., 2002; Sanderson et al. 2008). The fact that genetic testing has been developed and is available for a particular condition may send an implicit message about the importance of genetic factors in the etiology of that condition, which in and of itself may have consequences for anyone participating in testing. Alternatively, simply participating in genetic testing may heighten the salience of genetic makeup as a causal factor for that individual, if perhaps only temporarily.

Relationship Between Causal Attributions and Health Outcomes

Part of the importance of assessing causal attributions lies in the fact that people do not act on objective evidence, but rather on their own lay explanations and beliefs relating to health and illness, of which causal attributions play a significant part (Leventhal, Brissette, & Leventhal, 2003). Evidence has accumulated across a wide range of conditions that causal attributions are predictive of a number of health-related outcomes, including health behaviour (e.g., Locher, Burgio, Goode, Roth, & Rodriguez, 2002; Runions, Arnaert, & Sourial, 2006; Weinman, Petrie, Sharpe, & Walker, 2000); coping, psychological adjustment and well-being (e.g., Else-Quest, LoConte, Schiller, & Hyde, 2009; Moss-Morris, Petrie, & Weinman, 1996; Roesch & Weiner, 2001); treatment seeking and adherence to treatment (e.g., Brewer et al., 2000; Locher et al., 2002; Orbell, Hagger, Brown, & Tidy, 2006); and physical health and functional status (e.g., Bar-On,

Gilutz, Maymon, Zilberman, & Cristal, 1994; Schiaffino & Revenson, 1992; Segerstrom, Taylor, Kemeny, Reed, & Visscher, 1996; Wearden, Hynd, Smith, Davies, & Tarrier, 2006).

For example, causal attributions have been found to predict changes in diet and physical activity following myocardial infarction (Weinman et al., 2000), recurrence of myocardial infarction up to eight years later (Low, Thoresen, Pattillo, & Fleischmann, 1993), and subsequent morbidity (Affleck, Tennen, Croog, & Levine, 1987; Low et al., 1993). Researchers also discovered that attributions for recent hypo- or hyperglycemic episodes among type 1 diabetics predicted level of glycemic control over a period of one year, and this association was partially mediated by self-management behaviours such as insulin administration, glucose testing and following dietary guidelines (Wearden et al., 2006). While the vast majority of research examining the role of causal attributions in health-related areas has focused on individuals with diagnosed disease, causal attributions also have been found to predict motivation to adopt preventative health behaviours among healthy individuals (Figueiras & Alves, 2007).

Thus, it is evident from the existing literature that beliefs people hold about the causes of health threats can have an effect at each stage of clinical management (Sensky, 1997). An analysis of attributions people form for health threats may therefore allow both researchers and clinicians to better understand and predict patients' cognitions and behaviours directed toward both maintaining or regaining good health.

Theoretical Frameworks for Examining Causal Attributions

Most of the research to date examining the implications of causal attributions for health has been conducted using one of two theoretical frameworks, both of which are

described below.

   Leventhal's Common Sense Model. According to Leventhal's Common Sense Model (CSM) of self-regulation of health and illness (Leventhal, Brissette, & Leventhal, 2003; Leventhal, Meyer, & Nerenz, 1980), people form cognitive representations of health threats in order to make sense of them and to manage them. Health risk information is thought to activate threat representations (also referred to as illness representations), which in turn guide the identification and execution of action plans to eliminate or minimize the threat (Leventhal et al., 1980, 2003). The CSM posits a parallel process model whereby people form simultaneous cognitive and emotional representations of a threat (see Figure 1). As cognitive representations guide actions to manage health threats, emotional representations guide activities to regulate emotions evoked by health threats (Leventhal et al., 1980, 2003). Individuals evaluate the efficacy of their efforts to regulate the health threat and corresponding emotions and incorporate this feedback into their representations.

   In addition to personal experience with an illness or health threat, threat representations are thought to change over time based on information acquired through family, friends, health care providers, media, and cultural beliefs (Leventhal et al., 1980, 2003). Given that personal experience is unique, very different representations may emerge among people with seemingly similar experiences. In turn, these variations are expected to evoke different responses among patients exposed to the same threat. Thus, a personalized approach is needed to ascertain how each patient understands a particular health threat.
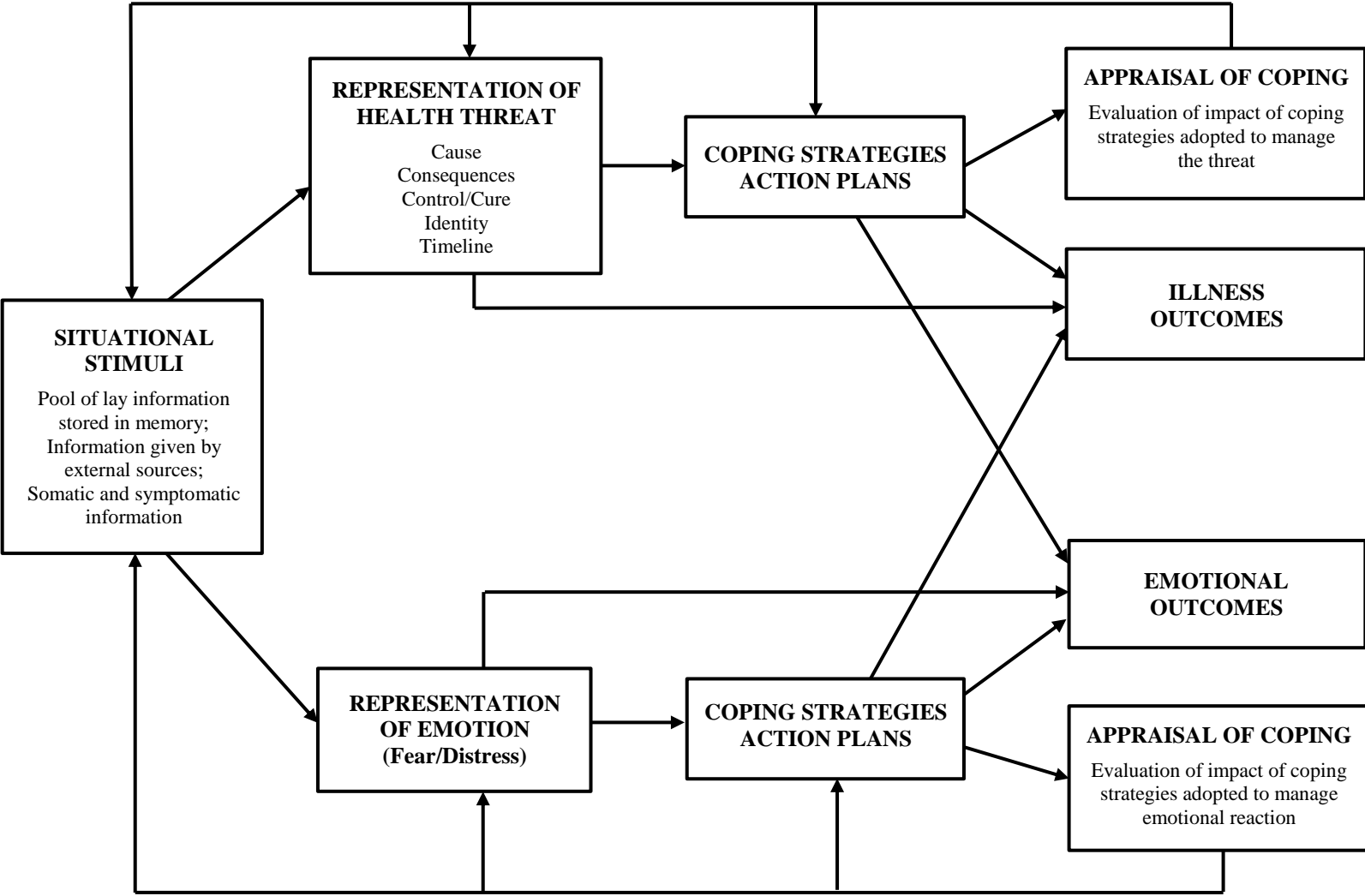
Figure 1. Leventhal's Common Sense Model of self-regulation of health and illness.

The content of threat representations is believed to be comprised of five dimensions: (1) *identity*, beliefs about the threat label (e.g., "I think I have the flu") and the symptoms associated with it; (2) *causes*, beliefs about what factors are responsible for causing a particular illness; (3) *consequences*, the expected physical, psychological and social sequelae of an illness (e.g., "I cannot go to work while I have the flu"); (4) *timeline*, expectations about the duration and course of the illness; and (5) *controllability/cure*, beliefs about the extent to which the illness can be controlled or cured, including the efficacy of coping behaviours and treatment.

How exactly the dimensions interact and affect each other has not been well articulated within the CSM (Marteau & Weinman, 2006). The pattern of intercorrelations that emerged between the CSM dimensions in a meta-analysis of empirical studies that adopted the CSM as a framework showed that the dimensions of consequences, control/cure, identity and timeline followed a pattern that supported their construct and discriminant validity across a variety of illness types (Hagger & Orbell, 2003). The cause dimension was excluded from the analysis due to methodological issues stemming from wide variation in causes between different illnesses. After controlling for associations with other dimensions, bivariate correlations revealed that participants who viewed their illness as having a strong illness identity viewed that illness as less controllable and as having more serious consequences; and patients who saw themselves as having an illness with a more acute timeline perceived the illness as having fewer serious consequences and saw themselves as having a greater degree of control over the illness. That some bivariate correlations were attenuated or nonsignificant when associations with other dimensions were controlled for demonstrates the complexity and interdependent nature of

the relationships between the dimensions (Hagger & Orbell, 2003). Despite the fact that the cause dimension was not included in the meta-analysis, findings suggest that given this interdependence among the other four dimensions, we can expect variation in the causal domain of threat representations to impact other dimensions as well. For example, illnesses with genetic origins may be regarded as having more severe consequences or being less controllable (Shiloh, 2006). In what ways causal beliefs in general, and genetic causal beliefs in particular, relate to other components of illness representations requires further examination.

According to the CSM, threat representations act as a filter and an interpretive framework for incoming stimuli, that guides action taken to manage health threats (Leventhal et al., 1980, 2003). Threat representations have been shown to predict coping responses to illness, including decisions to seek health care (e.g., Grunfeld, Hunter, Ramirez, & Richards, 2003; Walsh, Lynch, Murphy, & Daly, 2004) and adherence to medical interventions and recommended self-management behaviours (e.g., Griva, Myers, & Newman, 2000; Horne & Weinman, 2002). The above-mentioned meta-analysis conducted by Hagger and Orbell (2003) found some support for a consistent pattern of relationships between individual dimensions and particular types of coping behaviour (Hagger & Orbell, 2003). For example, greater perceived consequences, stronger illness identity, and perception of a more chronic illness were significantly correlated with use of avoidance/denial, while greater perceived control/cure beliefs were positively associated with use of problem-focused coping, cognitive reappraisal, and seeking social support.

Evidence also exists linking threat representations with illness and emotional outcomes, including psychological distress and well-being (e.g., Fortune, Richards, Griffiths, & Main, 2002; Kemp, Morley, & Anderson, 1999), and indicators of physical functioning and disease status (e.g., Griva et al., 2000; Heijmans, 1999; Scharloo et al., 1998). Hagger and Orbell (2003) also examined and found evidence of significant associations between each of the four dimensions included and indicators of both psychological well-being and physical functioning. For example, stronger cure/control beliefs were significantly correlated with increased psychological well-being, social functioning and vitality, and were inversely related to psychological distress and disease state. Conversely, beliefs in a more chronic timeline, more serious consequences, and a stronger illness identity, were associated with lower adaptive and increased maladaptive illness and emotional outcomes (Hagger & Orbell, 2003). The small number of studies available for analysis prevented the authors from being able to test the CSM's proposition that illness representations impact outcomes indirectly via their direct impact on coping strategies (Leventhal et al., 1980).

Like other components of illness representations, causal beliefs can be further delineated (Shiloh et al., 2002). Attribution theory provides a framework to understand better the process of how causal beliefs drive and motivate both behaviour and affect when one is faced with a health threat.

Weiner's attribution theory. As there is no single, generally accepted attribution theory, the term *attribution theory* refers to a collection of models and theories that are concerned with the process by which people form inferences about the cause of events (i.e., causal attributions) and the consequences of these causal explanations (Fiske &

Taylor, 1991). The focus of attribution theory is on perceptions of causation rather than actual causes or accuracy of causal beliefs, as these perceptions will influence subsequent cognitive, affective and behavioural responses. Attribution theorists assert that people are motivated to make causal attributions in order to not only gain an understanding of their world and past events, but also to guide future behaviour and enable greater predictability and control over their environment (Harvey & Weary, 1984; Kelley, 1971). By locating a cause, individuals may assess which particular actions may be taken, if any, to minimize the recurrence of negative outcomes in the future, as well as maximize the probability of desirable outcomes (Weiner, 1985).

According to Weiner's attributional theory of motivation and emotion (Weiner, 1985), causal attributions can be classified along three main dimensions: *locus*, *stability*, and *controllability*, each of which has predictable psychological and behavioural consequences. An internal locus of causality implies the behaviour was a result of volitional or dispositional factors originating from the actor while an external locus implies that contextual factors compelled or constrained the actor to behave in a particular way. It is important to note the distinction between internal versus external locus of causality and Rotter's (1966) internal versus external *locus of control* construct. While an internal locus of causality implies that the cause is internal to the individual, it need not be under the individual's control. For example, while the locus of a genetic cause is classified as internal, genetic composition is not considered to be within an individual's control. The stability of a cause refers to its perceived degree of permanence over time. Controllability indicates the degree to which a cause is perceived to be under our volitional control (Weiner, 1985). A single event may have multiple causes, located

differentially on one or more of the three dimensions. Perceived causality may vary not only between individuals, but also within an individual at different times, both for the specific cause itself and the location of a specific cause on a given dimension (Weiner, 1985).

Weiner (1985) proposed that each causal dimension is linked to a specific set of emotions generated by an event outcome. In particular, the locus of a cause is believed to have implications for an individual's feelings of pride or self-esteem, such that positive outcomes ascribed to the self are associated with increased self-esteem and pride whereas decreased self-esteem and shame are experienced when a negative outcome is attributed to the self. Attributing a stable cause to an outcome is expected to intensify affect, and negative stable outcomes in particular are expected to generate feelings of hopelessness or anxiety. Negative outcomes attributed to causes believed to be under our control (internal, controllable) are expected to elicit feelings of shame and guilt whether the recipient of the outcome is ourselves or others, whereas if the cause was under another person's control it is expected to lead to anger. Positive outcomes resulting from causes under another individual's control (external, controllable) are expected to yield gratitude. Where causes are not controllable, outcomes are expected to be attributed to luck and may intensify initial emotions of happiness or sadness. However, while Weiner (1985) expected these dimension-affect relationships to be "quite prevalent" (p. 564), they are not considered invariant and do not always follow, nor are these attributions considered a necessary precondition for the experience of these emotions.

According to Weiner's attribution theory, causal attributions are thought to have a direct impact on expectancy of successful goal attainment (Weiner, 1985). Specifically,

Weiner maintains that following an outcome, changes in expectancy of success in the future will occur based on the perceived degree of stability of the cause of the event. If the outcome of an event is ascribed to a stable cause, then that outcome experienced in the past will be anticipated with increased expectancy, or expected to recur, in the future. Thus, success ascribed to a stable cause (e.g., "I quit smoking because it's in my nature to always try hard and succeed at everything I do"), would increase anticipation of future success and failure ascribed to a stable cause would strengthen expectation of subsequent failure (e.g., "I failed at quitting smoking because I am genetically predisposed toward addiction like my father"). On the other hand, there is likely to be greater uncertainty about future outcomes if causal factors are perceived as unstable. Thus, while instability for the cause of a negative event would be desirable, it would be discouraging for the cause of a positive event as one would be less confident that it would recur in the future.

Causal attributions are purported to exert an indirect influence on behaviour via their impact on expectancy and affect, which are conceptualized as direct determinants of behaviour (Weiner, 1985). Greater expectancy for success is expected to result in engaging in behaviour to reinstate a cause. Persistence may be further enhanced by feelings such as guilt, a consequence of greater perceived controllability. Alternatively, one may cease trying if expectations of future success are low and feelings of hopelessness are high.

Given the above, knowledge of where a causal attribution lies on all three dimensions leads to predictable emotional, cognitive and behavioural outcomes. For instance, internal, unstable and controllable attributions for negative outcomes, such as a belief that poor diet resulted in a heart attack, should lead to lowered self-esteem (internal

locus), feelings of guilt but a belief that something can be done (high controllability), and increased expectation of coping success since diet is very modifiable (low stability). Taken together then, we would expect motivation toward persistent and intense action to resolve or address the adverse outcome. Conversely, external, stable and uncontrollable attributions, such as a belief that a polluted environment causes asthma, should preserve self-esteem (external locus), decrease expectation for recovery (high stability), and result in beliefs that nothing can be done (low controllability). Hence, this pattern of attribution is more likely to result in helplessness and resignation, with little likelihood of motivating any coping response to attempt to address the problem (Roesch &Weiner, 2001).

While Weiner's model was originally developed and received a lot of empirical support in the context of achievement-related behaviour, Weiner (1985) suggested and cited evidence that the model had a much wider range of applicability and that the motivational sequence outlined would follow any outcome that can be construed as attainment or nonattainment of a goal, including what Weiner referred to as "personal or social 'failures'" (p. 567) such as depression, loneliness and smoking. To date, attribution theory has been used across a wide range of fields including education, sports psychology, clinical and counselling psychology, and health psychology.

Using Weiner's attribution theory as a conceptual framework, Roesch & Weiner (2001) conducted a meta-analysis to test a model of the relations between causal attributions, coping strategies and psychological adjustment among medical populations, including individuals with a physical illness or those undergoing a medical procedure. In the context of a health threat or condition, Weiner's theory (1985) would predict that attribution for the cause plays a role in determining whether efforts are made to manage

or eliminate the threat or condition, and if so, which types of behaviour, or coping

methods, are utilized. Causal attributions were hypothesized to have both a direct effect

on psychological adjustment and an indirect effect through the use of various coping

strategies. Findings generated support for the model. Individuals who explained the cause

of their illness as more internal, unstable and controllable reported using both more

approach forms of coping and emotion-focused coping, and were more well-adjusted as a

result (Roesch & Weiner, 2001). In contrast, individuals who made more stable

attributions reported using more avoidant coping methods, which was associated with

poorer psychological adjustment. Individuals who made internal, unstable and

controllable attributions were presumed to have high expectations regarding their ability

to reverse or minimize the impact of their illness. Thus, this pattern of attributions

motivated coping responses that ultimately resulted in better psychological adjustment.

On the other hand, those who made stable attributions were presumed to believe that little

or nothing could be done to prevent or minimize any negative effects of the illness,

leading to unmotivated cognitive and behavioural responses (avoidant coping strategies),

and ultimately to poorer psychological adjustment (Roesch & Weiner, 2001).

The following section considers the impact of attributions to genetic causes on

one form of coping response, seeking treatment.

Causal Attributions, Perceived Treatment Effectiveness and Treatment Seeking

According to Leventhal's Common Sense Model (Leventhal et al., 1980, 2003),

perceptions of the cause of a health threat guide the selection of coping procedures in a

direct fashion, using what Leventhal referred to as "if–then" rules (Leventhal et al.,

1997), which are based on how that threat is believed to be avoided or controlled. For

example: *if* high blood pressure is caused by being unfit *then* exercise will reduce it. Given that methods to address genetic causes directly are not available, it has been suggested that threat representations with genetic causes are likely to initiate one of two responses: (i) if seen as immutable, genetic causes may lead to the belief that there are no coping responses that can eliminate or reduce the threat, prompting inaction; (ii) biological-based actions (e.g., taking medication) may be viewed as more effective at controlling the threat compared to behavioural-based methods (Marteau & Weinman, 2006). Therefore, if genetic testing for a condition results in or strengthens genetic causal explanations, either one of these responses may be increasingly observed. Some support for the second response comes from a study by Senior and Marteau (2007), who found that providing genetic testing feedback of increased predisposition to heart disease increased the extent to which the condition was perceived as being caused by genetic make-up (Marteau et al., 2004), which in turn reduced the expectation that a low fat diet would be effective and increased the expectation that lipid lowering medication was effective.

An assessment of the impact of genetic risk information on the perceived effectiveness of interventions for various health problems (obesity, heart disease, depression, and diabetes) was a secondary aim of the systematic review by Collins et al. (2011) described earlier. Data from the one clinical and three analogue studies included in the review found no effect of personalized genetic feedback on perceived effectiveness of behavioural interventions. Only one clinical and one analogue study examined the impact of genetic information on perceived efficacy of medical interventions, of which, the clinical study found no effect and the analogue study (Wright et al., unpublished

study, as cited in Collins et al., 2011) found a small effect indicating that individuals who received genetic feedback perceived medical interventions as more effective compared to those who did not receive personalized genetic feedback.

An analogue study conducted by Wright et al. (2003), not included in the above systematic review, provides some evidence that learning of a genetic predisposition may result in greater endorsement of biological, versus behavioural, means of reducing risk. Smokers who were asked to imagine having tested positive for a genetic susceptibility to nicotine dependence did *not* perceive themselves as having less control over stopping smoking compared to smokers who imagined having tested negative. However, they were more likely to report that they would use the medication Zyban to assist with quitting and were less likely to rely on willpower.

Similar findings have been reported for individuals undergoing genetic testing for familial hypercholesterolemia (FH), a hereditary disorder associated with high LDL cholesterol and risk for early cardiovascular disease that can be modified through lifestyle change and medication. Those for whom FH was confirmed by genetic testing believed more strongly six months later that their cholesterol levels were determined by genetics, believed less strongly in the efficacy of lowering dietary fat intake and believed somewhat more strongly ($p = .06$) in the efficacy of medication to reduce cholesterol level (Marteau et al., 2004). These same individuals however did not exhibit lower perceived control or increased fatalism over FH, cholesterol levels, or heart disease and did not differ on perceived relative risk of a heart attack. Therefore, genetic testing appeared to diminish beliefs in the effectiveness of behavioural risk-reduction strategies while somewhat strengthening beliefs in the effectiveness of biological strategies. While

attitudes and beliefs changed, no differences in behaviour (smoking, diet, exercise, and medication adherence) emerged. However, participants were already aware of their family history of FH and their increased risk and were generally already engaged in treatment and risk-reducing behaviours to reduce their elevated cholesterol levels.

Further analysis from this study (Senior & Marteau, 2007) revealed that aside from genetic testing results, attributions to genetic causes were associated with perceiving medication as an effective method to reduce risk, while attributions to behavioural causes were related to perceiving changes to diet as effective for reducing risk. Perceived effectiveness of medication was associated with greater adherence to medication in cross-sectional, but not longitudinal, analyses. Perceiving dietary intervention as more effective was not associated with dietary fat intake in either cross-sectional or longitudinal analysis. However, the authors note that among their sample dietary fat intake was already very low at baseline given that participants were treatment seeking patients clinically diagnosed with FH, limiting the variance in this measure. Whether a similar pattern of results would be found among samples not already engaged in risk-reducing behaviour remains to be seen.

Similar findings have been reported for mental health disorders as well. Phelan, Yang, and Cruz-Rojas (2006) found evidence that pharmacological treatments are seen as more effective when mental illness is attributed to genetics. Two vignette studies were conducted with nationally representative samples and results showed that respondents who thought genetic factors contributed to the psychiatric problem presented (either major depression or schizophrenia) were twice as likely to recommend prescription medication and psychiatric hospitalization for the individual described in the vignette, but

were no more likely to recommend seeing a psychiatrist, therapist, or general medical practitioner. Hence, results suggest that genetic attributions for a mental illness are associated with increased confidence in more extreme (psychiatric hospitalization) or biomedical (medication) forms of treatment, but not consultation with any type of mental health or medical professional. Similar findings have been reported with other samples, whereby biological causes for mental illness were associated with greater perceived effectiveness of biological treatments (e.g., medication), and likewise for psychological causes and perceived effectiveness of psychological treatments (e.g., psychotherapy) (Iselin & Addis, 2003; Whittle, 1996).

In light of findings that individuals maintain a sense of control following genetic testing over a health threat while perceived effectiveness of treatment is modified (Marteau et al., 2004; Wright et al., 2003), Marteau and colleagues formed the hypothesis that when a risk is modifiable, personalized genetic risk information does not alter the extent to which people perceive they have control over a condition or health threat, but instead seems to alter their appraisals of how control can be most effectively achieved (Marteau et al., 2004). In order to investigate this possibility further, it has been recommended that future studies include measures of *both* perceived control and perceived treatment effectiveness in order to better evaluate the relationship between these two variables (Collins et al., 2011; Marteau & Weinman, 2006). A thorough investigation of this question would also benefit by including an assessment of perceived treatment control, the belief that medications or health professionals can control a health condition (Wright et al., 2007).

An initial attempt to gain a better understanding of mechanisms by which people associate genetic causal information with increased effectiveness of pharmacological interventions and reduced effectiveness of non-pharmacological interventions was conducted recently by Wright et al. (2012). Participants were randomly assigned to read an experimental vignette that varied by health condition (heart disease, obesity or depression), severity (high, low), and cause (environmental, family history, genetic test, family history + genetic test), and were subsequently asked to rate the effectiveness of both pharmacological and nonpharmacological treatments for the health condition presented. Where the manipulations had a significant effect on perceived effectiveness, multiple mediation analysis was used to identify whether perceived severity, causal attributions, or perceived control mediated the impact of the manipulation on perceived effectiveness. The findings varied between each health condition. For heart disease, participants who read vignettes outlining genetic causes reduced their subsequent ratings of perceived effectiveness of nonpharmacological treatments, but not perceived effectiveness of medication. The relationship between the vignette cause manipulation and the perceived effectiveness of nonpharmacological treatment was mediated by both causal attributions and perceived control. Thus, mention of environmental causes in the heart disease vignette was associated with a stronger perception of the hypothetical patient having control over his heart disease and greater endorsement of lack of exercise and poor diet as causal factors, which both in turn increased perceived effectiveness of diet and physical activity as treatment. With respect to depression, genetic causes only increased perceived effectiveness of medication for more severe depression, an effect mediated by perceived control. Cause and severity manipulations did not impact

perceived effectiveness of treatment for obesity. Findings provide some preliminary evidence that variation in perceived treatment effectiveness may be partially due to changes in perceived control. However, contrary to earlier findings that perceived control remained stable following real or imagined genetic testing while perceived effectiveness changed (Marteau et al., 2004; Wright et al., 2003), this study found that perceived control had been affected by the manipulation of cause for both heart disease and depression vignettes. Thus, further research is necessary to determine whether the findings are replicated in other samples and for other health conditions, as well as under real-life conditions.

If further research concludes that genetic testing or genetic causal beliefs are associated with increased perceived effectiveness of biological based treatment and decreased perceived effectiveness of psychosocial treatments, certain concerns may be raised about how the genetics revolution will impact treatment seeking for mental and physical health problems. If attributions to genetic causes increase in frequency, help seeking may shift increasingly toward biological interventions. While this outcome may be adaptive by encouraging the use of effective, biologically based ways of reducing risk such as taking medication, it may be maladaptive if it leads to diminished acceptance and use of effective behavioural methods. It may also be problematic if lower confidence in behavioural methods is accompanied by smokers having less confidence in their own ability to cope with nicotine cravings and temptations (Wright et al., 2003).

If this is the case, then it may be necessary to develop interventions to prevent genetic testing feedback from having a negative effect on use of behavioural risk reduction strategies (Senior & Marteau, 2007). Such an intervention may include

attempts to broaden the individual's causal model so that it includes the interaction of both genetic and behavioural causes to attempt to increase the perceived relevance of behaviour change for reducing risk, but not diminish the value of biologically based treatments (Marteau & Weinman, 2006). Further research identifying mechanisms mediating genetic testing and treatment decisions will provide some indication of the cognitions that need to be targeted to encourage health promoting behaviour and to optimize the motivational impact of genetic risk information (Marteau & Weinman, 2006).

Seeking Smoking Cessation Treatment

Despite their established efficacy, the majority of quit attempts are made without the assistance of smoking cessation treatment (Cokkinides et al., 2005; Shiffman et al., 2008). For example, a survey of American smokers found that approximately 64% who made a quit attempt in the previous year did so without any assistance (Shiffman et al., 2008). A recent survey of smokers in the United Kingdom found that of those who made a quit attempt in the past 12 months, just over half (51%) had used some form of intervention (Kotz, Fidler, & West, 2009). When smokers do choose to seek treatment, stop smoking medications (including NRT) are the most commonly used, while fewer choose psychosocial treatments such as group or individual counselling (Kotz et al., 2009).

Insight into some of the determinants of decisions to use smoking cessation medications and behavioural stop-smoking support has been provided by recent research conducted by Vogt and colleagues (Vogt, Hall, & Marteau, 2008, 2010). Semi-structured interviews conducted with 27 smokers revealed that the decision to use a particular

smoking cessation intervention was influenced by a smoker's beliefs regarding that intervention's: (i) effectiveness, (ii) desirability, and (iii) accessibility.

Accessibility concerns for smoking cessation medications included the cost of NRT and the effort and time required to get a doctor's prescription (Vogt et al., 2008). Certain features of smoking cessation medications also made them undesirable to use. Some smokers felt reliance on medication reflected an inability to deal with their own problems and therefore meant they had failed or were weak-willed and would lead to a diminished sense of achievement upon quitting smoking. Smokers also anticipated a range of adverse physical effects from using medication.

A few different reasons were given for why medications were perceived as ineffective in helping smokers quit. Some smokers did not believe that medications would control their cravings. Another reason some smokers gave was that NRT and bupropion had little or no ability to increase willpower, which was deemed an important factor in resisting cravings and remaining abstinent. Finally, medications were viewed as unable to address reasons for smoking other than nicotine dependence (e.g., stress, boredom, social reasons), which was especially important for smokers who did not perceive themselves as addicted to nicotine (Vogt et al., 2008).

Accessibility barriers for group support included travelling to meetings and difficulty coordinating meeting times with one's schedule, waiting lists, and finding out where group support was provided (Vogt et al., 2010). Similar to using medication to help quit, using behavioural support was perceived as a personal failure because the expectation was that they should be able to quit without any outside assistance. Additional undesirable aspects of behavioural support mentioned included the

expectation that group members would be annoying, and being uncomfortable and inhibited in a group setting (Vogt et al., 2010).

With respect to the effectiveness of behavioural support, some smokers expected group and individual support to be helpful by providing social support, information and advice, and additional motivation to quit smoking, such as to avoid disappointing others (Vogt et al., 2010). However, some smokers did not expect to learn anything from counselling and believed that talking about smoking would not resolve their issues and therefore would not help them quit smoking. In particular, they failed to see how talking about smoking could help reduce their cravings for cigarettes. Some also expected that group advisors/facilitators had never smoked and would not understand them. Similar to findings for medication, some smokers perceived neither group nor individual support as a source of the willpower perceived as crucial for stopping smoking.

Based on the above qualitative findings, Vogt and colleagues conducted a survey of 212 smokers in England to estimate the frequency of these outcome expectations and their relative importance in predicting smokers' motivation to use smoking cessation medications and behavioural stop-smoking support (Vogt et al., 2008, 2010). Survey findings revealed that 35% of smokers intended to use NRT to quit smoking, while only 10% intended to use bupropion (Vogt et al., 2008). Demographic variables and level of nicotine dependence were not associated with intentions to use pharmacological aids. Only 41% of smokers believed that NRT would be effective at helping them quit smoking and even fewer, 20%, believed that bupropion would be effective (Vogt et al., 2008).

Regression analysis revealed several independent predictors of intention to use NRT. Namely, smokers were less motivated to use NRT if they were high in self-efficacy to stop smoking, expected NRT to cause adverse effects, had not used NRT in the past, and did not anticipate that NRT would control cravings and help them stop smoking. Effectiveness outcome expectations (e.g. ''NRT would increase my chances of stopping smoking") were the second strongest predictor of intention to use NRT, following past use of NRT. Further mediation analysis revealed that effectiveness outcome expectations significantly mediated the effect of craving control outcome expectations ("NRT would reduce my cravings for cigarettes") on intentions to use NRT. Thus, it appears that expectations of effectiveness were determined mainly by the perceived ability of nicotine dependence medications to control cravings.

Independent predictors of intentions to use bupropion indicated that smokers were less motivated to use bupropion if they had weaker intentions to stop smoking, had an aversion to medication ("taking bupropion would be putting unnecessary drugs into my body"), and did not expect that bupropion would help them quit. Similar to findings for NRT, mediation analysis found that effectiveness outcome expectations were determined mainly by expectations that bupropion would control cravings. Effectiveness outcome expectations emerged as the strongest predictor of intentions to use bupropion.

The same survey found that 21% of smokers intended to use group support for their next quit attempt and 12% intended to use individual support (Vogt et al., 2010). As with smoking cessation medication, demographic variables and level of nicotine dependence were not associated with intentions to use behavioural support. A total of 30% of respondents expected that using group support would help them stop smoking

and 26% believed the same for individual support (Vogt et al., 2010).

Effectiveness outcome expectations also emerged as independent predictors of both group and individual support, in addition to other predictors (intentions to stop smoking for both types of support, self-efficacy to stop smoking and annoyance outcome expectations for group support). Once again, perceived effectiveness emerged as a strong predictor of intentions to use behavioural support (Vogt et al., 2010). Mediation analyses revealed that both self-efficacy to use group or individual support and support outcome expectations (expectation that one would get valuable support and encouragement and would learn useful things about quitting smoking from the advisor and/or other group members) predicted intentions to use group and individual support, respectively, through effectiveness outcome expectations (Vogt et al., 2010).

In summary, a large number of smokers did not expect that using cessation support or medication would be effective to help them stop smoking and perceptions of effectiveness were strong predictors of intentions to use these interventions to assist with a quit attempt. Findings were consistent with other studies that have examined determinants of smokers' motivation to use pharmacological stop-smoking support (Etter & Perneger, 2001; Shiffman, Ferguson, Rohay, & Gitchell, 2008). For example, Etter and Perneger (2001) found that only 16% of participants agreed that "NRT helps people quit smoking". Shiffman and colleagues found that only 27% of smokers believed that NRT was effective and perceived effectiveness predicted intentions to use NRT during their next quit attempt (Shiffman et al., 2008). Hammond, McDonald, Fong, and Borland (2004) found that many Canadian smokers did not believe the following cessation methods would increase their likelihood of quitting: nicotine replacement therapies

(36%), bupropion (35%), group counselling (50%), and counselling from a health professional (66%). In addition, 78% of smokers believed that they were just as likely to quit on their own as they were with assistance. Participants who perceived cessation methods to be effective at baseline were almost twice as likely to intend to quit and to have made a quit attempt at 3-month follow-up, and were more than three times more likely to have adopted cessation assistance when doing so (Hammond et al., 2004).

Causal Attributions and Smoking

The following is a review of the research conducted to date on causal attributions for smoking. In particular, the research has been directed toward understanding how attributions made by individuals who have quit smoking play a role in whether they relapse or successfully maintain abstinence from smoking.

Smoking relapse. To date, research investigating the implications of variations in causal attributions that smokers make has focused primarily on the role that attributions play in the relapse process following a quit attempt. Marlatt and Gordon's (1985) model of the smoking relapse process proposes that attributional processes are an important determinant of whether an initial slip or lapse (smoking one or more cigarettes after quitting) will lead to a full-blown relapse (resuming regular smoking). This attribution process is part of a cognitive-affective reaction referred to as the *abstinence violation effect* (AVE), conceptualized as being comprised of two factors: (a) a causal attribution for the slip, and (b) an affective reaction to the attribution. Marlatt and Gordon (1985) proposed that the intensity of the AVE is increased when causal attributions for a slip are internal, stable, uncontrollable, and global (e.g., lack of willpower), accompanied by emotional reactions of guilt and self-blame. Self-blame may undermine perceived

efficacy and hinder recovery following a slip (DiClemente, 1986). Conversely, the intensity of the AVE is decreased by external, unstable, controllable, and specific causal attributions (e.g., a failure to use effective coping skills in a specific situation). The global-specific dimension originates from learned helplessness theory (Abramson, Garber, & Seligman, 1980) and refers to the extent to the which the cause is thought to be specific to one situation versus generalizing across multiple situations (e.g., "I have difficulty resisting the temptation to smoke when I feel stress" vs. "I always end up giving in to my cravings").

Several independent studies have gathered evidence in support of an abstinence violation effect as proposed by Marlatt & Gordon (1985). For example, Curry, Marlatt, and Gordon (1987) found that smokers who relapsed following an initial lapse reported significantly more internal, stable, and global attributions (higher AVEs) and reported more guilt compared to those who slipped but did not relapse back to regular smoking. Even when other factors potentially associated with relapse (e.g., change in perceived control, feelings of guilt) were taken into consideration, the AVE still emerged as the strongest predictor of relapse. Attributions in response to hypothetical lapses prior to quitting did not predict relapse. Findings provide support for implementing clinical interventions to help individuals who lapse avoid making self-blaming internal, stable, and global causal attributions which increased risk of full relapse.

An important distinction to make when translating this conceptual model into clinical interventions is that of characterological versus behavioural self-blame (Janoff-Bulman, 1979). While both of these types of self-blame represent an internal locus of causation, they have distinct implications and sequelae. It is generally more adaptive to

encourage individuals to attribute personal responsibility to controllable behavioural

factors (such as a lack of coping skills that can be learned and practiced) rather than to

uncontrollable characterological deficits (such as a weak will). Characterological self-

blame (Janoff-Bulman, 1979) brings about helplessness and dysphoric mood.

Behavioural self-blame, on the other hand, promotes recovery from setbacks (Anderson,

1983; Janoff-Bulman, 1979; Tennen, Affleck, & Gershman, 1986; Timko & Janoff-

Bulman, 1985). Therefore, the important dimensions to address appear to be the stability

and controllability of causes, rather than locus.

A more recent study of the abstinence violation effect was conducted by Shiffman

and colleagues using ecological momentary assessment to capture participant self-reports

within minutes of a lapse or significant temptation episode, thus eliminating retrospective

bias (Shiffman et al. 1997). Results indicated that participants who attributed their lapses

to more controllable causes experienced more guilt but also felt more encouraged

following their lapses. Internality and stability of attributions were not associated with

mood or efficacy. Participants in this study had sought treatment for smoking cessation

and were selected for high initial motivation and efficacy, possibly limiting the range of

relevant variables and generalizability of the findings.

A study by Eiser and colleagues (Eiser, van der Pligt, Raw, & Sutton, 1985; Eiser

& van der Pligt, 1986) found that smokers who made more stable attributions for *other*

smokers' failed attempts to quit perceived themselves as more addicted to smoking, were

less confident in their ability to successfully quit, and had weaker intentions to stop

smoking in the near future. More internal attributions for other smokers' failures to quit

was associated with lower perceived addiction, but did not predict confidence in ability to

quit or strength of intentions to quit. At 12-month follow-up, smokers were asked to report attributions for their own personal failure to quit smoking. Significant but small correlations were discovered between attributions for one's own failed quit attempts and those of other smokers ($r = .26 - .36$), with attributions for the self tending to have greater stability and internality. Path analysis supported a model whereby greater perceived stability of the cause of other smokers' failed quit attempts was negatively related to personal confidence about ability to give up smoking. Confidence, in turn, was associated with stronger intentions to quit, and intention predicted actual abstinence attempts made during follow-up. Internality of attributions did not predict confidence in ability to quit (Eiser et al., 1985; Eiser & van der Pligt, 1986). The attributional dimension of controllability was not assessed at either time point.

Cessation maintenance. Mullen, Pollak, and Kok (1999) examined attributions for success at maintaining abstinence from smoking among a population of women who had quit smoking during pregnancy and enrolled in a cessation maintenance intervention trial. Approximately 63% of women who quit smoking during pregnancy return to smoking within 6 months after giving birth (e.g., Mullen, Richardson, Quinn, & Ershoff, 1997). At 28 weeks gestation the women were asked to report the most important reason for their success in abstaining from smoking while pregnant and to rate this reason on stability, internality, and controllability. Increased stability of cited reasons for cessation during pregnancy was associated with significantly greater odds of remaining abstinent the first year after giving birth. Internality of causes for abstinence during pregnancy was associated with increased likelihood of having remained abstinent at 6 weeks postpartum, and controllability did not predict postpartum smoking status. A similar pattern emerged

again at postpartum; among those women who maintained abstinence through 6 weeks postpartum, stability of attributions for continued success enhanced the odds of remaining abstinent at 3 and 12 months postpartum, internality of attributions predicted abstinence at 3 months only, and controllability ratings did not predict future smoking (Mullen et al., 1999). Further analysis suggested that the effect of attributional stability on subsequent smoking was fully mediated by self-efficacy for not smoking across a variety of situations. Thus, attribution for successful abstinence to more stable causes appear to increase confidence in one's ability to maintain a behaviour change.

Another smaller study (Schmitz, Rosenfarb, & Payne, 1993) investigated attributions for successful abstinence among 26 smokers who had enrolled in a 6-week smoking cessation intervention. Findings revealed a significant difference in attribution ratings according to smoking status at 3-month follow-up; non-smokers were more likely to have attributed their earlier coping success (during treatment and earlier follow-ups) to more controllable factors. While internality and stability scores were also consistently higher over time for those not smoking at three months, group differences did not reach significance (perhaps owing to the small sample size). Pretreatment ratings for coping success in hypothetical situations did not predict outcome. This is consistent with Curry et al.'s (1987) findings, which failed to support the notion of an attributional style predisposing one to stronger AVEs in actual lapse episodes.

Harackiewicz, Sansone, Blair, Epstein, and Manderlink (l987) examined how different treatment components and method of treatment presentation can influence attributions for treatment outcome, and the subsequent impact of these attributions on initial behaviour change and its maintenance over a longer term period. Harackiewicz and

colleagues manipulated the externality of treatment by comparing self-help manuals with and without a drug component (nicotine gum) and by comparing an internal versus external motivational orientation of the program. Rather than use a single bipolar internal-external scale, internal and external attributions were assessed separately, as the authors argued that attributions might be purely external (e.g., "The drug stopped my smoking"), purely internal (e.g., "I worked hard"), or both internal and external attributions may be present at the same time (e.g., "I was able to use the drug effectively"). Two types of attributions were assessed: (a) attributions for the decision to quit, assessed prior to beginning treatment, and (b) attributions for success or failure following treatment, assessed at follow-up 6 weeks after baseline.

All patients were advised to stop smoking by following the guidelines in the manual they received. Each patient was randomly assigned to one of four experimental conditions: (a) intrinsic gum: nicotine gum and a self-help manual with an intrinsic motivational orientation; (b) extrinsic gum: nicotine gum and a self-help manual with an extrinsic motivational orientation; (c) intrinsic self-help: self-help manual only, with an intrinsic motivational orientation; and (d) control: a brief booklet with minimal tips for stopping smoking. The three motivational treatment programs were identical in content but differed in emphasis. Intrinsic manuals focused on the role of the individual's own efforts in smoking cessation, while the extrinsic manual highlighted the doctor's prescribed program.

Patients who received nicotine gum were more successful than the intrinsic self-help and the control groups in initial cessation but were less successful than the intrinsic self-help condition in maintaining abstinence. Patients in the intrinsic self-help group

remained abstinent longer *(M* = 29.8 weeks) than patients who had quit in either gum

condition (intrinsic gum, *M* = 15.9 weeks, or extrinsic gum, *M* = 16.9 weeks). As

hypothesized, treatment condition appeared to have an effect on type of attributions

made, particularly for success at quitting smoking. Successful quitters in the extrinsic

gum condition were more likely to attribute their success to external factors than quitters

in either intrinsic condition. Patients who were unsuccessful also made more internal

attributions for their failure to quit in the two intrinsic conditions than in the extrinsic

gum condition.

Attributions made also interacted with type of treatment in influencing treatment

outcomes. In the extrinsic gum condition, those who made more external attributions for

their initial decision to quit were more likely to quit compared to those who made fewer

external attributions. However, individuals in the intrinsic conditions who made more

external attributions for their decision to quit were less likely to quit. Furthermore, those

who quit and made more external attributions for their initial success were more likely to

relapse in the intrinsic self-help condition but were more likely to remain abstinent in the

gum conditions (Harackiewicz et al., l987). Further research is necessary to identify

mechanisms responsible for the interaction between treatment condition and type of

causal attributions. Findings suggest that treatment benefits may be maximized when

there is a match between the attributional orientation of the participant and of the

program. The results also indicate that external attributions may not always have

deleterious effects. Rather, their effects may depend on the treatment context,

highlighting the complexity of attributional processes in behaviour change and

maintenance (Harackiewicz et al., l987).

Measurement of Causal Attributions

At least three methodological issues to consider when studying causal attributions have been identified in the literature. First, how will causal attributions be elicited? Two primary methods of eliciting causal attributions can be found in published research. The first method uses cued attribution lists, for which participants are asked to endorse causes from a list devised by the researcher, usually on the basis of theory or past empirical evidence using open-ended methods. Cued attribution lists may have one of two response formats: dichotomous "yes" or "no" responses or rating scales. Some studies also ask participants to identify which attributions they regard as the most important causal factors. Based on responses to individual causal items on such lists, researchers may also use exploratory factor analysis (EFA) to derive scales of causal beliefs, yielding a smaller number of more general causal factors. Disadvantages of using such a list are that a researcher may omit potentially important causal explanations held by participants (Wearden et al., 2006), and increased demand effects may yield agreement with greater number of items than other methods.

The second most commonly utilized method is to ask participants to generate attributions freely by asking open-ended questions such as "What do you think is causing you to smoke?" The open-ended responses are then coded by judges into categories determined by the researcher. Open-ended questions, although harder to analyse and quantify, avoid the problem of overlooking potentially important causes and do not lead to demand effects (Turnquist, Harvey, & Andersen, 1988). Furthermore, it has been argued that the beliefs elicited using open-ended techniques are more accessible (Aday & Cornelius, 2006), and therefore presumably more important in determining behaviour.

However, it has also been argued that this method may prompt a causal search that might not otherwise have taken place (Wearden et al., 2006). To avoid this potential issue, an alternative open-ended approach is to document causal attributions that arise spontaneously while patients talk generally about their illness. While attributions elicited this way may prove to be more clinically relevant, they are substantially more difficult to measure.

Research has been conducted to attempt to determine whether different methods of assessing attributions yield comparable results. Gudmundsdóttir, Johnston, Johnston, and Foulkes (2001) compared different methods of obtaining attributions (cued, elicited, and spontaneous) from 100 myocardial infarction patients and found that the types of attributions made were very similar across all assessment methods and the most commonly mentioned causes were the same. However, a greater number of causes were endorsed when cued attribution lists were used than when they were asked openly, either spontaneous or elicited (Gudmundsdóttir et al., 2001).

A systematic review conducted by Hall, French, and Marteau (2003) examining attributions following a serious event found that attributions were less likely to be associated with negative outcomes if they were elicited using open-ended questions than if other methods were used, such as rating causes on a list provided by researchers. The authors offered three plausible explanations for this finding. First, responses to open-ended questions may be less reliable because responses are coded by judges. Second, open-ended responses are typically coded according to the presence or absence of a cause, resulting in less variance and lower statistical power compared to questionnaire rating measures, attenuating correlations. Third, people list fewer attributions in response

to open-ended questions (Gudmundsdóttir et al., 2001), and are perhaps not as likely to report attributions that are less socially acceptable.

French, Marteau, Senior, and Weinman (2005) compared two of the more commonly used methods of eliciting beliefs about the causes of myocardial infarction: respondents both generated their own list of causes for myocardial infarction and also rated a list of causes provided to them using a structured questionnaire. No differences were found in either the frequency or rated importance of attributions according to whether they were generated by either the respondent or the experimenter. However, there was evidence that different patterns of attributions were produced based on whether respondents rated attributions dichotomously (i.e., yes/no) or on a scale. This finding is consistent with a previous systematic review of 54 studies of causal beliefs for coronary heart disease that concluded the type of attributions made was not affected by whether the causes were generated by respondents or provided by experimenters but did find that attributions to stressors and fate or luck were more likely to be reported in studies that used rating scales than in studies that used dichotomous ratings (French, Senior, Weinman, & Marteau, 2001).

Two potential explanations have been offered for why different methods may elicit different attributions (French et al., 2001). First, open-ended methods may elicit beliefs that are cognitively accessible or come to mind easily (Aday & Cornelius, 2006), whereas structured methods may also elicit beliefs that are accessible with sufficient prompting; that is, closed-ended items may increase the cognitively accessibility of relevant beliefs or constructs (Higgins & King, 1981). Additionally, the difference could be related to response sets: while interviews are more likely to elicit socially desirable

responses compared to self-administered questionnaires (Bowling, 2005), closed-ended questions may be more susceptible to response acquiescence or "yea-saying" (Aday & Cornelius, 2006).

Based on research comparing different methods of assessing attributions, French et al. (2005) offer several tentative recommendations concerning the measurement of causal attributions for illness. First, questionnaire measures should be preferred over simple open-ended measures such as a listing task. Open-ended assessments of causal beliefs have less variance than questionnaire ratings and this lack of variance diminishes the predictive ability of these measures. Sufficient preliminary exploratory work using open-ended measures can help ensure that structured questionnaires capture important beliefs. Where there is relatively little existing research on causal beliefs, there is a stronger case for using open-ended techniques as a preliminary step.

Multi-trait multi-method analyses conducted by French et al. (2005) also raised concern that questionnaire items of causal beliefs contain substantial common method variance (error variance attributable to measurement method, shared by variables measured with the same method), resulting in over-estimated loadings onto causal belief factors. In the absence of alternative measurement techniques to address this psychometric issue, the authors argue for avoiding principal components analysis, which exacerbates this issue of inflated estimates of component loadings (Fabrigar, Wegener, MacCallum, & Strahan, 1999). Instead, they recommend that other methods of factor analysis, such as common or principal axis factor analysis, be used in place of principal components analysis.

A second general methodological issue concerns the classification of attributions. Causal attributions about illness are usually classified either by content into attribution types or categories such as 'stress' or 'chance' or, alternatively, according to their position along a number of underlying dimensions such as locus, controllability, and stability. The dimensional method allows comparisons across different types of events that do not share causes. In a review of the literature in health psychology, Roesch and Weiner (2001) found an overwhelming majority of studies used attribution categories rather than attribution dimensions. Relatively little has been written about the relative merits of these two approaches to assessing attributions and as of yet the issue has not been adequately explored (Hall et al., 2003). While locus and controllability have received the most attention to date, "causes must be classified according to all dimensions to achieve maximum predictive power. Because each dimension has a unique behavioral or affective consequence, limiting analysis to one dimension is simply a mistake" (Amirkham, 1998, p. 1007).

Participants may be asked to rate illness causes along those dimensions, or investigators may code open-ended illness attributions along them. Assuming that the researcher can accurately translate the meaning of the subject's causal attributions into causal dimensions is what Russell (1982) refers to as the "fundamental attribution researcher error" because of the risk that the researcher may perceive the cause quite differently than the attributor. Weiner (1985) noted that the placement of a causal attribution in terms of causal dimensions is subjective and can vary substantially between individuals and across different situations. Evidence exists that people do not generally agree on where specific causes should be located on Weiner's dimensions (Krantz &

Rude, 1984). For example, one individual may perceive luck as more internal ("I've always been a lucky person") than another ("It was random, luck of the draw"). Therefore, it has been recommended that researchers directly assess how the attributor perceives the causal attribution he or she has made for an outcome, such as by assessing underlying causal dimensions using the Causal Dimension Scale (Russell, 1982; Russell, McAuley, & Tarico, 1987).

Attributional models such as Weiner's theory (1985) often imply that causes are important only by way of their underlying dimensional properties, which has led some to ignore attribution type in favour of an analysis of causal dimensions. However, Krantz and Rude (1984) provided evidence in support of assessing *both* attribution type and dimensional ratings, suggesting each may represent a distinct underlying process. Ninety-six undergraduate students were asked to imagine that they had received a low exam grade and on this basis to answer three different types of attribution questions using 6-point Likert-type scales. Analyses were performed to determine how well these different types of ratings predicted depression scores. The cause-rating method entailed rating the influence of four different causes: effort, ability, task difficulty, and bad luck. The dimension-rating method asked participants to rate causality on each of three dimensions: stability, locus, and globality. Two variations of the dimension-rating method were used, one asking for ratings on the major cause (previously rated as having the greatest influence) or for causes in general. Findings revealed that participants' dimension ratings did not always agree with theoretical predictions for dimensions underlying the four causes listed. For example, concordance between theoretical prediction and actual ratings on the stability and globality of the four causes ranged from approximately 48% to 63%.

Most importantly, causes and dimensions each made a unique contribution to the prediction of depression scores. Possible explanations for the unexpected utility of ratings for specific causes were offered by the authors. First, each cause may have unique properties that cannot be captured by general rules or dimensions. Secondly, it may indicate additional underlying dimensions have yet to be identified. Yet another possible explanation is that participants' introspections about dimensions underlying causal beliefs are incomplete or implicit compared to the causes themselves. The two dimension-rating methods were moderately correlated and each made a unique contribution to the prediction of depression scores.

The third methodological issue to consider when assessing attributions of cause is the precise choice of event. This may require making a distinction between attributions for illness onset and for ongoing illness events. For example, while the onset of diabetes may be attributed to heredity factors, current blood glucose control may be attributed to behavioural factors such as diet and physical activity. Causal attributions for the onset of diabetes may be of limited relevance to day-to-day management behaviour in patients with long-standing diabetes (Wearden et al., 2006). Similarly, Amirkham (1998) found that attributions for outcomes of already attempted coping responses were overall superior predictors of coping responses and distress compared to attributions for the stressful event itself. A study asked AIDS and AIDS-related complex patients to provide attributions for their illness vs. attributions to factors that may contribute to possible improvement. Results demonstrated that the degree to which cause of illness was attributed to oneself was significantly correlated with dysphoria, whereas degree to which improvement was attributed to oneself was significantly negatively correlated with

dysphoria and positively related to health behaviour change (Moulton, Sweet, Temoshok, & Mandel, 1987). French, Maissi, and Marteau (2005) also discovered that sometimes researchers and participants may interpret the word "cause" differently (such as for chronic longstanding causes for a myocardial infarction versus acute causes). Therefore, clarity on the researcher's part is required when designing questions and methods to assess attributions.

Overall, it is evident that relationships between attributions and outcomes are not consistently found, owing somewhat to variation in study methodology, such as the various ways in which methodology choices can impact attributions elicited as outlined earlier. Therefore, it is important that findings are replicated with different methodologies in order to have greater confidence in their accuracy (French, Marteau, Senior, & Weinman, 2002).

Rationale for the Current Study

In summary, a few primary concerns have been raised regarding the emergent applications of genetic testing in healthcare, in particular with respect to its potential impact on patient health behaviour: (1) does genetic feedback lead to decreased perceived control or fatalism over health threats; (2) will genetic feedback impact health behaviour performance and motivation to change health behaviour; and (3) does genetic feedback prompt changes in the perceived effectiveness and uptake of different types of interventions (e.g., behavioural vs. biomedical)? In light of the small number of studies conducted to date, equivocal findings, and significant heterogeneity in study design and populations, the empirical evidence is largely inconclusive and further research is needed to resolve these issues.

One variable that has received little attention in this line of research is that of causal attributions. Yet, a possible mediator or moderator of genetic testing outcomes may be changes in causal explanations for the condition involved. To the extent that genetic testing prompts a meaningful change in causal beliefs underlying the health condition involved (e.g., Senior & Marteau, 2007), theory predicts further changes in motivation, affect and behaviour. In order to further investigate the relationship between causal attributions and variables relevant to the three questions posed above, the current study will examine causal attributions in the context of smoking, a well-established and widely acknowledged health threat. In particular, the purpose of the current study is to examine whether causal attributions influence smokers' intentions to quit smoking and to seek smoking cessation treatment, with an emphasis on perceived control and perceived effectiveness of treatment as mediating variables. To this end, the current study will test a causal model incorporating these variables among a general population of smokers. The outcome variables of interest in the current study are intentions to quit smoking and intentions to seek treatment during a future quit attempt. Behavioural intentions are used in the current study as a "proof-of-principle" outcome, prior to investing greater resources in tracking behavioural outcomes in smokers over time (Campbell et al., 2000; Vogt et al., 2008). Intentions are considered the most proximal predictors of behaviour (Norman, Conner, & Bell, 1999), and a meta-analysis of experimental evidence has established that a change in intention is associated with a change in behaviour (Webb & Sheeran, 2006).

The current literature review did not identify any published research that had

conducted a comprehensive examination of smokers' causal attributions for current smoking, as the literature to date has focused on attributions made among those who have made a quit attempt (i.e., attributions for slip or relapse, or successful maintenance of a quit attempt). The single study located that did assess smokers' causal attributions for current smoking was conducted by Wright et al. (2007), however, the assessment of causal attributions in this study focused entirely on genetic causes. The current study aims to extend this line of investigation by incorporating a more comprehensive assessment of causal attributions, factors associated with decisions to seek treatment, and differentiation between personal and treatment control beliefs.

Hypotheses

The overall research question this study sought to examine was: Do causal attributions for current smoking among daily smokers influence their intentions to quit and to seek treatment for smoking cessation? To examine this research question, two models were developed, representing a series of hypotheses regarding how causal types and causal dimensions might influence intentions to quit smoking and to seek smoking cessation treatment via their effects on a set of mediating variables.

Causal attributions were assessed using two different approaches. Participants were asked to list in an open-ended format the causes of their smoking and to then rate these causes (as a group) along scales based on Weiner's (1985) proposed dimensions of locus of causality, controllability and stability. The controllability dimension was divided into two scales according to locus of causality, with separate scales assessing control with an internal locus (internal control) and control with an external locus (external control). Afterward, participants were presented with a list of potential causes and asked to provide

ratings on the extent to which this was a causal influence on their smoking. The ratings were then submitted for exploratory factor analysis (EFA) to extract a smaller number of causal types or categories. Accordingly, two models were tested to separately assess the effect of causal type and causal dimensions on the dependent variables.

Hypotheses for causal type model. Based on an examination of the items, it was conjectured that at least three general factors would be extracted during exploratory factor analysis: (i) genetic/biological – it was believed that genetic factors would load onto the same factor as other physiologically based items; (ii) psychological – items relating to emotions and stress were expected to load onto one factor; and (iii) social – items that reflect the influence of other people on one's smoking were expected to form a distinct factor. Based on this assumption, a preliminary causal model was developed (see Figure 2) and the following hypotheses were made.

Hypothesis 1: Causal types will be associated with perceived behavioural control. While genetic and biological causes have an internal locus of causality, they are unlikely to be perceived as being under direct control by the individual (personal control). This is consistent with the findings of Wright et al. (2007), who found that smokers who perceived genes as a cause of their smoking reported significantly lower perceived control over smoking compared to smokers who did not. Thus, it was hypothesized in the current study that biological/genetic causal attributions will be negatively associated with perceived behavioural control.

With some exceptions, such as genetics, causal attributions with an internal locus are generally associated with greater perceived controllability (e.g., McAuley et al., 1992). Given that the psychological causal factor hypothesized here is expected to
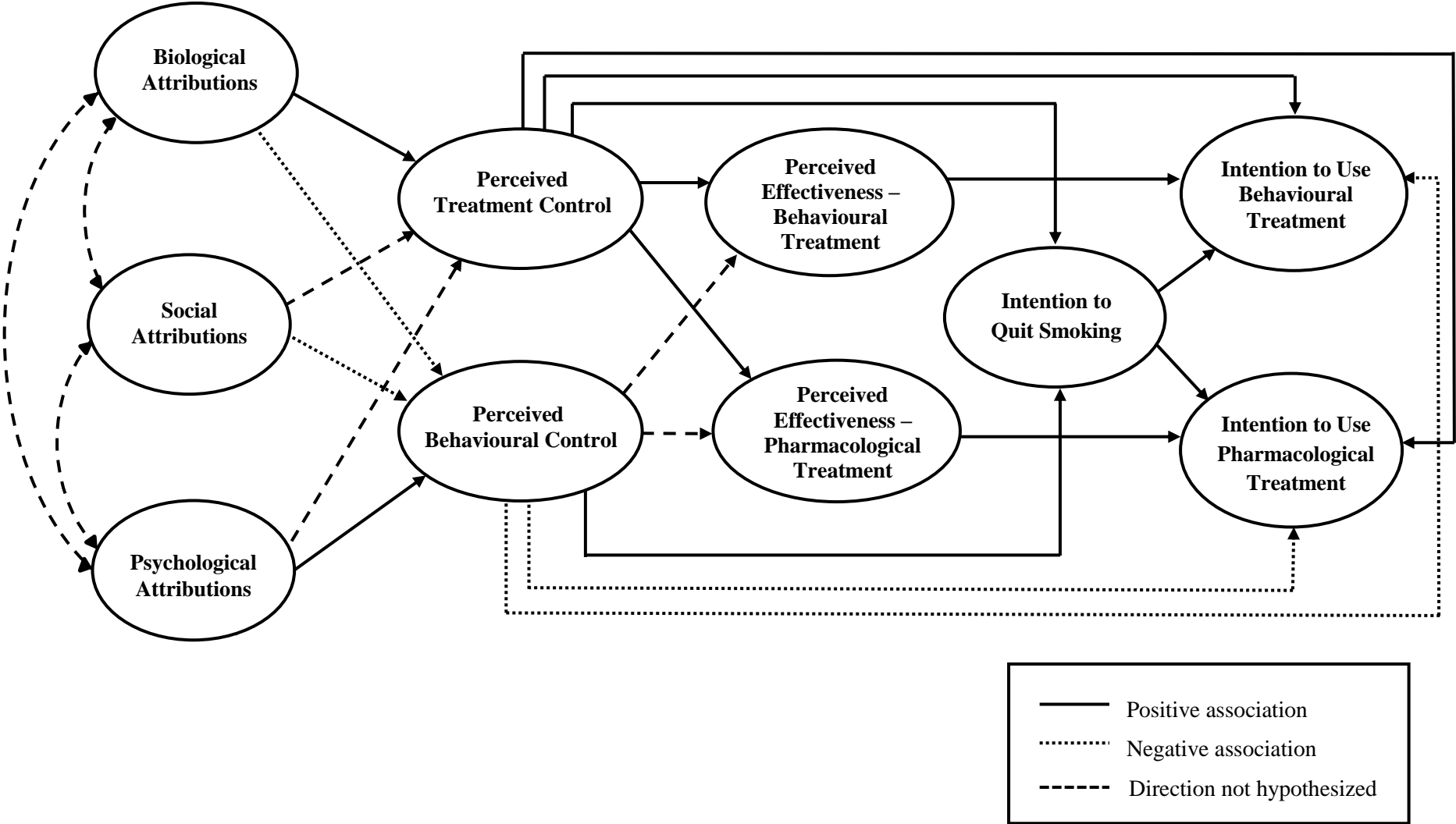
Figure 2. Preliminary hypothesized causal type model.

represent causes that are largely considered to have an internal locus (e.g., emotions), it is hypothesized that psychological causal attributions will be positively associated with perceived behavioural control. Since attributions to social causes have an external locus, it is hypothesized that they will be associated with decreased perceived behavioural control.

Hypothesis 2: Causal type will be associated with perceived treatment control. In light of the fact that few studies have tested for associations between causal attributions and treatment control, and that causal attributions vary between studies and illnesses and do not correspond well to the causal attribution categories hypothesized here, there was not sufficient prior evidence upon which to base hypotheses about associations between causal attributions and treatment control in the current study. However, based on the hypothesis made by Marteau et al. (2004) that beliefs about how control is most effectively achieved shift away from behavioural strategies after genetic risk feedback, a positive association between biological causal attributions and treatment control is incorporated into the model. For someone who believes that their physiology is driving their smoking, perhaps the only foreseeable way to change or manipulate this cause is indirectly through the use of pharmacological intervention, which is ascribed control over the behaviour. Paths between treatment control and both psychological and social causal attributions will be tested in an exploratory manner; therefore no directional hypotheses are made.

Hypothesis 3: Perceived treatment control will be positively associated with perceived effectiveness and intention to use pharmacological and psychosocial treatment. A stronger belief that treatment can control smoking is hypothesized to be related to

greater intentions to use treatment. While a link between treatment control and perceived

effectiveness of treatment has not been published, Bradley et al. (1987) found that levels

of both perceived personal control and perceived medical control predicted the type of

treatment selected by individuals with type 1 diabetes. Specifically, greater perceived

medical control and lower perceived personal control predicted the choice of insulin

pump over more conventional injection regimens. The authors suggest the insulin pump

may (falsely) appear to place fewer behavioural demands on the patient. In fact,

individuals with higher levels of perceived medical control exhibited poorer glycemic

control using the insulin pump, as they likely overestimated the ability of the insulin

pump to regulate blood sugar and underestimated their role in treatment outcome.

Additional research has also found a link between treatment control and seeking mental

health services (Vanheusden et al., 2009), and adherence to medication (Bucks et al.,

2009).

In the current study, it is suggested that perceived treatment control will be

positively associated with both types of treatment because the ability of treatment in

general to have some control over smoking would appear to be a necessary (but not

sufficient) prior condition for the belief in the effectiveness of a particular treatment.

Consistent with the hypothesis of Marteau et al. (2004) and the findings of Bradley et al.

(1987), it is expected that the association of perceived treatment control with perceived

effectiveness and intentions to use treatment will be stronger for pharmacological

treatment versus psychosocial treatment, given the greater behavioural demands of

counselling and self-help treatments versus pharmacological treatments.

Hypothesis 4: Perceived behavioural control will be negatively associated with

perceived effectiveness and intention to use pharmacological and psychosocial treatment.
Vogt et al. (2008, 2010) found that smokers high in self-efficacy to stop smoking had

lower intentions to use NRT, bupropion, and group counselling; therefore it is

hypothesized that perceived behavioural control will be negatively associated with

intention to use both pharmacological and psychosocial treatments. Whether this

association is mediated via beliefs about perceived effectiveness will be tested in an

exploratory manner.

　　　Hypothesis 5: Perceived behavioural control and perceived treatment control will

both be positively associated with intention to quit smoking. Perceived behavioural

control is theorized and demonstrated to be associated with intentions to perform the

same behaviour (Ajzen, 1985, 1991; Armitage & Conner, 2001). In the current study,

higher levels of perceived control over continued smoking, whether at a personal level or

achieved via treatment, are anticipated to prompt intentions to quit, while perceived lack

of control is believed to discourage plans to quit.

　　　Hypothesis 6: Perceived effectiveness of pharmacological treatment will be

positively associated with intentions to use pharmacological treatment and perceived

effectiveness of psychosocial treatment will be positively associated with intentions to use

psychosocial treatment. Previous research has established that perceptions of perceived

effectiveness of both pharmacological and psychosocial smoking cessation treatments

predict intentions to use these same types of treatment (Vogt et al., 2008, 2010); it is

anticipated that these findings will be replicated in the current study.

　　　Hypothesis 7: Intention to quit smoking will predict intention to use

pharmacological and psychosocial treatments. Given that intention to use treatment

presumes an intention to make a quit attempt, it is hypothesized that intention to quit

smoking is positively associated with intentions to use both forms of treatment. Past

research has found an association between intention to stop smoking and intentions to use

both behavioural support and cessation medication (Vogt et al., 2008, 2010).

Hypothesis 8: Causal attribution factors will correlate. As exogenous factors, the

causal attribution factors in each model will be permitted to covary. No directional

hypotheses are made about the direction or significance of these correlations.

Hypotheses for causal dimension model. Based on a lack of research assessing

both causal dimensions and treatment beliefs or threat/illness representations, the

following hypotheses for causal dimensions are largely exploratory. Hypotheses 3-7

above are presumed for the causal dimension model as well. See Figure 3 for a diagram

of the model to be tested.

Hypothesis 9: Controllability will be associated with perceived behavioural

control and perceived treatment control. It is hypothesized that perceived behavioural

control will be positively associated with internal control and negatively associated with

external control. Conversely, perceived treatment control is hypothesized to be negatively

associated with internal control and positively associated with external control.

Hypothesis 10: Stability will be negatively associated with both perceived

behavioural control and perceived treatment control. A negative outcome attributed to a

cause that is perceived as stable is theorized to be associated with poorer expectations for

success in the future and greater hopelessness (Weiner, 1985). Thus, it is expected that

greater perceived stability of the causes of one's smoking will be negatively associated

with perceptions of both personal control and treatment control.
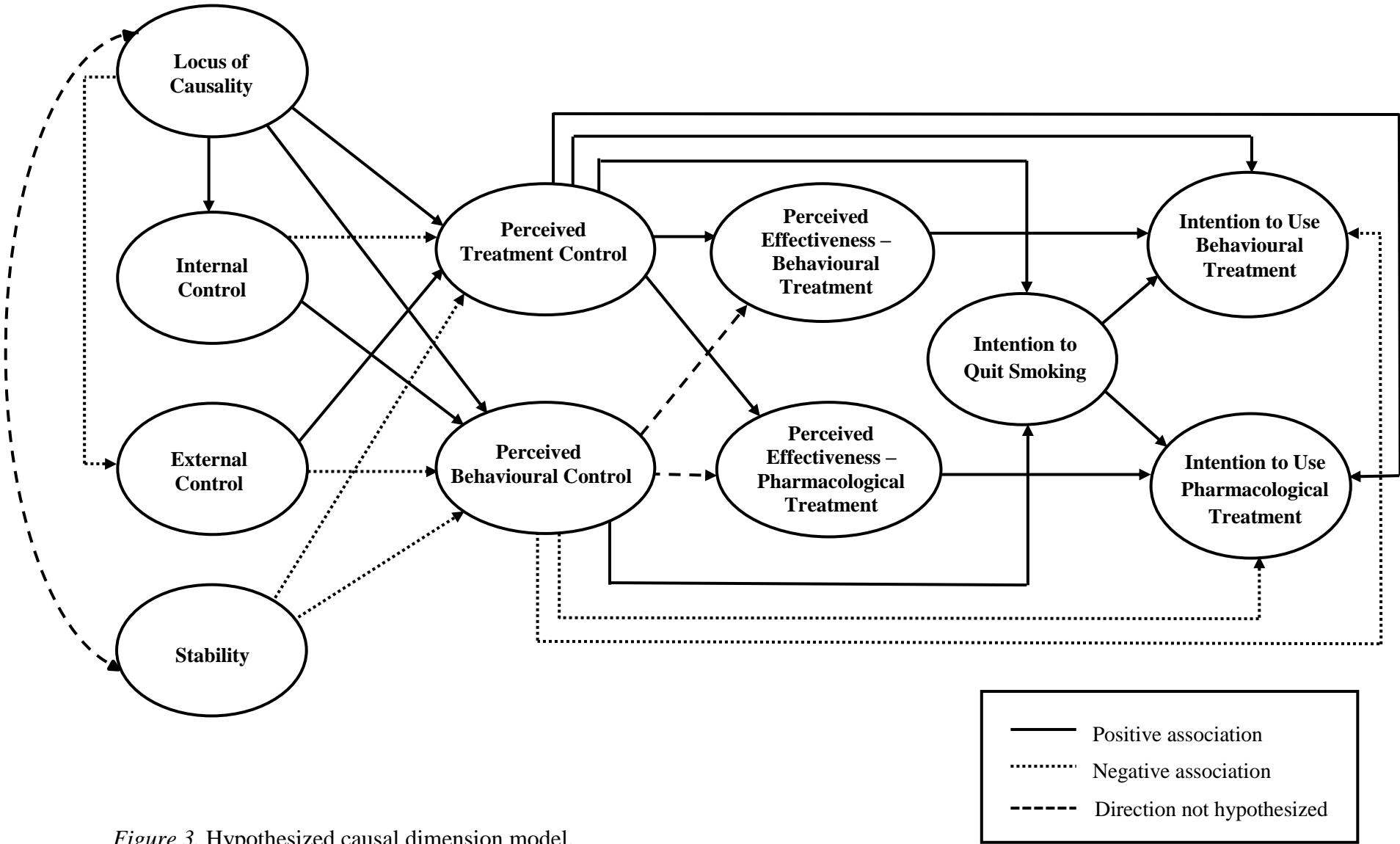
Figure 3. Hypothesized causal dimension model.

Hypothesis 11: Locus of causality (internal) will be positively associated with internal control and negatively associated with external control, and will exert both a direct and indirect effect (via internal and external control) on perceived behavioural control and perceived treatment control. It is hypothesized that the relationship between locus of causality and perceived behavioural control and perceived treatment control will be partially or fully mediated by internal and external control. The Locus of Causality scale is structured such that higher scores reflect an internal locus of causality; scores on this scale have been found to positively correlate with internal control and negatively correlate with external control (McAuley et al., 1992) and the same associations are hypothesized here. With respect to perceived control, it is hypothesized that indirect effects of locus of causality on perceived treatment control and perceived behavioural control, via internal and external control, will be found (see Figure 3). Additionally, a direct positive effect on perceived treatment control is hypothesized, as treatment is primarily aimed at the individual and internal factors, rather than external factors. A direct positive effect on perceived behavioural control is also hypothesized, as the construct assumes an internal locus.

CHAPTER III

METHOD

Participants

Participants were part of the Ontario Health Study (OHS), a longitudinal study of a large cohort of Ontarians designed to track and analyze the development of cancer and other disease, through ongoing collection of data on health status and various risk factors. All participants completed the OHS online baseline questionnaire between 2010 and 2012, and had consented to be contacted in the future about ancillary research studies approved by the OHS.

Inclusion criteria for the current study required that all participants were (1) aged 18 years or older and (2) current daily smokers (at least one cigarette per day). Additional criteria were also used to select a subsample of 5000 participants to be sent an invitation to participate in the current study. The Ontario Health Study has a policy to contact participants no more than once every three months for participation in research. In light of the recruitment of a large sample of participants in the Greater Toronto Area (GTA) to visit the OHS Assessment Centre close to the period of recruitment for the current study, all participants who reported living in the GTA (postal code beginning with M) were excluded. For similar reasons, participants aged 25-34 were oversampled by a small amount (see paragraph below). Non-English speaking OHS participants were also excluded. Only participants who had self-reported smoking daily (at least one cigarette per day for the past 30 days) in the OHS baseline questionnaire were contacted in order to maximize the probability of reaching current daily smokers.

A stratified sampling procedure was used with proportional allocation to

approximate the age distribution of current smokers in the OHS sample, with some

adjustment to oversample participants 18-39 by 10%. An equal number of men and

women were selected within each stratum (18-29, 30-39, 40-49, 50-59, 60-69, 70-79,

80+), with the exception of the 18-29 and 30-39 strata, which were short 11 and 19 men,

respectively, due to over-representation of women in the original OHS sample. Thus, a

total of 4,970 individuals received an e-mail invitation from the OHS to participate in the

current study.

Of those contacted, 560 followed the link to begin the survey. Given that smoking

status may have changed since completion of the OHS baseline questionnaire, all

participants were first asked about current smoking status (see Appendix B) to determine

eligibility. A total of 459 participants indicated that they were currently smoking daily

and were eligible for the current study; 12 occasional smokers, 42 non-smokers, and 47

who did not provide a response were not eligible to participate in the current study. A

further 41 participants were excluded due to excessive missing data (more than 25% of

all data points were missing), leaving a final sample of 418 participants.

Sample demographics and smoking characteristics are presented in Table 1. The

mean age was approximately 50 years (range = 19 to 69 years), and participants on

average had started to smoke daily 33 years prior. The average number of cigarettes

smoked per day was 18 (range = 3-65). Approximately a quarter of the sample were light

smokers ($\leq$ 10 cigarettes/day), and a similar number were considered heavy smokers ($\geq$

20 cigarettes/day). The majority of the sample had a moderate level of nicotine

dependence and had made at least one previous quit attempt. The sample was

Table 1

Sample Demographic and Smoking Characteristics

Variable	*N*	%
Demographic Characteristics		
Age (years), *M* (*SD*)	49.9	11.3
Gender		
Male	181	43.3
Female	237	56.7
Ethnicity		
White/European	379	90.9
South Asian	7	1.7
Aboriginal	4	1.0
Arab	4	1.0
Black/African/Caribbean	3	0.7
East Asian	2	0.4
South East Asian	1	0.2
Other	17	4.1
Level of Education		
Elementary school	4	1.0
High school	120	28.8
Trade, technical or vocation school, apprenticeship training or technical CEGEP	41	9.8
Diploma from a community college, pre-university CEGEP or non-university certificate	137	32.9
University certificate (below Bachelor's level)	20	4.8
Bachelor's degree	63	15.1
Graduate degree	32	7.7

Variable	N	%
Employment Status		
Employed full-time	216	52.0
Employed part-time	36	8.7
Unemployed	22	5.3
Retired	68	16.4
Disability	40	9.6
Homemaker	11	2.7
Student	8	1.9
Other	14	3.4
Relationship Status		
Married or living with a partner	251	60.3
Single (never married)	76	18.3
Separated or divorced	75	18.0
Widowed	14	3.4
Smoking Characteristics		
Number of cigarettes/day		
0-10	113	27.2
11-20	185	44.5
20-30	95	22.8
30+	23	5.5
Level of nicotine dependence (HSI)		
Low	81	19.5
Moderate	303	72.8
High	32	7.7
Years since onset of daily smoking, *M* (*SD*)	33.1	12.0
Number of previous quit attempts		
0	35	8.5
1-3	171	41.3
4-9	123	29.7
10+	85	20.5

Variable	*N*	%
Past use of cessation aids		
Nicotine replacement therapy (NRT)	291	69.6
Bupropion (Zyban)	158	37.8
Varenicline (Champix)	129	30.9
One-on-one advice/counselling from a healthcare professional	99	23.7
Group counselling/support groups	47	11.2
Quitline/telephone counselling	47	11.2
Internet-based support	68	16.3
Self-help manuals	130	31.1
Alternative therapies	110	26.3

Note. CEGEP = Collège d'enseignement général et professionnel (College of General and Vocational Education); HSI = Heaviness of Smoking Index. Complete data are presented without estimation or imputation; less than 1% of data were missing on each variable. Sample size varies due to missing data. Unless otherwise noted, frequency and percentage are reported.

predominantly of White/European ethnicity and the majority were currently employed, married or living with a partner, and had completed post-secondary education. Approximately two-thirds of the sample had previously used Nicotine Replacement Therapy, while approximately one-third or less had used each of the other treatments (see Table 1).

Measures

Screening item. Prior to completing the survey, a single screening question ensured that participants were current daily smokers (see Appendix B). Anyone who did not currently smoke daily was informed that they were not eligible for the study (see Appendix C).

Causal dimensions. Weiner's (1985) causal dimensions were assessed using the Revised Causal Dimension Scale (CDSII; McAuley et al., 1992). Participants were first asked to list all the causes of their current smoking (see Appendix D). They were then asked to think about all the causes they listed and rate them as a group on a series of 9-point semantic differential scales (see Appendix D). Each scale corresponded to one of the three causal dimensions described by Weiner (1985): locus of causality, stability, or controllability. The CDSII is a revision of the original Causal Dimension Scale (CDS; Russell, 1982), designed to address concerns raised regarding the validity and reliability of the original Controllability scale (e.g., Russell, McAuley, & Tarico, 1987; Vallerand & Richer, 1988). In place of the Controllability scale, the authors of the CDSII created two new scales – Personal Control and External Control – to separately assess perceived level of internal and external volitional control over a causal event. Because another scale used in the current study is labelled Personal Control, the Personal Control subscale of the

CDSII is herein referred to as the Internal Control scale to avoid confusion. The Stability

and Locus of Causality scales remain unchanged in the CDSII. Confirmatory factor

analysis provided evidence that a four-factor model provided a better fit than a three-

factor model that combined External Control and Internal Control into a single dimension

(McAuley et al., 1992). Each scale is comprised of three items, for a total of 12 items.

McAuley et al. (1992) reported an average Cronbach's alpha value of .79 and .82 across

four samples for the Internal Control and External Control scales, respectively, and .67

for both the Locus of Causality and Stability scales. In the current study, internal

consistency was adequate for Internal Control ($\alpha = .81$) and External Control ($\alpha = .74$),

but was poorer for Locus of Causality ($\alpha = .63$) and Stability ($\alpha = .57$). Overall, the

Cronbach's alpha values were similar or somewhat lower than those reported by

McAuley et al. (1992). Higher scores indicate internal locus of causality and greater

levels of stability and controllability.

   Types of causal attributions. The assessment of causal type utilized in the current

study was adapted from the Causes subscale of the Revised Illness Perception

Questionnaire (IPQ-R; Moss-Morris et al., 2002). The IPQ was designed to assess the

five components of illness representations outlined in Leventhal's Common Sense Model

(Leventhal, et al., 1980). The original IPQ was revised by Moss-Morris and colleagues to

improve its psychometric properties and to extend the scope of the questionnaire by

adding additional items and three new subscales. The IPQ and IPQ-R have been adapted

for use with a wide variety of illness populations (see Hagger & Orbell, 2003 for a

review), as well as for use with healthy populations to examine health-related behaviour

(Figueiras & Alves, 2007). Validation of the IPQ-R was carried out with a sample of

eight different illness groups including individuals with HIV, multiple sclerosis, asthma, type 2 diabetes, and chronic pain.

The item and response format used in the IPQ-R is retained in the current study. The assessment of cause in the IPQ-R presents respondents with a list of 18 possible causes, which can be tailored to the population and condition being studied. Items from the IPQ-R that were deemed relevant to smoking (e.g., "overwork" or "my personality") were retained and some revised, while those that were judged not relevant (e.g., "a germ or virus" and "pollution in the environment") were deleted. Additional causal items were added based on attributions and motives listed for adult smoking in the extant literature, as well as literature examining causal attributions for various health conditions or health in general. In total, a list of 18 possible causes for current smoking was compiled (see Appendix E). The IPQ-R instructions were also adapted for smoking. Instructions ask participants to "Please indicate how strongly you agree or disagree that the following are causes of your current smoking" on a 5-point Likert scale from 1 = "strongly disagree" to 5 = "strongly agree". As per IPQ-R scoring instructions, ratings for causal attributions were analyzed using exploratory factor analysis to identify underlying factors. Following the IPQ-R, participants were also asked to list any additional causes not presented and to name the single most important cause of their smoking (see Appendix E).

Perceived treatment control. Smokers' perceptions of the ability of treatment to control their smoking were assessed using the Treatment Control subscale of the IPQ-R (Moss-Morris et al., 2002). All items were adapted for smoking (see Appendix F for original and adapted items). The scale consists of five items (e.g., "Treatment can control my smoking") rated on a 5-point Likert scale from 1 = "strongly disagree" to

5 = "strongly agree". Moss-Morris et al. (2002) reported good internal consistency of the

scale (α = .80) and acceptable test-retest reliability over a three-week period (.50 − .63).

Internal consistency of the scale was also good in the current study (α = .86)

Perceived behavioural control. Perceived behavioural control over smoking was

assessed using both the Personal Control subscale of the IPQ-R (Moss-Morris et al.,

2002) and additional items assessing perceived self-efficacy. Ajzen (2002) reviews

evidence demonstrating that perceived behavioural control is a higher-order construct that

is comprised of two correlated yet distinct lower-level components: self-efficacy (ease or

difficulty of performing a behaviour) and controllability (beliefs about the extent to

which performing the behaviour is up to the actor). As such, he contends that measures of

perceived behavioural control should contain items that assess both self-efficacy as well

as controllability. Depending on the purpose of the investigation, a decision can be made

to aggregate all items and treat perceived behavioural control as a unitary factor, as in the

current study, or assess them separately.

The IPQ-R Personal Control subscale (see Appendix G) was adapted in the

current study to assess perceived personal control over smoking, instead of illness. The

scale consists of six items (e.g., "I have the power to influence my smoking"), which are

rated using the same 5-point Likert response format as the Treatment Control subscale.

The scale had good internal consistency (α = .81) and acceptable test-retest reliability

over a 3-week period (.46 −.57; Moss-Morris et al., 2002). The Personal Control scale

also had good internal consistency in the current study (α = .77).

Four items were used to assess self-efficacy to stop smoking (see Appendix H).

Two items were based on previous research (Hall, Bishop, & Marteau, 2006; Vogt et al.,

2008, 2010) and were obtained with permission to include in the current study ("How confident are you that you could stop smoking if you wanted to?" and "How easy would it be for you to stop smoking if you wanted to?"). A Cronbach's alpha value of .88 was reported for these two items, indicating high internal consistency (Vogt et al., 2008, 2010). Two additional items were drawn from previous research by Sanderson et al. (2008), that had demonstrated sufficient internal consistency ($\alpha = .73$) together with a third item. Responses for all four items were rated on a 7-point Likert-type scale, with different endpoints (e.g., 1 = "not at all confident" to 7 = "extremely confident"). Internal consistency for these four items was good ($\alpha = .84$), as was internal consistency of the perceived self-efficacy and Personal Control items combined ($\alpha = .83$). Higher scores indicate greater levels of perceived behavioural control.

Perceived treatment effectiveness. Perceived effectiveness of nine different treatments was assessed using two items for each treatment (see Appendix I). The first item ("How effective do you think each of these treatments would be to help you stop smoking?") was adapted from Wright et al. (2012) and is rated on a 7-point Likert-type scale from 1 = "not effective at all" to 7 = "very effective". The second item ("To what extent do you believe the following treatments would increase your chances of quitting smoking?") was adapted from Vogt et al. (2008, 2010) and is rated on a 7-point scale from 1 = "strongly disagree" to 7 = "strongly agree". Two items were utilized for improved reliability over a single item (Spector, 1992). Higher responses indicated higher levels of perceived effectiveness. In the current study, ratings of the perceived effectiveness of treatment were split into two groups for analysis: pharmacological (NRT, bupropion, varenicline) and psychosocial (one-on-one counselling, group counselling,

quitline, Internet-based social support). Internal consistency was excellent for ratings of the perceived effectiveness of psychosocial treatments ($\alpha = .93$) and pharmacological treatments ($\alpha = .86$). Higher scores indicate greater perceived effectiveness of treatment.

Behavioural intentions. Intention to quit smoking was assessed using five items. Four items were adapted from Vogt et al. (2008, 2010) and asked "Do you intend to stop smoking in the next month [6 months]?" and "How likely is it that you will stop smoking in the next month [6 months]?" Each item had two versions to assess intention to quit: (i) within the next month, and (ii) within the next six months. A fifth item based on Stages of Change theory (Prochaska & DiClemente, 1983) asked participants what their plans were with respect to quitting smoking within several time periods listed in an ordinal scale format, from currently making a quit attempt to not planning to quit smoking ever (see Appendix J). The Cronbach's alpha for the five items was .91, indicating excellent internal consistency.

Intentions to seek the same nine treatments for which perceived effectiveness was assessed were measured using similar question stems as for intention to quit smoking (see Appendix J). Vogt et al. (2008, 2010) used these same items to assess treatment intentions and reported Cronbach's alpha values ranging from .83 to .87, indicating good internal consistency. All intention items adapted from Vogt and colleagues were rated on 7-point Likert-type scales. Treatment intentions were also grouped into two types of treatment (pharmacological and psychosocial) for data analysis. Internal consistency was good for intentions to use psychosocial treatments ($\alpha = .93$) and pharmacological treatments ($\alpha = .80$). Higher scores indicate stronger intentions to quit or seek treatment.

Level of nicotine dependence. Level of nicotine dependence was assessed in the

current study using the Heaviness of Smoking Index (HSI; Heatherton, Kozlowski,

Frecker, Rickert, & Robinson, 1989), a shortened version of the Fagerström Test for

Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991).

The FTND is a widely used self-report measure of nicotine dependence consisting of six

forced-choice items regarding smoking behaviour and preferences. A coefficient alpha of

.61 was reported by the FTND scale developers (Heatherton et al., 1991). A recent review

of current methods of assessing tobacco dependence concluded that despite less than

ideal psychometric properties, the "continued use of the FTND is promoted by the

presence of substantial prior research, its ability to predict relapse, and its brevity" (Piper,

McCarthy, & Baker, 2006, pp. 349-350) and pointed out that while new measures of

tobacco dependence have been constructed, further research is necessary to establish their

reliability and validity. The HSI is comprised of two questions from the FTND, assessing

number of cigarettes smoked per day and time to smoke first cigarette upon waking.

These questions were shown to account for most of the predictive value of the FTND and

have been validated as providing similar results (Heatherton et al., 1989; Kozlowski,

Porter, Orleans, Pope, & Heatherton, 1994). The HSI has been found to predict quit

attempts and maintained abstinence (Chabrol, Niezborala, Chastan, & de Leon, 2005),

providing evidence of validity. As the current survey already asks respondents to report

the number of cigarettes smoked per day, this question was simply categorized per the

HSI scoring instructions to derive the overall score. The second item asks participants

how much time elapses before they smoke their first cigarette in the morning (see

Appendix K). The Cronbach's alpha value for both items that comprise the HSI was .65

in the current study, similar to previously reported values in validity and reliability

research (Etter, 2005; Heatherton et al., 1991). Scores were used to categorize level of

nicotine dependence as low (0-2), moderate (3-4) or high (5-6).

Genetics knowledge. Knowledge of the influence of genetics on degree of

nicotine dependence, or addiction to smoking, was assessed using four statements

developed by Quaak and colleagues (Quaak, Smerecnik, van Schooten, de Vries, & van

Schayck, 2012), which are included in the current study with permission (see Appendix

L). The questions were modified to remove the "don't know" response option in order to

encourage participants to respond even if they are unsure of the veracity of their

response. Previous research has found a very high rate, as high as 60%, of selecting

"don't know" to these or similar questions (Quaak et al., 2012).

Smoking behaviour and history. Participants were asked to report what treatment

methods they have used in the past to assist with quit attempts (see Appendix M).

Participants were also asked general background questions about their smoking history

including the age at which they started smoking daily, the number of cigarettes they

smoke per day on average, and past history of quit attempts (see Appendix N).

Sociodemographic information. Participants were asked to provide

sociodemographic information including age, sex, ethnicity, country of residence,

relationship status, level of education, and occupational status (see Appendix O).

Procedure

Following approval from the Ontario Health Study (OHS), and ethical clearance

from the Research Ethics Boards at both the University of Windsor and University of

Toronto, invitations to complete the survey (see Appendix P) were e-mailed to

participants directly from the OHS. Each e-mail included an embedded link to a unique

URL, which permitted identification of participants by the OHS only. Individuals who chose to click on the link were directed to the Letter of Information (see Appendix Q), which provided more detail about the study. After reading the Letter of Information, participants were able to indicate consent by clicking "I agree to participate" at the bottom of the page, which linked to the survey. Participants who clicked "I do not agree to participate" were not able to continue to the survey. Ten participants continued to the survey without providing a response to the consent item, of which nine were eligible for the study. With clearance from the University of Windsor Research Ethics Board, data for these nine participants was included in the current study, as completion of the items was considered consent to participate. The survey was hosted on FluidSurveys.com. The survey data was secured using Secure Socket Layer (SSL) encryption and all data was stored in Canada. The survey was open for three weeks and no incentive for participation was offered.

Before any questions, participants were first presented with survey instructions (see Appendix R), which included directions for how to save responses and return to the survey at a later date or time, as well as how to withdraw from the study and discard all previous responses. The first question screened for eligibility; current daily smokers were able to continue with the survey, while non-respondents and occasional or former smokers were directed to another page to notify them that they were ineligible (see Appendix C).

To protect participant confidentiality, no identifying information was collected at any point. After completing the survey, participants were provided information on free

resources in Ontario for help with quitting smoking (see Appendix S). Participants were

informed that a brief report of the study findings would be available online by June 2013.

The online survey presented measures in the following order: frequency of current

smoking, demographic questions, causal attributions (CDSII and then IPQ-R on a

separate page), perceived effectiveness of smoking cessation treatment, perceived

treatment control, perceived behavioural control, intentions to use smoking cessation

treatment, intention to quit smoking, smoking behaviour and history, and genetic

knowledge questions. Perceived effectiveness and treatment intentions were assessed

using two sets of similarly phrased items (see Appendices I and J); both versions of items

measuring the same construct were interspersed with other items and never presented

adjacently or on the same page to minimize the occurrence of providing identical ratings

due to satisficing (i.e., taking shortcuts to reduce cognitive effort required to respond;

Krosnick, 1991). All questions, except those that were open-ended, were mandatory with

"prefer not to answer" response options added to provide the option of not responding,

while preventing inadvertent missing responses.

Data Analyses

Preliminary data screening and descriptive analyses. Prior to conducting

analyses, data were screened for missing data and to ensure that assumptions of relevant

statistical tests were met. Where any assumptions were violated, appropriate changes or

corrections were made and are noted. Descriptive analyses, including frequencies, means

and standard deviations, and bivariate correlations are presented for study variables.

Qualitative analysis of open-ended responses. Responses to open-ended

questions about the causes of current smoking were analyzed using content analysis in

order to identify the type and frequency of specific causes to which smoking was

attributed. In particular, the inductive content analysis method described by Elo and

Kyngäs (2008) was utilized, as it is recommended when there is not sufficient previous

knowledge about a topic. After an initial open coding, during which notes and codes were

freely generated, a coding scheme was developed combining lower-level codes into a

smaller set of more broad higher-order categories. Thus, the categories were derived

primarily from the data (inductive approach) rather than pre-existing theory (deductive

approach). However, the analysis was not purely inductive as consideration was also

given to ensuring that the specific causes participants were asked to rate in a subsequent

question (IPQ-R) were represented in the coding scheme in order to allow comparison of

the responses to these two different types of question formats. Using this coding scheme,

20% of randomly selected cases ($n = 84$) for each question were coded by a second coder

and inter-rater reliability was assessed using Cohen's kappa statistic (Cohen, 1960), a

widely used measure of agreement between two coders that takes into account agreement

due to chance. A Cohen's kappa of .80 or greater is generally considered a good level of

agreement between coders and indicates that the coding is reliable (Neuendorf, 2002;

Yardley, 2008). An initial 100 cases were coded by the secondary coder for practice and

to identify any issues with the categories or coding scheme; these codes were not retained

and not included in calculation of Cohen's kappa. Any disagreement between coders was

discussed until consensus was reached.

   Factor analysis and structural equation modeling. Exploratory factor analysis

(EFA) was used to identify latent factors from the list of potential causes of smoking

presented to participants. Structural equation modeling (SEM) methods were used to test

the hypothesized models. A two-step modeling approach (Anderson & Gerbing, 1988) was followed, whereby the hypothesized structural model was first respecified as a measurement model and analyzed using confirmatory factor analysis (CFA) to establish the suitability of the factor structure, prior to evaluating hypotheses about causal relations among factors.

The model chi-square goodness-of-fit statistic and corresponding p value were used to test overall model fit. If the p value associated with the chi-square statistic is greater than .05, then the model is accepted as having good fit and the exact-fit hypothesis of no difference between the model-implied population covariances and the observed sample covariances is not rejected (Barrett, 2007). However, because the chi-square statistic is sensitive to sample size and "in large samples virtually any model tends to be rejected as inadequate" (Bentler & Bonett, 1980), model fit indices that are less sensitive to sample size are also reported to assess model fit. Following the recommendations of Boomsma (2000), the following absolute fit indices are reported: (1) the estimated Root Mean Square Error of Approximation (RMSEA; Steiger & Lind, 1980), with 90% confidence interval; and (2) the Standardized Root Mean Square Residual (SRMR; Bentler, 1995). In addition to these two absolute fit indices, an incremental fit index is also reported, the Comparative Fit Index (CFI; Bentler, 1990). RMSEA values equal or less than .05 are considered indicative of close fit, values .05 to .08 indicate reasonable fit, values .08 to .10 indicate mediocre fit, and values greater than .10 indicate poor fit (Browne & Cudeck, 1993; MacCallum, Browne, & Sugawara, 1996). Hu & Bentler (1999) recommend cut-off values close to .08 for SRMR and close to .95 for CFI as indicating good fit (Hu & Bentler, 1999).

Where fit indices indicated a poor fit of the model to the data, post hoc modifications were made on both an empirical and theoretical basis. Any modifications made to the model were made one at a time. Where nested models are compared, chi-square difference test statistics were calculated to test whether the change in overall fit in the model (improvement or decrement) is statistically significant. Where non-hierarchical models are compared, the Bayesian Information Criterion (BIC; Raftery, 1993; Schwarz, 1978) comparative fit index is reported. Lower values of the BIC indicate better fit, and a difference between BIC values greater than 10 is considered strong evidence in favour of one model over another (Kass and Raftery, 1995).

Sample size. Although no firm rule or consensus exists regarding recommended sample size for SEM or CFA, a general rule of thumb is that sample size should be at least 200. Scenarios where a sample size larger than 200 may be necessary include when an estimation method other than maximum likelihood is being used, a complex model is being tested, or variables are severely non-normal (Kline, 2011). As none of these conditions applied to the current study, a minimum sample of 200 was considered sufficient. A somewhat larger sample size of at least 300 is preferable for EFA (Tabachnick & Fidell, 2007). The sample size in the current study exceeds these requirements.

Statistical software. EFA, CFA and SEM were performed using Mplus version 7 (Muthén & Muthén, 2012a). All other analyses were performed using SPSS version 20 (IBM Corp., 2011).

CHAPTER IV

RESULTS

Data Screening

Missing data. The complete dataset for all eligible participants ($N = 459$) was screened to determine the pattern and extent of missing data using SPSS 20.0 Missing Value Analysis, which revealed that 9.2% of all data values were missing, across 73.4% of cases and 96.9% of variables. As expected upon examination of missing data patterns, Little's Missing Completely at Random (MCAR) test was significant, χ^2 (12677) = 13229.53, $p < .001$, confirming that the data was not MCAR. Listwise deletion—utilizing only cases with complete data across all variables—may produce biased parameter estimates when data are not MCAR (Wothke, 2000). Even when data are MCAR, the reduction of sample size due to listwise deletion can lead to substantially larger standard errors and decreased power to detect effects during hypothesis testing. In the current study, a complete case analysis would have reduced the sample size from 459 to 122, with insufficient power for testing the hypothesized model.

Thus, cases with missing data were retained and missing data methods that make use of all available information and do not require MCAR data were selected. The choice of which missing data procedure(s) to use was made foremost on theoretical and empirical grounds after a review of the relevant literature, but was restricted as well by the capability of current software programs. No single missing data method reviewed was appropriate or ideal for all of the different analyses performed in the current study, and thus two different (but very similar) methods were used.

To date, empirical evidence "has unequivocally established the superiority" (Enders & Peugh, 2004, p. 1-2) of two modern approaches to analyzing datasets with missing information: maximum likelihood (ML) estimation and multiple imputation (MI). Compared to conventional methods such as listwise deletion, ML and MI estimates exhibit less bias and greater efficiency (power) under many conditions (e.g., Allison, 2003; Collins, Schafer, & Kam, 2001; Enders & Bandalos, 2001; Wothke, 2000). The theoretical foundations of ML and MI methods are related, and thus when they are implemented in comparable ways they have been shown to produce results that are very similar (e.g., Allison, 2003) or even "virtually indistinguishable" (Collins et al., 2001, p. 331).

The current study utilized both multiple imputation and maximum likelihood methods. Maximum likelihood estimation is most commonly achieved in SEM using full information maximum likelihood (FIML), which was used here. Both methods require that the user input a set of variables (model) that are used to estimate the missing values. MI uses the information on these other variables to impute missing values and output a series of complete datasets that are then used for substantive analysis, with results combined (pooled) across all imputed datasets. MI is relatively flexible in terms of the range of analyses it can be applied to and is available in a wide range of statistical software.

In contrast, FIML does not impute values. Instead, computations analogous to replacing missing data points are performed at the same time the substantive model of interest is tested (Enders & Bandalos, 2001). That is, the model tested is the same model used to estimate missing information. Currently, FIML is available in most SEM software

programs and can be used for most analyses available in these programs, with some

exceptions. FIML has been recommended as the best choice for handling missing data in

structural equation modeling (Allison, 2003). While several SEM software programs are

capable of analyzing MI datasets, there are limitations to using this method with respect

to the ability to produce pooled fit indices. Based on this knowledge, the decision was

made to use FIML in the current study when testing EFA, CFA, and SEM models.

  With both missing data methods, missing values for each participant are estimated

from the observed data for that case. Thus, in addition to considering the degree of

missing data across the entire sample, consideration needs to be given to the extent of

missing data across a single case. While there is no guideline regarding how much

missing data across an individual case harms performance of ML or MI methods, the

decision was made to delete 41 cases with more than 25% of missing data due to

concerns that there is insufficient information on that case to yield unbiased estimates. Of

these 41 cases, more than 50% of data points were missing for the vast majority (85.4%,

$n = 35$) and 90% or more data was missing for almost half (46.3%, $n = 19$). Deleting

these 41 cases left a final sample of 418 participants.

  Data was missing on only 3.0% of the values for the final sample of 418;

however, it was still diffusely spread across 70.8% of cases and 90.7% of variables.

Little's MCAR test was still significant, $\chi^2 (11705) = 12262.80$, $p <. 001$, indicating the

data was still not MCAR. However, methods that assume data is MCAR still perform

very well if the data are merely missing at random (MAR). While there is no test to

determine if data is MAR, it is thought to be quite plausible in many cases (Schafer &

Graham, 2002). The plausibility of MAR is improved by including potential causes or

correlates of missingness, as well as variables that are correlated with those containing

missing data, in the model used to estimate missing values (Collins et al., 2001). Based

on a series of simulation studies, Collins et al. (2001) concluded that "the inclusion of

these variables is at worst neutral, and at best extremely beneficial" (p. 348), with a

possibility of increased statistical power and reduced bias.

As stated above, EFA and SEM were carried out using FIML and the model used

to predict missing values was the same as the substantive model tested. All other analyses

were performed with multiple imputed datasets. With limitations on the number of

variables that can be entered into a multiple imputation model, total scores were entered

as predictors instead of individual items where possible, and the variables to be predicted

were split into several different models with similar items together, typically according to

measure or scale. All of the items to be predicted within a particular model were also

predictors of other items being predicted. For example, each missing value on a CDSII

item was predicted by information contained in the general set of predictor variables used

for all variables (outlined below), but also by all other CDSII items. The following set of

variables were included as predictors in all multiple imputation models: age, gender, HSI

score, CDSII subscale scores, IPQ-R Treatment Control and Personal Control scores, and

total scores on perceived self-efficacy, causal type factors, perceived effectiveness of

psychosocial treatment, perceived effectiveness of pharmacological treatment, intention

to use psychosocial treatment, intention to use pharmacological treatment, and intention

to quit. Overall, the models used for FIML and MI were very similar but not identical.

Assumption of multivariate normality. After deleting cases with excessive

missing data, histograms, skewness and kurtosis values were examined for evidence of

univariate non-normality. The causal attribution rating for habit showed severe skew and kurtosis (absolute values greater than 2 and 7, respectively; Finney & DiStefano 2006) and lack of variability in responses, with 72.3% ($n = 295$) of participants selecting "strongly agree", 24.8% ($n = 101$) selecting "agree", and only 2.9% ($n = 12$) selecting one of the other three response options. Given the strong ceiling effect and lack of variation among responses, this variable was dropped from all inferential analyses and missing data procedures. A few other causal attribution items showed evidence of non-normality. In particular, ratings for stress and physiological addiction to nicotine exhibited the greatest levels of non-normality, both having moderate skewness and kurtosis. Non-normality in the form of mild to moderate skew and kurtosis ($< \pm 2$) was evident across other items in the hypothesized model. CDSII scale items revealed some mild and moderate skew and kurtosis. Non-normality was also observed across perceived effectiveness items; items pertaining to pharmacological treatments exhibited only mild deviation from normality, whereas distributions for most of the other treatments were moderately skewed. Treatment control items were either normally distributed or deviated from normality minimally. There was evidence of mild and moderate skew and kurtosis across several perceived behavioural control items. Moderate skewness was observed across almost all intention items, and severe skew was observed for perceived effectiveness of quitline/telephone counselling.

Maximum likelihood methods assume that data are multivariate normal, which is not tenable in the current study given evidence of univariate non-normality. With complete data, maximum likelihood produces parameter estimates (variances, regression coefficients and covariances) that remain consistent under non-normality; however,

estimates of standard errors do not remain consistent (Savalei, 2010). Estimates that are

consistent converge to true values with increasing sample size and therefore, are

approximately unbiased in large samples (Allison, 2003). One commonly utilized and

widely available approach to handling non-normality in SEM is maximum likelihood

estimation with adjustments made to standard errors and the model chi-square to reduce

the effect of non-normality, known as robust standard errors and the Satorra–Bentler

scaled chi-square statistic (Satorra & Bentler, 1994). Yuan and Bentler (2000) extended

Satorra and Bentler's corrections for non-normality to incomplete data. The resulting

robust standard errors and Yuan-Bentler T_2* test statistic have been found to perform

very well and outperform other methods with which they have been compared (Enders,

2001; Gold, Bentler, & Kim, 2003; Savalei & Bentler, 2005). Despite being completely

analogous to the widely available Satorra–Bentler corrections with complete non-normal

data, the Yuan-Bentler corrections for incomplete non-normal data are not available yet

in many software packages. The estimation method used in the current study to handle

missing data and non-normality was the MLR estimation method in Mplus, which

produces maximum likelihood parameter estimates with robust standard errors and a chi-

square test statistic that is asymptotically equivalent to the Yuan-Bentler T_2* test statistic

(Muthén & Muthén, 2012b). MLR estimation was used for EFA, CFA and SEM.

Outliers. Prior to conducting EFA, CFA and SEM, the data were screened for the

presence of multivariate outliers on the set of variables to be included in that analysis

using Mahalanobis distance values *(p < .001)*. Multivariate outliers were identified for the

set of IPQ-R causal attribution ratings submitted for EFA *(n = 7)*, as well as for the

variables included in the CFA and SEM models containing IPQ-R causal attribution

ratings ($n = 31$) and CDSII causal dimension scores ($n = 27$). These cases were examined and not deemed to be a result of researcher or participant error. All final models were tested with outliers excluded and yielded almost identical results; CFI values were on average .01 higher with outliers excluded but RMSEA and SRMR values differed even less, and the same factor loadings and path coefficients were still significant. Thus, all 418 cases were retained for all analyses.

Exploratory Factor Analysis of Causal Attribution Ratings

The MLR estimated correlation coefficients and descriptive statistics for ratings on the individual IPQ-R items are presented in Table 2. The most highly endorsed causes (aside from habit) were physiological addiction to nicotine and stress. On average, participants did *not* endorse genetics, heredity, overwork, social pressure, or family/relationship problems as causes of their smoking at present.

Data screening prior to EFA was conducted with SPSS, as several data screening features were not available in Mplus; listwise deletion was used to handle missing cases ($N = 396$) during data screening, since factor analysis in SPSS cannot be performed with multiple imputed datasets. Factorability of the correlation matrix was assessed using the Kaiser-Meyer-Olkin (KMO) Measure of Sampling Adequacy, inspection of off-diagonal elements of the anti-image correlation matrix, and Bartlett's Test of Sphericity (Field, 2005). The KMO measure was .85, well above the .50 cut-off recommended as minimally sufficient (Kaiser, 1974). Measures of sampling adequacy for individual items (off-diagonal elements of the anti-image correlation matrix) also exceeded the .5 cut-off and Bartlett's test was significant, $\chi^2(136) = 2630.14$, $p < .001$. Together these results indicate that the causal attribution items were suitable for factor analysis. All zero-order

Table 2

Causal Attribution Item Estimated Means, Standard Deviations, and Bivariate Correlations (N = 418)

Item	M	SD	1	2	3	4	5	6	7	8
1. Heredity	2.91	1.28	–							
2. Brain chemistry	3.57	1.09	.19***	–						
3. Genetics	2.72	1.20	.55***	.35***	–					
4. Physiological addiction to nicotine	4.31	0.97	.20*	.36***	.24***	–				
5. Feeling down	3.33	1.30	.13**	.10*	.18***	.15**	–			
6. Anxiety and worrying	3.67	1.22	.14**	.10*	.17***	.14**	.76***	–		
7. Mental attitude	3.19	1.30	.09	.11*	.16**	.16**	.68***	.56***	–	
8. Emotional state	3.72	1.12	.10*	.17***	.18***	.12*	.76***	.67***	.59***	–
9. Seeing other people smoke	3.45	1.18	.37***	.14**	.26***	.24***	.26***	.27***	.26***	.25***
10. Social pressure	2.16	1.09	.18***	.09	.20***	.09	.27***	.23***	.24***	.24***
11. Family/relationship problems	2.66	1.24	.15**	.10	.26***	.09	.50***	.45***	.38***	.45***
12. Stress	4.28	0.93	.15**	.19***	.16**	.18***	.45***	.60***	.38***	.51***
13. Work/school problems	3.29	1.26	.12*	.17***	.14**	.09	.44***	.50***	.41***	.44***
14. Financial stress/worries	3.18	1.34	.14**	.05	.25***	.09	.50***	.62***	.40***	.44***
15. Overwork	2.65	1.26	.23***	.21***	.37***	.09	.33***	.41***	.25***	.34***
16. Lack of willpower	3.95	1.12	.07	.12*	.13**	.34***	.19***	.17***	.22***	.14**
17. My personality or character	3.49	1.07	.11*	.27***	.24***	.20***	.14**	.14**	.21***	.18***

*p < .05. **p < .01. ***p < .001.

Item	9	10	11	12	13	14	15	16	17
9. Seeing other people smoke	–								
10. Social pressure	.34***	–							
11. Family/relationship problems	.36***	.45***	–						
12. Stress	.31***	.19***	.39***	–					
13. Work/school problems	.28***	.31***	.47***	.53***	–				
14. Financial stress/worries	.26***	.29***	.47***	.50***	.43***	–			
15. Overwork	.26***	.32***	.45***	.37***	.55***	.46***	–		
16. Lack of willpower	.11*	.09	.07	.19***	.10*	.14**	-.01	–	
17. My personality or character	.10*	.17***	.17***	.02	.05	.16**	.05	.11*	–

*$p < .05$. **$p < .01$. ***$p < .001$.

correlations between items were less than .9 in absolute value (see Table 2) and the determinant of the correlation matrix was greater than .00001, providing evidence that multicollinearity was not a concern. Inspection of bivariate scatterplots did not reveal evidence of curvilinearity or heteroscedasticity. As described earlier, the assumption of multivariate normality was violated with mild and moderate levels of skew observed among some causal attribution variables; therefore, MLR estimation was used to extract factors. The oblique Geomin rotation was used to permit correlations between factors.

Items exhibiting low factor loadings ($< \pm.30$), low communalities ($< .20$) or high cross loadings ($> \pm.30$) were candidates for elimination until a factor structure with acceptable fit and simple structure was obtained. No universal standard exists for a lower limit for factor loadings, and while the choice of a cut-off has been described as a matter of researcher preference (Tabachnick & Fidell, 2007), researchers commonly use an absolute value of .3 (Field, 2005).The lower and upper limit on the number of factors extracted for each analysis was set to 1 and 5, respectively.

The initial set of items analyzed included all causal attribution items, excluding habit. The chi-square test of overall model fit rejected the exact fit hypothesis for each factor solution. Examination of model fit indices suggested the five-factor solution was an acceptable fit to the data (see Table 3, Item Set 1). However, low factor loadings ($\leq .24$) and communality (.13) for the personality item suggested it shared very little common variance with other variables, and on this basis was dropped.

The EFA was repeated and the chi-square test of overall model fit again rejected the exact fit hypothesis for each factor solution (see Table 3, Item Set 2). A communality estimate greater than one (Heywood case) was evident in the five-factor solution,

Table 3

Model Fit for Exploratory Factor Analysis of Causal Attribution Items (N = 418)

Item Set	Description	# of Factors	MLR χ^2	df	RMSEA	90% CI	CFI	SRMR	BIC
1	Initial set[a]	1	754.88***	119	.113	[.105, .121]	.708	.093	20701.23
		2	498.02***	103	.096	[.087, .104]	.819	.061	20464.77
		3	332.28***	88	.081	[.072, .091]	.888	.047	20352.57
		4	269.66***	74	.080	[.069, .090]	.910	.037	20337.66
		5	170.36***	61	.065	[.054, .077]	.950	.030	20322.76
2	Personality deleted	1	696.47***	104	.117	[.109, .125]	.721	.093	19456.02
		2	465.27***	89	.101	[.092, .110]	.823	.059	19226.40
		3	291.69***	75	.083	[.073, .093]	.898	.046	19119.15
		4	284.20***	62	.093	[.082, .104]	.895	.035	19100.13
		5	–						
3	Personality and willpower deleted	1	638.23***	90	.121	[.112, .130]	.732	.094	18187.78
		2	418.76***	76	.104	[.094, .114]	.833	.056	17953.17
		3	237.35***	63	.081	[.071, .093]	.915	.042	17845.08
		4	**172.56***	**51**	**.076**	**[.063, .088]**	**.941**	**.034**	**17830.95**
		5	–						

Item Set	Description	# of Factors	MLR χ^2	df	RMSEA	90% CI	CFI	SRMR	BIC
4	Personality,	1	576.18***	77	.125	[.115, .134]	.748	.091	17042.94
	willpower, and	2	388.58***	64	.110	[.100, .121]	.836	.055	16817.86
	physiological	3	200.82***	52	.083	[.071, .095]	.925	.038	16711.91
	addiction deleted	4	–						
		5	122.55***	31	.084	[.069, .100]	.954	.024	16709.81

Note. Measures of model fit for factor solutions that included Heywood cases are not reported. Values for the retained factor solution are shown in bold. BIC = Bayesian Information Criteria; CI = confidence interval; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual.

[a]All causal attribution items excluding habit.

***$p < .001$.

rendering the factor solution invalid, and suggesting that it was not the correct number of factors to extract (Tabachnick & Fidell, 2007). While none of the factor solutions exhibited acceptable fit according to model fit indices, the most satisfactory fit was for the three-factor solution. While the BIC favoured a four-factor solution, a recent simulation (Preacher, Zhang, Kim & Mels, 2013) found that the RMSEA performed better than the BIC at identifying the 'true' number of factors in EFA. Therefore, the three-factor model was interpreted. Given poor fit, the factor solution was examined to identify any items that were candidates for deletion. The communality estimate for the 'lack of willpower' item was very low (.07), as were loadings on each factor (≤ .25), and on this basis was deleted.

The EFA was again repeated, with both personality and willpower items excluded. The chi-square values for each factor solution were significant, however, model fit indices supported the extraction of four factors (see Table 3, Item Set 3). Once again, the five-factor solution revealed a Heywood case, suggesting it was not the correct model to retain. The four-factor solution showed somewhat acceptable fit, with the RMSEA just under .08 and the CFI close to .95. The physiological addiction item had a low communality estimate (.14), and its highest factor loading (.35) was not much greater than the cut-off of .30. Therefore, the decision was made to reanalyze the EFA with this item deleted.

The three- and five-factor solutions for the fourth EFA analysis showed similar goodness of fit (see Table 3, Item Set 4); while the RMSEA and BIC values were almost identical, the CFI and SRMR values were more favourable for the five-factor solution. However, an examination of the pattern of factor loadings for the five-factor solution

revealed that the fifth factor was poorly defined, with no individual item loading higher

on this factor compared to the other four factors. On this basis, the five-factor solution

was rejected. The four-factor solution contained a Heywood case and therefore was not

valid. Based on the RMSEA, CFI and SRMR values, the three-factor solution was a

poorer fit to the data compared to the four-factor solution of the previous EFA (Table 3,

Item Set 3).

Thus, the four-factor solution with the physiological addiction item included

showed the best fit to the data. Additionally, the pattern of factor loadings appeared to

have good simple structure and interpretability (see Table 4), so this factor solution was

retained. The first factor was labelled Biological Attributions and items that loaded onto

this factor reflected both genetics and other biologically rooted causes, such as

physiological addiction to nicotine. The second factor was labelled Psychological

Attributions and comprised primarily of negative affective states, and 'mental attitude' or

thinking patterns that impact appraisal of life events and play a role in determining

emotional outcomes. The third factor was labelled Social Attributions and items reflected

both direct and indirect social factors that may prompt smoking for some individuals.

More direct social factors include seeing others smoke, while more indirect social factors

included relational problems and overwork. In some cases family or relationship

problems may include lack of supportiveness in trying to abstain from smoking, such as

where only one partner may be attempting to quit, and therefore may be seen as a more

direct cause as well. The fourth factor was labelled Stress Attributions and included a

general "stress" item, as well as problems or stress related to finances or work and

school. There appeared to be some overlap between the social and stress factors, as both

Table 4

Rotated Causal Attribution Item Factor Loadings (N = 418)

	Factor 1 *Biological Attributions*	Factor 2 *Psychological Attributions*	Factor 3 *Social Attributions*	Factor 4 *Stress Attributions*
Item		Rotated Factor Loadings		
Heredity (runs in my family)	**0.661***	0.009	0.022	-0.032
Brain chemistry	**0.419***	-0.029	-0.085	0.163
Genetics	**0.797***	0.016	0.063	-0.038
Physiological addiction to nicotine	**0.350***	0.096	-0.126	0.093
Feeling down (e.g., sad, lonely, empty, depressed)	0.004	**0.999***	0.007	-0.049
Anxiety and worrying	-0.010	**0.543***	-0.019	0.447*
My mental attitude (e.g., thinking about life negatively)	0.014	**0.637***	0.060	0.053
My emotional state	0.016	**0.664***	0.016	0.191*
Seeing other people smoke	0.243*	0.031	**0.307***	0.086
Social pressure	0.028	0.023	**0.644***	-0.111
Family/relationship problems	-0.012	0.189*	**0.630***	0.041
Overwork	0.184*	-0.078	**0.426***	0.320
Stress	0.023	0.060	0.034	**0.720***
Work/school problems	-0.068	0.030	0.389*	**0.460***
Financial stress/worries	0.027	0.180*	0.240	**0.407***

	Factor Correlations			
Biological Attributions	–			
Psychological Attributions	0.197*	–		
Social Attributions	0.354*	0.477*	–	
Stress Attributions	0.234*	0.571*	0.467*	–

*$p < .05$

overwork and work/school problems cross-loaded on both factors. The anxiety/worrying item also cross-loaded onto the Stress Attributions factor.

All four factors were positively correlated. The highest correlation was between psychological and stress attribution factors, not unexpected given the association between stress and negative emotional states. The social attributions factor was also moderately correlated with both psychological and stress attribution factors. Correlations with the biological attributions factor were lower, and were lowest between the biological and psychological attribution factors.

Scale Reliability

Cronbach's alpha reliability coefficients were computed for all measures of latent variables. Refer to Table 5 for Cronbach's alpha values for each measure, the possible range and observed range, and the MLR estimated mean and standard deviations. All Cronbach's alpha values were acceptable ($> .70$), except the values for Biological Attributions ($\alpha = .65$), Stability ($\alpha = .57$), and Locus of Causality ($\alpha = .63$). Item-level statistics were examined and no items were deemed to be detracting from internal consistency. Kline (2011) notes that somewhat lower levels of score reliability can be tolerated in latent variables methods compared with observed variable methods, and thus the measures were retained.

Descriptive Statistics

Study variable means. On average, agreement with each causal type was not high, falling between a neutral rating ("neither agree nor disagree") and "somewhat agree" (see Table 5). The exception was Social Attributions, for which the average rating fell between a neutral rating and "somewhat disagree". On the CDSII subscales, ratings

Table 5

Estimated Means and Standard Deviations, Reliability Coefficients, and Observed and Possible Ranges for Measures of Latent Variables (N = 418)

Variable	*M*	*SD*	Cronbach's Alpha	Observed Range	Possible Range
Biological Attributions	3.30	0.96	.65	1 − 5	1 − 5
Psychological Attributions	3.43	1.14	.89	1 − 5	1 − 5
Social Attributions	2.72	0.88	.70	1 − 5	1 − 5
Stress attributions	3.50	1.06	.72	1 − 5	1 − 5
Internal Control	5.63	2.31	.81	1 − 9	1 − 9
External Control	3.74	2.19	.74	1 − 9	1 − 9
Stability	5.55	1.90	.57	1 − 9	1 − 9
Locus of Causality[a]	6.46	2.06	.63	1 − 9	1 − 9
Perceived Behavioural Control	3.68	0.96	.83	1.4 − 5.8	1 − 5.8
Perceived Treatment Control	3.33	0.94	.86	1 − 5	1 − 5
Perceived Effectiveness of Pharmacological Treatment	2.82	1.88	.86	1 − 7	1 − 7
Perceived Effectiveness of Psychosocial Treatment	2.33	1.35	.93	1 − 7	1 − 7
Intention to Quit	3.38	1.41	.91	1 − 6.2	1 − 7
Intention to Use Pharmacological Treatment	2.88	1.74	.80	1 − 7	1 − 7
Intention to Use Psychosocial Treatment	2.05	1.29	.93	1 − 7	1 − 7

[a] Higher scores indicate internal locus.

fell close to the scale midpoint for both Internal Control and Stability. Locus of Causality was on average above scale midpoint and indicated that participants tended to view the causes of their smoking as being internal. The average ratings on External Control were below scale midpoint and indicated that participants did not tend to view their smoking as being under the control of external factors.

Average ratings on Perceived Behavioural Control and Perceived Treatment Control were approximately at scale midpoint, indicating that on average participants felt minimal control over their smoking. Average ratings on perceived effectiveness of both types of treatment and intentions to use treatment were at the lower range of the scale, indicating that many participants did not have high expectations for available treatments to help them quit smoking, and had little intention to use these treatments. Finally, intentions to quit fell somewhat below scale midpoint, indicating that intentions to quit were overall not strong.

Intercorrelations. See Table 6 for intercorrelations between measures of all latent variables. The causal attribution factors extracted from ratings on individual causal items exhibited very low or non-significant correlations with almost all other study variables. Attribution factors most highly correlated with perceived effectiveness and intentions to use psychosocial treatments. The only variable Internal Control was significantly correlated with, aside from other causal measures, was Perceived Behavioural Control ($r = .28$). External Control and Locus of Causality were correlated significantly but weakly with several non-causal variables (all rs $\leq .16$; see Table 6). Stability was significantly negatively correlated with Perceived Treatment Control ($r = -.29$), Perceived Behavioural Control ($r = -.36$), Perceived Effectiveness of Pharmacological Treatment

Table 6

Estimated Correlations Between Study Variables (N = 418)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Biological Attributions	–													
2. Psychological Attributions	.20***	–												
3. Social Attributions	.29***	.47***	–											
4. Stress Attributions	.15**	.62***	.55***	–										
5. Internal Control	-.16**	-.10*	-.03	-.08	–									
6. External Control	.14**	.04	.22***	.08	.08	–								
7. Locus of Causality	.01	-.06	-.07	-.06	.32***	-.11*	–							
8. Stability	.11*	.01	.02	.09	-.18***	.08	.29***	–						
9. Perceived Treatment Control	.01	.05	.08	.01	.05	.13**	-.07	-.29***	–					
10. Perceived Behavioural Control	-.08	-.12*	-.05	-.09	.28***	-.01	.03	-.36***	.35***	–				
11. Perceived Effectiveness: Pharmacological	.11*	.13*	.12*	.03	.07	.05	.15**	-.16**	.38***	.14**	–			
12. Perceived Effectiveness: Psychosocial	.17***	.19***	.24***	.17**	.04	.16**	.11*	-.07	.29***	.11*	.30***	–		
13. Intention to Quit	.05	.17**	.09	.10*	.03	.07	.01	-.24***	.34***	.34***	.26***	.21***	–	
14. Treatment Intention: Pharmacological	.12*	.12*	.06	.03	-.01	.07	.14**	.00	.29***	.05	.59***	.16**	.22***	–
15. Treatment Intention: Psychosocial	.16**	.18***	.19***	.15**	-.09	.15**	.04	.00	.20***	.04	.15**	.70***	.17***	.23***

*$p < .05$. **$p < .01$. ***$p < .001$.

(r = -.16), and Intention to Quit (r = -.24). The highest intercorrelation between causal attributions and causal dimensions was between External Control and Social Attributions (r = .22).

As expected, perceived effectiveness of treatment and intentions to use the same type of treatment were highly correlated (see Table 6). There was a low correlation between intentions to use psychosocial and pharmacological treatments (r = .23), as well as perceived effectiveness of both types of treatment (r = .30). Perceived Treatment Control was slightly more highly correlated with perceived effectiveness of treatment than intentions to use treatment, indicating the operation of other factors besides perceived effectiveness in determining intention to use treatment. Perceived Behavioural Control exhibited very weak correlations with perceived effectiveness of treatment and was not significantly associated with intention to use treatment. Both Perceived Behavioural Control and Perceived Treatment Control exhibited the same strength of correlation with Intention to Quit Smoking. Perceived Behavioural Control and Perceived Treatment Control were significantly correlated (r = .35).

Content Analysis of Open-Ended Responses

After an initial lower-level coding of the open-ended responses to the questions asking participants to list the cause(s) of their current smoking (see Appendix D) and to name the single most important cause (see Appendix E), causes were classified into 14 different categories (including an 'other' category). Each category with exemplifying quotes is presented in Table 7. Frequency of agreement or mention of each category, along with Cohen's kappa values for inter-rater reliability, are presented in Table 8.

Table 7

Content Analysis of Open-Ended Responses: Causal Attribution Categories and Sample Quotes

Category	Sample Quotes
Genetics/heredity • genetics • heredity	"heredity" "its run in the family"
Addiction • addicted • cravings • nicotine	"addicted to nicotine" "…I think I continue to smoke due to the cravings I get every hour or two. I do feel addicted."
Brain chemistry	"BRAIN CHEMISTRY"
Social influence • early family experience • image or wanting to fit in • friends/family/partner smoking	"I think I smoke because family members smoked and it reminds me of my loved ones…" "Started smoking to be cool…" "I smoke only when I am at my place of employment and when with other people who smoke."
Drinking alcohol/drug use • smoking while drinking alcohol or using drugs	"I think that it's closely linked to drinking. I do not actually like the taste of cigarettes and only smoke when drinking coffee, wine, or beer…" "…I cannot drink alcoholic beverages if I cannot smoke as the cravings become quite severe when drinking."
Stress • stress • relaxation	"…it's a good stress reliever…" "I smoke when I feel stressed and it makes me feel more calm." "...too much stress" "…It helps me to relax before and after work, as I have a stressful job."
Weight control • weight loss • appetite control • prevent weight gain	"I use smoking for weight control" "…curbs appetite…" "I smoke because …I am worried about the effects on my metabolism if I quit and gaining weight."
Habit • habit • routinely accompanies other activities • unconscious behaviour	"mainly out of habit" "It is also a habit so i smoke when but I get behind the wheel, with my coffee in the morning, after I eat, and before I go to bed." "It has simply become a habit. Just like always reading before bed or turning lights off in a room, I don't even think about it anymore, I just do it."
Enjoyment/pleasure • enjoy smoking	"…very much enjoy it…" "I enjoy the feeling of smoking…" "absolute enjoyment"

Category	Sample Quotes
Psychological/emotional • smoking in response to or to regulate emotions (anxiety, depression, anger, etc.) • personality • mental attitude	"... all psychological/emotional reasons, not physical addiction" "I often find myself feeling lonely and depressed and smoking makes me feel better…" "Now I can't quit because when I get ticked off I need a smoke." "Going without causes some degree of anxiety and discomfort" "my mental attitude" "state of mind"
Cognitive benefits • increased focus and concentration • improved creativity	"During the work day, it sharpens my mind and helps me focus." "Helps me concentrate/stay focused and stops drowsiness."
Lack of willpower or motivation to quit	"…although I think about quitting on almost a daily basis, I don't have much motivation." "although i have tried in the past to quit it makes it hard for me. i guess i lack motivation and willpower."
Boredom • boredom • fill time	"I smoke out of boredom…" "…something to do" "…at times it's simply to fill time."
Other • oral fixation • identity • time alone/break • etc.	"Need to put something in my mouth? Food…nails…cigarettes..." "I do feel that it's an engrained part of my identity." "One I like to smoke it is my reason to stop and take a break."

Table 8

Frequency of Causal Attributions According to Question Format (N = 418)

	Cued attribution list (IPQ-R)[a]		Open-ended list of causes		Most important cause		Cohen's kappa
	n	%	*n*	%	*n*	%	
Genetics/heredity	188	45.0	1	0.2	3	0.7	0.66
Addiction	343	82.1	124	29.7	95	22.7	0.98
Habit	396	94.7	115	27.5	110	26.3	0.94
Social influence	253	60.5	132	31.6	11	2.6	0.98
Drinking alcohol/drug use	–	–	11	2.6	2	0.5	1.00
Stress	363	86.8	132	31.6	78	18.7	0.93
Weight control	–	–	14	3.3	2	0.5	1.00
Enjoyment/pleasure	–	–	72	17.2	34	8.1	0.98
Psychological/emotional	363	86.8	39	9.3	32	7.7	0.97
Cognitive benefits	–	–	6	1.4	0	0	0.79
Lack of willpower or motivation to quit	307	73.4	5	1.2	27	6.5	0.93
Boredom	–	–	46	11.0	6	1.4	1.00
Brain chemistry	230	55.0	0	0	1	0.2	1.00
Other	–	–	31	7.4	14	3.3	0.44
Not sure/unknown	–	–	8	1.9	4	1.0	–
No response	0	0	25	6.0	23	5.5	–

Note. Percentages do not sum to 100% as more than one cause could be selected or listed.

[a] Frequency of "agree" and "strongly agree" responses.

Overall, Cohen's kappa values indicated a high level of reliability in the coding.

However, Cohen's kappa values were low for the 'genetics/heredity' and 'other'

categories. There was disagreement on only a single case for the genetics/heredity

category, however, because there was only one other case in this category in the

subsample, the disagreement had a substantial impact on the Cohen's kappa value. The

case was unique (no other code was similar in content) and the disagreement was not

believed to reflect on reliability of the category or its definition. The 'other' category was

hard to define and therefore it was difficult to establish inter-rater reliability.

Number and Type of Causal Attributions Endorsed

The number of causes to which smoking was attributed varied according to

question format, being higher for responses to the cued attribution list (IPQ-R). The total

number of causes for which participants selected "agree" or "strongly agree" from the

IPQ-R cued attribution list ranged from 0 to all 18 causes. The mean number endorsed

was 9.5 ($SD = 3.8$). Only two participants did not agree with any cause; the remaining

416 participants agreed with at least one cause. Approximately one-quarter of participants

(24.6%, $n = 103$) agreed with 1-6 causes, close to half agreed with 7-12 causes (51.2%,

$n = 214$), and the remaining (23.7%, $n = 99$) agreed with 13-18 causes.

To more accurately compare the number of causes endorsed between different

questions, the number of causes agreed with on the cued attribution list was also

calculated using the categories on which open-ended responses were coded (see Table 8).

For example, genetics and heredity are two separate items on the cued attribution list, but

are one category and therefore only coded once for open-ended responses. Using these

categories, the number of causes with which participants agreed with decreased and now

ranged from 0 to 5, with a mean of 3.9 ($SD = 1.1$). Almost three-quarters (72.2%, $n = 302$) of the sample agreed with 4 or 5 causes. Proportion of agreement was highest for habit, stress, and psychological/emotional causes (see Table 8), which were endorsed by almost the entire sample. Genetics and heredity were endorsed the least frequently.

Though several categories were not captured in the cued attribution list (e.g., boredom, enjoyment), the number of causes agreed with was still somewhat higher compared to the open-ended question that asked participants "What do think are the causes or reasons for your smoking now?" Whether the 'other' category was included or excluded, the number of causes listed in this open-ended question ranged from 0 to 5, with a mean of 1.7 ($SD = 1.0$). Almost three-quarters of the sample (72.2%, $n = 302$) listed 1 or 2 causes for their current smoking. The most commonly reported causes were social influence, stress, addiction, and habit.

While participants were asked to list causes of their *current* smoking, it was evident that many references to social influence in the open-ended list were referring to the past and why they began smoking initially, often decades ago. Because context was not always provided, it was not possible to reliably separate those who were referring to social factors as a past influence only and those who still find it a causal influence on their smoking today. Therefore, the decision was made to code it as a current causal factor, and interpret with caution. Very few (2.6%) participants listed a social factor as the most important cause of their current smoking, while habit, addiction, and stress remained the most frequently mentioned causes. This supports the impression that social influence was generally not considered an important causal factor at present, but was an important factor in initial smoking uptake.

In order to examine the relationship between causal dimensions and causes reported in open-ended responses, the mean scores on the CDSII subscales were compared using independent t-tests for participants who reported a particular cause versus those who did not. Given the number of comparisons, a Bonferroni correction to control the familywise error rate was applied (to each open-ended question separately), and a *p* value of .001 was used as the threshold for significance. Few statistically significant findings were observed. Those who listed drinking alcohol or drug use as one of the causes of their smoking reported significantly higher scores on the Internal Control scale, $M = 6.87$, $SD = 1.20$ vs. $M = 5.58$, $SD = 2.10$, $p < .001$. Those who reported that drinking alcohol or drug use was the most important cause of their smoking had significantly lower scores on the External Control scale, $M = 2.33$, $SD = 0.0$ vs. $M = 3.84$, $SD = 1.97$, $p < .001$. Finally, those who listed addiction as one of the causes of their smoking had lower scores on the Internal Control scale compared to those who did not list addiction, $M = 4.98$, $SD = 2.16$ vs. $M = 5.88$, $SD = 2.00$, $p < .001$. Scores on the Stability and Locus of Causality subscales did not significantly differ based on causal attributions listed in open-ended questions.

Several other differences approached significance at $p \leq .01$. Similar to the finding above, those who reported addiction as the single most important cause of their smoking had lower mean scores on Internal Control compared to those who did not, $M = 5.08$, $SD = 2.17$ vs. $M = 5.77$, $SD = 2.04$, $p = .004$. Those who listed addiction as a cause of their smoking also tended to rate the stability of causes higher overall, $M = 5.82$, $SD = 1.75$ vs. $M = 5.33$, $SD = 1.74$, $p = .01$. Those who listed enjoyment/pleasure as a cause of their smoking rated causes overall as more internal,

$M = 6.86$, $SD = 1.73$ vs. $M = 6.22$, $SD = 1.72$, $p = .004$. Those who reported that

enjoyment/pleasure was the most important cause of their smoking rated having a higher

level of internal control over the causes of their smoking, $M = 6.51$, $SD = 1.78$ vs.

$M = 5.53$, $SD = 2.10$, $p = .009$. For infrequently mentioned causes, small cell sizes and

low power may have permitted only larger effect sizes to be detected.

Relationship of Causal Attributions with Level of Nicotine Dependence and Treatment History

   Causal attributions and level of nicotine dependence. Actual level of

physiological dependence on nicotine varies across individuals, and may impact the type

of causal attributions an individual forms. To examine this, the associations between

Heaviness of Smoking Index (HSI) scores and both causal attribution types and causal

dimensions were examined. HSI category (low, moderate, high) was not associated with

mention of any particular type of causal attribution provided in open-ended responses (all

ps > .05). HSI score was correlated significantly, but minimally, with ratings on causal

dimensions and causal types. HSI score was positively correlated with Stability, $r = .21$,

$p < .001$, and negatively correlated with Internal Control scores, $r = -.22$, $p < .001$. HSI

was not correlated with Locus of Causality and External Control scores. HSI score was

also correlated with Biological Attributions, $r = .20$, $p < .001$, Psychological Attributions,

$r = .17$, $p < .001$, and Stress Attributions, $r = .15$, $p = .003$.

   Causal attributions and smoking cessation treatment history. The associations

between prior use of smoking cessation treatment and both causal dimensions and type of

causal attributions were examined. CDSII and IPQ-R ratings were compared for the

group of individuals who had previously used a particular cessation aid or treatment

versus those who had not. Bonferroni correction was applied to control the familywise

error rate, and a $p < .001$ was the cut-off for significance. No significant findings

emerged for scores on each CDSII scale. Participants who had previously used

varenicline had a slightly higher overall rating on Biological Attributions, $M = 3.57$,

$SD = 0.78$ vs. $M = 3.29$, $SD = 0.81$, $p = .001$. A similar result approached significance for

NRT, such that those who had previously used NRT had a somewhat higher overall

rating on Biological Attributions compared to participants who had not used NRT,

$M = 3.58$, $SD = 1.04$ vs. $M = 3.24$, $SD = 1.13$, $p = .003$.

Given the very large number of comparisons required to examine the association

between prior use of cessation aids and each type of cause coded in open-ended

responses, the analysis was instead limited to the top five most frequently cited causes:

addiction, habit, stress, enjoyment/pleasure and psychological/emotional. While social

influence was one of the top cited causes for the open-ended list of causal attributions, as

mentioned previously, there is good reason to believe that social factors were an

important reason for initial uptake of smoking versus continued smoking, and therefore it

was not analyzed; it also was not one of the top five most important causes. Applying a

Bonferroni correction to each open-ended question separately, a p value of .001 was the

threshold for significance.

Those who listed addiction as one of the causes of their smoking more frequently

reported having used NRT previously compared to those who did not mention addiction,

83.2% ($n = 79$) vs. 65.6% ($n = 212$), $p < .001$. Similarly, those who reported that

addiction was the most important cause of their smoking were more likely to have

previously used NRT compared to those who did not, 82.3% ($n = 102$) vs. 64.3%

($n = 189$), $p < .001$. Those who indicated that enjoyment/pleasure was the most important cause of their smoking were significantly less likely to report having used self-help manuals to assist with quitting in the past, 8.8% ($n = 3$) vs. 33.3% ($n = 128$), $p = .001$.

Several other differences approached significance at $p \le .01$. Individuals who reported enjoyment/pleasure as the most important cause of their smoking were also less likely to report previously using NRT, 50.0% ($n = 17$) vs. 71.4% ($n = 274$), $p = .009$, bupropion, 17.6% ($n = 6$) vs. 39.6% ($n = 152$), $p = .011$, and varenicline, 11.8% ($n = 4$) vs. 32.6% ($n = 125$), $p = .012$. In addition, those who listed enjoyment/pleasure as a cause of their smoking were also less likely to have previously used NRT, 55.6% ($n = 40$) vs. 72.5% ($n = 251$), $p = .004$. Those who listed psychological/emotional causes of their smoking were more likely to have used group counselling, 23.7% ($n = 9$) vs. 10.0% ($n = 38$), $p = .011$, and alternative therapies, 47.4% ($n = 18$) vs. 24.2% ($n = 92$), $p = .002$. Whether or not habit or stress were mentioned as causes of smoking had no association with past use of smoking cessation treatment.

Genetics Knowledge

All four true/false genetics knowledge items presented to participants were true, however, only 36% to 51% of participants agreed with each statement (see Table 9). Approximately half of the sample believed that genes exist which increase the chance to become addicted to smoking, while less than half believed that genes exist that decrease the likelihood of becoming addicted to smoking, that the chance of being addicted is influenced by heredity, or that a genetic predisposition to nicotine dependence can be passed on to children even if the parent has not smoked. Thus, overall, knowledge regarding the influence of genetic factors on nicotine dependence was not high.

Table 9

Knowledge of the Influence of Genetic Factors on Smoking Addiction (N = 418)

Statement	True % (*n*)	False % (*n*)
The chance to become addicted to smoking is influenced by the presence of certain hereditary traits (genes).	47 (196)	53 (222)
Genes exist that increase the chance to become addicted to smoking.	51 (215)	49 (203)
A parent with a genetic predisposition to get addicted to smoking will transfer this predisposition to its children, even when the parent doesn't smoke or has never smoked.	37 (156)	63 (262)
Genes exist that decrease the chance to become addicted to smoking.	36 (151)	64 (267)

CFA Measurement Models

Confirmatory factor analysis was used to assess the measurement model component of each hypothesized model, to establish the suitability of observed variables as indicators of latent variables, prior to testing the structural model component that examines the relationships between latent factors.

The hypothesized models categorized treatment into pharmacological treatment and psychosocial treatments that address cognitions, behaviours and/or emotions as opposed to biological processes. While perceived effectiveness and intentions regarding alternative treatments were assessed, from a substantive perspective they do not cleanly fit into either category. 'Alternative' treatments encompass a wide range of treatments, some of which more closely resemble counselling (e.g., hypnosis), and some are analogous to pharmacotherapy (e.g., herbal supplements). The pattern of correlations reflected this; perceived effectiveness ratings for alternative treatments correlated to a similar extent with both pharmacological and non-pharmacological treatments, and were relatively low ($r \leq .40$). Thus, they were not included in the CFA or SEM models, but are included in descriptive results.

Latent factors can either be specified with individual items or aggregated items, also known as parcels (the sum or average of two or more items). Compared with aggregate-level data, item-level data tend to have lower reliability and are more unstable or "noisy" (Kline, 2011; Little, Cunningham, Shahar, & Widaman, 2002). As a result, some advocate reducing the number of items into fewer indicators (provided they are unidimensional) in order to reduce various sources of sampling error and improve model fit. However, some argue the use of parcels can obscure model misspecification (e.g.,

may mask cross-loadings). Little and colleagues (2002) contend that the decision to

parcel or not to parcel can be dictated by the objectives of the study. In particular, if one

seeks to understand the exact relations among the individual items, one should not parcel.

In such a case, missing a double loading or correlated error at the item level would

signify a failure to fully understand the pattern of the observed data. Alternatively, if the

relationships between constructs are what is of primary interest, parceling may be more

strongly warranted. In such a case, for example, "if a dual loading were eliminated

through aggregating items in order to specify a clean latent construct, then the goals of

the researcher are realized through parceling, not hindered by it" (Little et al., 2002,

p. 169). In light of this, it was determined that parceling items on latent factors would be

appropriate in the current study. Regardless of the research objectives, it is recommended

that each latent variable be measured by at least three or four indicators (Kline, 2011).

Therefore, parceling was not considered where a latent factor had four indicators or less.

Perceived effectiveness of each treatment was measured with two similarly

worded items per treatment (see Appendix I); the two items were summed to construct a

parcel for each treatment. In addition, perceived effectiveness of individual and group

counselling items were summed to create a perceived effectiveness of counselling parcel.

All four counselling items had inter-item correlations between .62 and .81 and excellent

internal reliability (Cronbach's α = .92). A parcel for intention to seek individual and

group counselling was also created; inter-item correlations between these items ranged

from .62 to .85, and the four items also had excellent internal reliability (Cronbach's

α = .91). The 10 items measuring perceived behavioural control were reduced to four

parcels. The first parcel was created by summing the four self-efficacy items. Inter-item

correlations for the four items ranged from .45 to .72 and had good internal reliability

(Cronbach's α = .84). Three parcels were created from the six Personal Control items,

which overall had good internal reliability (Cronbach's α = .77). As recommended by

Little et al. (2002), each negatively worded item that was reverse coded was combined

with a positively worded item, to reduce any negativity bias, positivity bias or

acquiescence bias. Aside from ensuring each negatively worded item was paired with a

positively worded item, all items were randomly assigned to a parcel (Little et al., 2002).

     Causal type factors. The initial hypothesized model (with Stress Attributions

added) provided somewhat acceptable fit to the data according to the RMSEA and

SRMR, but the CFI value was much lower than what is considered acceptable (see Table

10). In an effort to improve model fit, modification indices were inspected. The

modification indices suggested correlating several error terms would improve model fit.

     Correlating error terms in order to improve model fit should be applied

judiciously to avoid capitalizing on chance and fitting small idiosyncratic characteristics

that are unlikely to be replicated (MacCallum, Roznowski, & Necowitz, 1992); thus, any

model respecification that includes correlated errors must be supported by a strong

substantive and/or empirical rationale (Kline, 2011). Inspection of the modification

indices revealed several very large values that suggest correlating errors between

perceived effectiveness of a treatment and intention to use the same treatment. A

correlation between errors reflects the assumption that the corresponding variables share

at least one common cause (Kline, 2011), and in fact it is quite reasonable in this case to

expect that beliefs regarding the effectiveness of a treatment and intentions to use that

same treatment may share an underlying cause or set of causes that were not incorporated

Table 10

Model Fit for Measurement Model with Causal Types (N = 418)

Model	Path added	MLR χ^2	df	RMSEA	90% CI	CFI	SRMR
1	Initial model	2639.57***	685	.083	[.079, .086]	.760	.064
2	$E_{PE\ NRT} \leftrightarrow E_{TI\ NRT}$	2383.72***	684	.077	[.074, .080]	.791	.060
3	$E_{PE\ SELF} \leftrightarrow E_{TI\ SELF}$	2088.28***	683	.070	[.067, .074]	.827	.059
4	$E_{PE\ VAR} \leftrightarrow E_{TI\ VAR}$	1819.68***	682	.063	[.060, .067]	.860	.060
5	$E_{PE\ COUN} \leftrightarrow E_{TI\ COUN}$	1628.48***	681	.058	[.054, .061]	.884	.060
6	$E_{PE\ WEB} \leftrightarrow E_{TI\ WEB}$	1472.00***	680	.053	[.049, .056]	.903	.060
7	$E_{PTC\ 4R} \leftrightarrow E_{PTC\ 1R}$	1420.38***	679	.051	[.047, .055]	.909	.060
8	$E_{TI\ SELF} \leftrightarrow E_{TI\ WEB}$	1324.91***	678	.048	[.044, .052]	.921	.060

Note. CI = confidence interval; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual; E = error term; PE = perceived effectiveness; TI = treatment intention; COUN = counselling; NRT = nicotine replacement therapy; SELF = self-help materials; VAR = varenicline; WEB = web-based support; R = reverse-coded item.

***$p < .001$.

in the current model.

In order for a measurement model with error correlations to be identified (i.e., for it to be theoretically possible for the computer to derive a unique estimate for every model parameter), for every pair of factors there must be at least two indicators (one from each factor), whose error terms are uncorrelated (Kenny, Kasher, & Bulgur, 1998; Kline, 2011). Thus, one pharmacological treatment (bupropion) and one psychosocial treatment (telephone counselling), were not permitted to have correlated errors between their respective indicators on perceived effectiveness and treatment intention factors.

Modification indices also indicated that the correlation of additional error terms may improve model fit, and two correlations were deemed justifiable. Method effects (e.g., response bias) can be modeled with correlated errors, such as when positively and negatively worded items are both present, and may help achieve good model fit (e.g., Tomás & Oliver, 1999). Thus, the pair of errors for the two negatively worded items on the Perceived Treatment Control scale were allowed to correlate. Finally, error terms for the intention to use self-help materials and intention to use web-based support were allowed to correlate, as there is some overlap in the content of the items. The self-help treatment item included the use of web-based information and tools, and web-based social support is not only one means of gaining information, but also websites that contain forums for smoking cessation support in many cases contain additional resources on quitting smoking.

After allowing seven pairs of error terms to correlate, as described above, the model fit was considerably improved (see Table 10). RMSEA and SRMR decreased, and met cut-offs for acceptable fit, and the CFI value (.92) was close to the recommended .95

value (Hu & Bentler, 1999). While the CFI value would ideally be closer to .95, the

RMSEA value is considered more appropriate in confirmatory testing compared to the

CFI (Rigdon, 1996), and its value met the cut-off for what is considered close fit (Brown

& Cudeck, 1993). Thus, this final measurement model was retained for testing the

hypothesized structural model. See Table 11 for standardized factor loadings for each

latent factor. All standardized factor loadings were significant at $p < .001$.

Causal dimensions. Similar to what was found with causal type factors, the fit for

the initial hypothesized model was poor (see Table 12), and large modification indices

were produced suggesting the addition of correlations between several error terms would

improve model fit. Allowing the same seven error terms to correlate substantially

improved model fit, such that the final model had sufficiently good fit (see Table 12).

The RMSEA value was less than .05 and indicated close fit, the SRMR fell below the .08

recommended cut-off, and the CFI was close to .95. Thus, this measurement model was

deemed acceptable for testing the hypothesized structural model. See Table 13 for

standardized factor loadings for each latent factor. All standardized factor loadings were

significant at $p < .001$.

Structural Equation Models Predicting Intentions to Quit and Intentions to Utilize

Smoking Cessation Treatment

Causal type model. The initial hypothesized model (see Figure 2) was modified to

include the Stress Attributions factor. Because stress can be perceived as both internal

(our reaction to events and ability to cope with them) and external (demands placed on us

by our environment), with varying degrees of actual and perceived controllability based

Table 11

Standardized Factor Loadings for Measurement Model with Causal Types (N = 418)

	Latent Variable			
	Biological Attributions	Psychological Attributions	Social Attributions	Stress Attributions
Item	Standardized factor loadings			
Heredity	.64			
Brain chemistry	.42			
Genetics	.84			
Physiological addiction to nicotine	.33			
Feeling down		.90		
Anxiety and worrying		.84		
Mental attitude		.72		
Emotional state		.82		
Seeing other people smoke			.50	
Social pressure			.51	
Family/relationship problems			.70	
Overwork			.68	
Stress				.69
Work/school problems				.70
Financial stress/worries				.71

	Latent Variable			
	Perceived Behavioural Control	Perceived Treatment Control	Perceived Effectiveness-Pharmacological Treatment	Perceived Effectiveness-Psychosocial Treatment
Item	Standardized factor loadings			
Self-efficacy[a]	.61			
Whether or not I smoke depends entirely on me + My actions will have no effect on whether I quit smoking (R)[a]	.63			
What I do can determine whether I stop smoking + I have the power to influence my smoking[a]	.83			
Nothing I do will affect my smoking (R) + There is a lot which I can do to control my cravings and withdrawal symptoms [a]	.72			
There is very little treatment can do to stop my smoking (R)		.59		
Treatment for quitting smoking will be effective for me		.95		
Treatment can control my smoking		.84		
There is no treatment that can help my smoking (R)		.65		
Perceived effectiveness[a]				
NRT			.40	
Bupropion			.97	
Varenicline			.75	
Counselling				.79
Web-based support				.82
Self-help materials				.61
Telephone support/quitline				.95

	Latent Variable		
	Quit Intention	Treatment Intentions-Pharmacological	Treatment Intentions-Psychosocial
Item	Standardized factor loadings		
Intention to quit in next month[a]	.80		
Intention to quit in next 6 months[a]	.87		
Plan to quit	.83		
Intention to use treatment[a]			
NRT		.31	
Bupropion		.97	
Varenicline		.63	
Counselling			.72
Web-based support			.76
Self-help materials			.61
Telephone support/quitline			.96

Note. All factor loadings were significant at $p < .001$. NRT = nicotine replacement

therapy; R = reverse-coded item.

[a] Item parcel.

Table 12

Model Fit for Measurement Model with Causal Dimensions (N = 418)

Model	Path added	MLR χ^2	df	RMSEA	90% CI	CFI	SRMR
1	Hypothesized model	2354.90***	574	.086	[.083, .090]	.738	.067
2	$E_{PE\,NRT} \leftrightarrow E_{TI\,NRT}$	2104.05***	573	.080	[.076, .084]	.775	.063
3	$E_{PE\,SELF} \leftrightarrow E_{TI\,SELF}$	1798.57***	572	.072	[.068, .075]	.819	.062
4	$E_{PE\,VAR} \leftrightarrow E_{TI\,VAR}$	1527.18***	571	.063	[.059, .067]	.859	.063
5	$E_{PE\,COUN} \leftrightarrow E_{TI\,COUN}$	1333.34***	570	.057	[.053, .061]	.888	.062
6	$E_{PE\,WEB} \leftrightarrow E_{TI\,WEB}$	1178.34***	569	.051	[.047, .055]	.910	.062
7	$E_{PTC\,4R} \leftrightarrow E_{PTC\,1R}$	1128.36***	568	.049	[.044, .053]	.917	.062
8	$E_{TI\,SELF} \leftrightarrow E_{TI\,WEB}$	1033.77***	567	.044	[.040, .049]	.931	.062

Note. CI = confidence interval; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; SRMR = Standardized Root Mean Square Residual; E = error term; PE = perceived effectiveness; TI = treatment intention; COUN = counselling; NRT = nicotine replacement therapy; SELF = self-help materials; VAR = varenicline; WEB = internet-based support; R = reverse-coded item.

***$p < .001$.

Table 13

Standardized Factor Loadings for Measurement Model with Causal Dimensions
(N = 418)

	Latent Variable			
	Internal Control	External Control	Locus	Stability
Item	Standardized factor loadings			
Not manageable by you — Manageable by you	.79			
You cannot regulate — You can regulate	.81			
Over which you have no power — Over which you have power	.74			
Over which others have no control — Over which others have control		.62		
Not under the power of other people — Under the power of other people		.74		
Other people cannot regulate — Other people can regulate		.78		
That reflects an aspect of the situation — That reflects an aspect of yourself			.46	
Outside of you — Inside of you			.83	
Something about others — Something about you			.65	
Temporary — Permanent				.54
Variable over time — Stable over time				.51
Changeable —Unchangeable				.63

	Latent Variable			
	Perceived Behavioural Control	Perceived Treatment Control	Perceived Effectiveness- Pharmacological Treatment	Perceived Effectiveness- Psychosocial Treatment
Item	Standardized factor loadings			
Self-efficacy[a]	.61			
Whether or not I smoke depends entirely on me + My actions will have no effect on whether I quit smoking (R)[a]	.64			
What I do can determine whether I stop smoking + I have the power to influence my smoking[a]	.81			
Nothing I do will affect my smoking (R) + There is a lot which I can do to control my cravings and withdrawal symptoms [a]	.74			
There is very little treatment can do to stop my smoking (R)		.60		
Treatment for quitting smoking will be effective for me		.94		
Treatment can control my smoking		.85		
There is no treatment that can help my smoking (R)		.66		
Perceived effectiveness[a]				
NRT			.41	
Bupropion			.97	
Varenicline			.75	
Counselling				.79
Web-based support				.82
Self-help materials				.61
Telephone support/quitline				.95

	Latent Variable		
	Quit Intention	Treatment Intentions-Pharmacological	Treatment Intentions-Psychosocial
Item	Standardized factor loadings		
Intention to quit in next month[a]	.80		
Intention to quit in next 6 months[a]	.87		
Plan to quit	.83		
Intention to use treatment[a]			
NRT		.31	
Bupropion		.96	
Varenicline		.64	
Counselling			.72
Web-based support			.76
Self-help materials			.61
Telephone support/quitline			.96

Note. All factor loadings were significant at $p < .001$. NRT = nicotine replacement therapy; R = reverse-coded item.

[a] Item parcel.

on the source of the stress, it is challenging to put forward hypotheses about how attributions to stress will be related to the other variables in the model, without prior empirical evidence. Thus, the Stress Attributions factor was incorporated into the model, and its associations with Perceived Behavioural Control and Perceived Treatment Control were examined in an exploratory manner.

This revised hypothesized model did not converge. Two recommended solutions, increasing the number of iterations (Muthén & Muthén, 2012b) and entering different starting values (Muthén & Muthén, 2012b; Kline, 2011), did not resolve the convergence issue. As recommended by Kline (2011), a simpler model was tested with parameters added individually to detect whether a particular parameter was causing empirical underidentification. Without the path from the Stress Attributions factor to Perceived Treatment Control, the model did converge without problem, and is suspected to have caused empirical underidentification. Because this path was being tested in an exploratory manner and was not of primary interest, nor was it mediating a relationship between other variables, removal of this path was not considered a threat to the hypotheses of this study.

The respecified model (see Figure 4) was submitted for analysis; the chi-square test was rejected and other fit indices slightly deviated from acceptable cut-offs: MLR χ^2 (705, $N = 418$) = 1450.43, $p < .001$; RMSEA = .050 [.047, .054]; CFI = .908; SRMR = .085. Examining the individual parameter estimates, none of the path coefficients between each causal attribution type factor and either Perceived Treatment Control or Perceived Behavioural Control was statistically significant. The standardized and unstandardized path coefficients for Perceived Treatment Control on Biological
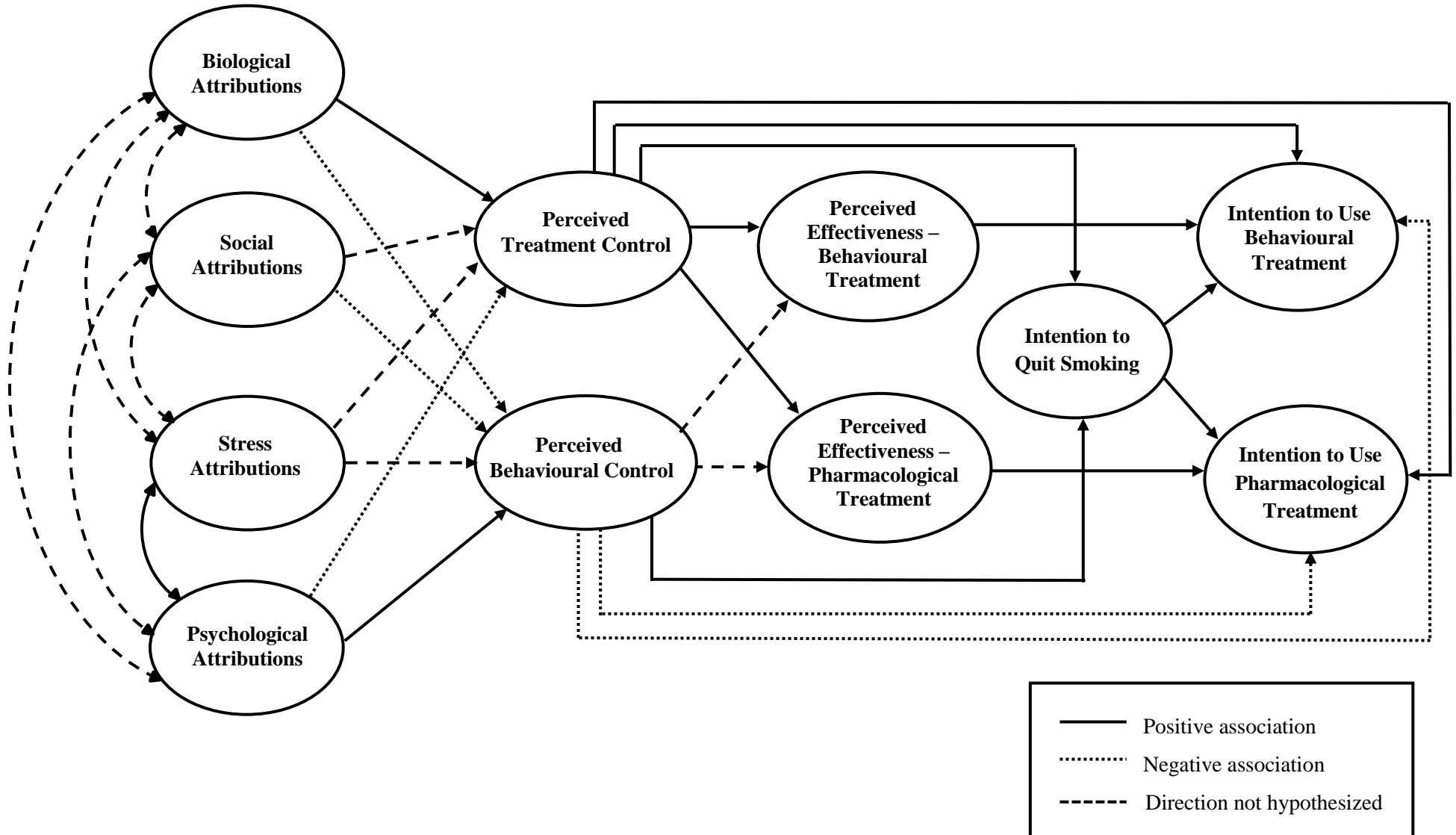
Figure 4. Revised hypothesized causal type model.

Attributions and Psychological Attributions were close to zero, suggesting that Stress

Attributions may have been very low as well, which could have been the source of

empirical underidentification and nonconvergence problems.

Several other path coefficients were not statistically significant. An examination

of the modification indices did not suggest that causal attribution factors would have

contributed toward prediction of any other variable in the model. This is not surprising in

light of the low correlations between causal attribution factors and other study variables

(see Table 6). Because the purpose of the model was to determine whether causal

attribution factors predict intentions to quit and to use smoking cessation treatment, and

the causal attribution factors did not significantly contribute to the model, the model was

not examined any further. The model fit could have been improved by the deletion of the

causal attribution factors and other non-significant paths between the other variables,

however, those additional variables are included in the second model incorporating causal

dimensions, and are examined further in that context.

Causal dimension model. The initial hypothesized model with causal dimensions

(see Figure 3) did not exhibit any convergence problems as seen with the previous model

incorporating type of causal attributions. While the chi-square test of exact fit was

rejected, MLR χ^2 (596, $N = 418$) = 1117.24, $p < .001$, the other fit indices indicated

acceptable fit: RMSEA = .046 [.042, .050], CFI = .923, SRMR = .073. Standardized

regression weights and associated p values were examined to determine if any of the

proposed model paths were nonsignificant. The following nine paths were found to be

nonsignificant, and were deleted one at a time, in the following order: (i) Locus of

Causality to External Control; (ii) Internal Control to Perceived Treatment Control;

(iii) External Control to Perceived Behavioural Control; (iv) Perceived Behavioural Control to Perceived Effectiveness of Pharmacological Treatment; (v) Perceived Behavioural Control to Perceived Effectiveness of Psychosocial Treatment; (vi) Intention to Quit Smoking to Intention to Use Pharmacological Treatment; (vii) Intention to Quit Smoking to Intention to Use Psychosocial Treatment; (viii) Perceived Treatment Control to Intention to Use Pharmacological Treatment; (ix) Perceived Treatment Control to Intention to Use Psychosocial Treatment. Each standardized regression weight was still non-significant after deletion of the previous path, and thus was still a candidate for deletion. The chi-square test of fit was again significant for the revised model, MLR χ^2 (601, $N = 418$) = 1117.95, $p < .001$, and the other fit indices indicated acceptable fit: RMSEA = .045 [.041, .049], CFI = .924, SRMR = .073.

Modification indices were examined to determine whether the addition of any paths could improve overall model fit. According to Byrne (2010), the most important factors to consider when deciding whether to include additional parameters into a model are the extent to which: (a) they are substantively meaningful, (b) the existing model already exhibits adequate fit, and (c) the Expected Parameter Change (EPC) is substantial. Additionally, she points out that scientific parsimony should be considered as well. Taking these factors into consideration, one additional path was a candidate for addition to the model, a path from Perceived Treatment Control to Perceived Behavioural Control. If an individual perceives that using a particular treatment will help control their smoking, and use of this treatment is under the individual's control, then this may lead to an increased level of Perceived Behavioural Control, though via a different pathway. When the path was added, the model fit statistics were very similar to the previous

model: MLR χ^2 (600, $N = 418$) = 1099.90, $p < .001$, RMSEA = .045 [.040, .049],

CFI = .926, SRMR = .072. In order to determine whether the model had statistically

significant increased fit, a chi-square difference test was attempted. However, subtraction

of the MLR χ^2 values yielded a negative value, as sometimes occurs when comparing

robust chi-square values (Asparouhov & Muthén, 2010). Satorra and Bentler (2010)

described calculation of an alternative robust chi-square to resolve the issue of negative

values, and instructions for its computation in Mplus are available (Asparouhov &

Muthén, 2010). Unfortunately, computation of the strictly positive Satorra-Bentler chi-

square difference test was not achievable with the current dataset and no other method of

calculating a chi-square difference test was available (L. Muthén, personal

communication, May 3, 2013). Thus, the BIC values were compared in lieu of a chi-

square difference test. The BIC value for the model without the path was 62436.90 and

the BIC value for the model with the additional path was 62424.17, a difference of 12.73.

As lower BIC values indicate better fit, and a difference in BIC values greater than 10 is

considered strong evidence in favour of one model over another (Kass & Raftery, 1995),

the model with the additional path was considered to have better fit and was retained.

Examination of the standardized regression coefficients and p values revealed that

the path from Locus of Causality to Perceived Treatment Control was no longer

significant in the revised model and was deleted. The fit of the model was once again

very similar to that of the previous model: MLR χ^2 (601, $N = 418$) = 1103.62, $p < .001$,

RMSEA = .045 [.041, .049], CFI = .926, SRMR = .073, BIC = 62421.51. Both the

RMSEA point estimate and its 90% confidence were below .05 and thus indicated close

fit of the model to the data (Browne & Cudeck, 1993; MacCallum et al., 1996). Similarly,

the CFI value was close to .95 and the SRMR was below .08, cutoffs recommended by

Hu & Bentler (1999) as indicating good fit. Thus the model was considered to have good

fit and was retained as the final model. See Figure 5 for the final version of the model.

Locus of Causality and Internal Control both had positive direct effects on

Perceived Behavioural Control. Locus of Causality also had a significant indirect effect

on Perceived Behavioural Control ($\beta = 0.10$, $p = .003$), mediated by Internal Control.

Stability had a direct negative effect on both Perceived Behavioural Control and

Perceived Treatment Control. Finally, External Control had a positive direct effect on

Perceived Treatment Control only. Perceived Treatment Control had a positive direct

effect on Perceived Behavioural Control and both Perceived Effectiveness of

Pharmacological Treatment and Perceived Effectiveness of Psychosocial Treatment.

Perceived effectiveness significantly predicted intention to use the same type of

treatment. Perceived Behavioural Control was not associated with perceived

effectiveness, but was negatively associated with intentions to use both types of

treatment. Finally, both Perceived Behavioural Control and Perceived Treatment Control

predicted Intention to Quit Smoking.

Table 14 summarizes the level of support for each study hypothesis based on the

above findings.
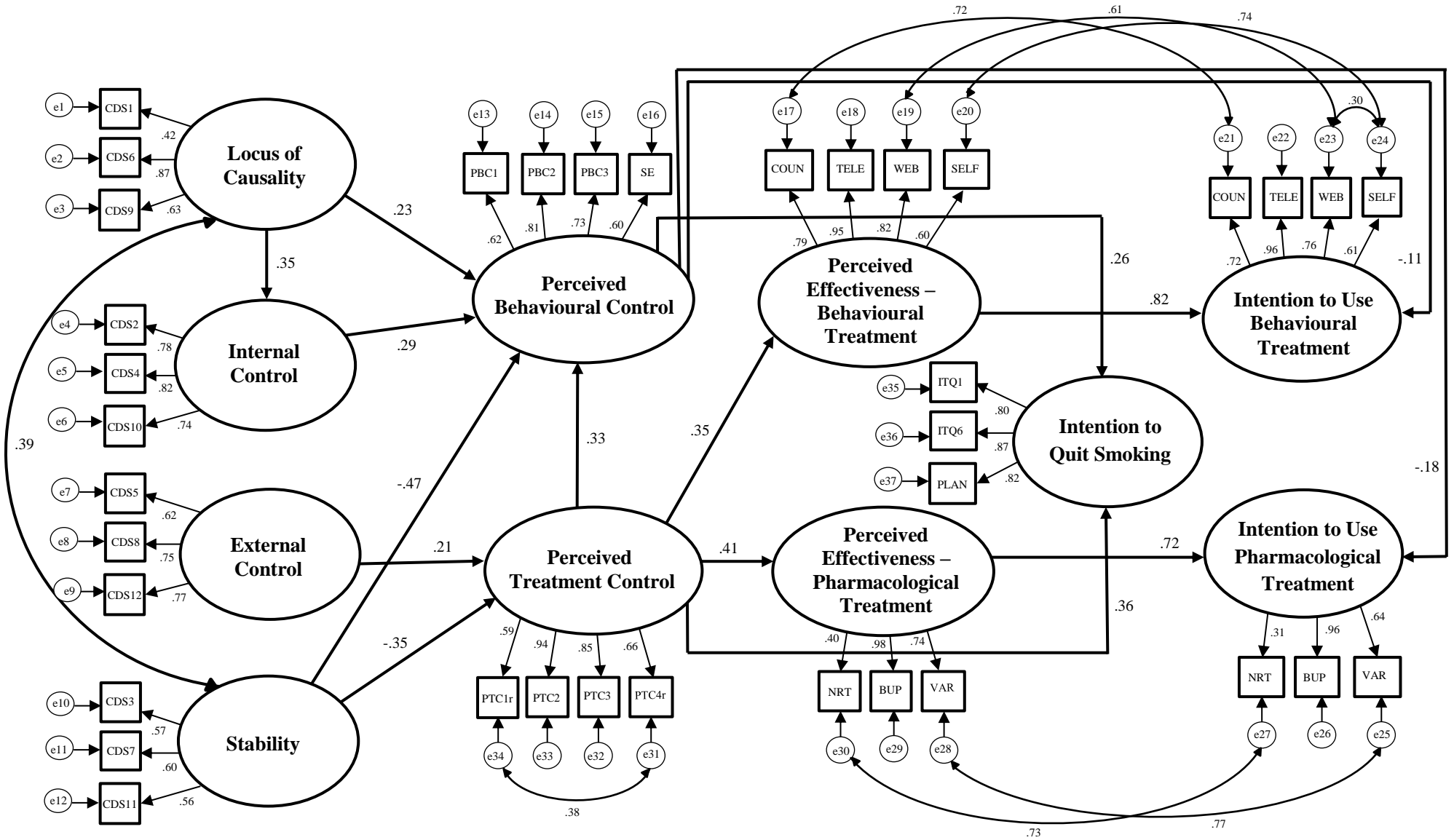
Figure 5. Final causal dimension model. Standardized robust maximum likelihood (MLR) parameter estimates are presented. All parameter estimates are significant at *p* < .05.

Table 14

Summary of Hypothesis Outcomes

Hypothesis number	Description	Outcome
1	Causal types will be associated with perceived behavioural control.	Not supported
2	Causal types will be associated with perceived treatment control.	Not supported
3	Perceived treatment control will be positively associated with perceived effectiveness and intention to use pharmacological and psychosocial treatment. Associations with pharmacological treatment will be stronger compared to psychosocial treatment	Partially supported
4	Perceived behavioural control will be negatively associated with perceived effectiveness and intention to use pharmacological and psychosocial treatment.	Partially supported
5	Perceived behavioural control and perceived treatment control will both be positively associated with intention to quit smoking.	Supported
6	Perceived effectiveness of pharmacological treatment will be positively associated with intentions to use pharmacological treatment and perceived effectiveness of psychosocial treatment will be positively associated with intentions to use psychosocial treatment.	Supported
7	Intention to quit smoking will predict intention to use pharmacological and psychosocial treatments.	Not supported
8	Causal attribution factors will correlate (exploratory; no directional hypotheses).	Supported
9	Internal control will be positively associated with perceived behavioural control and negatively associated with perceived treatment control. External control will be negatively associated with perceived behavioural control and positively associated with perceived treatment control.	Partially supported
10	Stability will be negatively associated with both perceived behavioural control and perceived treatment control.	Supported

Hypothesis number	Description	Outcome
11	Locus of causality will be positively associated with internal control and negatively associated with external control, and will exert both a direct and indirect effect (via internal and external control) on perceived behavioural control and perceived treatment control.	Partially supported

CHAPTER V

DISCUSSION

With increasing knowledge of the impact that genetics have on nicotine dependence and the efficacy of smoking cessation treatments, the prospect of tailoring treatment based on a smoker's genetic profile grows. Individualized treatment has the potential to yield greater quit success for patients with fewer adverse effects from medication, but concern about unintended negative consequences have also arisen. Concerns include whether feedback from genetic testing will lead to decreased perceived control over health and health behaviour, changes in motivation to change behaviour, and changes in the perceived effectiveness and utilization of different types of treatment. One potential mechanism through which genetic feedback might influence health behaviours is via beliefs about causal attribution. Causal beliefs for a health condition have been found to differ based on the results of genetic testing (Marteau et al., 2004), and theory predicts that differences in causal beliefs will subsequently lead to changes in motivation and behaviour (Leventhal et al., 1980; Weiner, 1985). Prior to examining whether causal attributions are altered by genetic feedback and whether they function as an intermediary variable between genetic feedback and psychological or behavioural outcomes, a better understanding of the nature of causal attributions for current smoking and the association of these attributions with intentions to quit and to seek smoking cessation treatment is highly beneficial.

The purpose of the current study was to examine the types of causal attributions made by individuals about their daily smoking and whether these causal attributions are associated with intentions to quit smoking and to seek various types of smoking cessation

treatment. To examine these associations, two causal models were developed,

representing a series of hypotheses regarding how causal types and causal dimensions

influence intentions to quit smoking and to seek smoking cessation treatment via their

effects on a set of mediating variables. The study findings supported the hypothesis that

causal dimensions had an influence on perceived control and intentions to quit and to

seek smoking cessation treatment, in part through perceptions of treatment effectiveness.

However, results failed to find similar associations for types of causal attributions. In the

following sections I review major findings of the current study and discuss whether they

lend support to previous research. Further, limitations and strengths of the study are

discussed and implications of this study's findings are explored. Finally, avenues of

possible future research are outlined.

Causal Attributions for Current Smoking

Types of causal attributions. The current study used two different methods to

assess the specific types of causes to which participants attribute their smoking, a cued

attribution list (IPQ-R) and two open-ended questions that asked participants to list the

causes of their current smoking and to select the single most important cause. The

number of causes to which smoking was attributed was higher for responses on the cued

attribution list compared to the open-ended listing task. Stress, habit, and addiction were

among the most frequent causes endorsed or listed on both measures. While participants

were frequently in agreement with psychological and emotional causes on the IPQ-R,

these causes were mentioned considerably less frequently in open-ended responses.

Open-ended questions encourage participants to report what can be easily recalled or

what comes to mind first, and thus researchers are thought to have confidence that these

issues are salient or important to respondents (Aday & Cornelius, 2006). In fact, average IPQ-R ratings for psychological and emotional causes were lower than causes mentioned more frequently in open-ended responses. Thus, perhaps psychological and emotional causes were applicable, but because they were not given as much importance, a prompt was required to enhance cognitive accessibility. It is also possible that due to stigma or social acceptability concerns surrounding these causes, respondents may have been hesitant to state these reasons in open-ended responses but were more comfortable agreeing on a rating scale.

Social influences were frequently mentioned in the open-ended listing task, though the context in which they were mentioned frequently suggested that participants were referring to social factors as a cause for initial uptake of smoking rather than continued smoking (e.g., "started long time ago as a casual smoker with friends just to join the fun and got stuck with the habit"). As well, social factors were often (but not always) discussed using the past tense and other causes with the present tense.

As so few participants mentioned genetics in open-ended responses, it is evident that genetics were not viewed by many as a very important influence on smoking. Furthermore, only approximately half the sample believed that genes exist that can increase one's chance to become addicted to smoking, and only a third believed a genetic predisposition to nicotine dependence could be passed on to a child even if the parent did not smoke. Thus, it appears that knowledge of genetic influence on nicotine dependence is not widespread. Even with this knowledge, it is unclear how many smokers would consider this information personally relevant without genetic testing. These findings are consistent with past research. Wright et al. (2007) found a mean rating of 2.6 on a genetic

attributions scale that ranged from 1 to 7, with higher scores indicating that genetics were an important cause of smoking; 35% of participants rated genetics as "not at all important". In addition, Quaak et al. (2012) found that only one-third of their sample of Dutch smokers believed that genes exist that can increase the chance to become addicted to smoking. Thus, it appears that many smokers in general are not aware that genetics have an influence on level of addiction to nicotine, and do not give much importance to genetics as a causal influence on their smoking.

The results of the current study indicate that a strictly biological view of smoking is not predominant among smokers in Ontario. In fact, both habit and stress were endorsed as frequently if not more (depending on the question format) than addiction as a cause of smoking, and other biological causes such as genetics and brain chemistry were among the least frequently endorsed or listed causes. Addiction has been referred to as a "brain disease", whereby "artificial substances purportedly 'flip a switch' in the brain such that, once activated, drug use moves from being voluntary to involuntary… especially as continued heavy use changes an individual's brain structure and function" (Dingel, Karkazis, & Koenig, 2011, p. 6). However, considerably fewer participants endorsed brain chemistry as a cause of their smoking compared to addiction. This suggests that many smokers may not equate addiction as a problem with brain functioning, and lay definitions of addiction may differ from that of the scientific and medical fields.

Some concern has been raised that an increasing focus on genetics in addictions research will lead to a narrowing view of addiction's etiology as entirely biological, and that this may create unrealistic expectations for treatment and an increased emphasis on

pharmaceuticals and decreased emphasis on modification of behaviours and environments (Dingel et al., 2011). A shift in research focus may impact the causal beliefs of researchers and health care professionals to whom these findings are disseminated, and in turn this may be communicated to smokers in various ways. The current findings suggest that this has not occurred in Ontario, though it does not exclude the possibility that this may occur in the future. It may be informative to continue to track whether causal beliefs among smokers and health care professionals change over time.

Associations between types of causes and causal dimensions. With the exception of addiction and alcohol/drug use, mention of a specific type of cause in open-ended responses was not associated with a significant difference on causal dimension scores. Listing alcohol or drug use as a causal influence on smoking was associated with higher ratings of internal control and lower ratings of external control over causes of smoking. Alcohol/drug use was mentioned very infrequently and appeared to refer to increased smoking while socializing (drinking with friends, drinking at a bar), and based on causal dimension scores does not appear to be the result of external pressure to smoke. While not significant, there was a trend for those who mentioned enjoyment/pleasure to rate themselves as having more internal control over the causes of their smoking, and for these causes to have a more internal locus. It is not surprising that enjoyment as a cause of smoking was associated with increased internality and personal control, as it signifies that smoking was driven by personal choice.

Those who listed addiction as a cause of their smoking rated level of internal control over causes significantly lower, and there was a tendency to rate causes as more stable as well. A similar association was reported by Eiser and colleagues (Eiser et al.,

1985), who found that the stability of other smokers' failed attempts to quit was associated with one's own perceived addiction to smoking, though controllability was not measured. According to the model tested and supported here, lower levels of internal control and higher levels of stability are associated with lower perceived behavioural control and lower perceived treatment control, which in turn predict decreased intention to quit and to seek treatment. Thus, we might expect individuals who attribute smoking to addiction to have decreased motivation to quit smoking or to seek assistance with quitting. Increasingly, views that smoking is a personal habit have largely been replaced, particularly in clinical settings, with the view that smoking is driven by an addiction to nicotine. Thus, it is not surprising that addiction was one of the most frequently mentioned causes in the current study. For these reasons, it is of concern that there may be negative unintended consequences to attributing smoking to addiction. However, the differences on causal dimension scores were quite small (less than 1-point on a 9-point scale) and are unlikely to translate into clinically meaningful differences on behavioural outcomes. A similar pattern of findings was observed for level of nicotine dependence as measured by the Heaviness of Smoking Index (HSI). HSI score was positively correlated with stability and negatively correlated with internal control, though the size of the correlation was small.

Overall, there was little correspondence between causes listed in open-ended responses and causal dimension scores. Furthermore, few significant correlations were observed between scores on causal type factors and causal dimension factors, and any statistically significant correlations were weak. Thus, causal dimension scores did not correspond highly with other measures of causal attributions. While no hypotheses were

formulated about associations between causal type and causal dimension scores, and little research has directly examined these links, it is somewhat surprising that additional significant associations did not emerge, given that one might anticipate some degree of shared understanding regarding the locus, controllability and stability of factors most frequently named as causes of smoking. However, the lack of association between causal dimensions and causal types in the current study may be partly or even largely accounted for by the fact that participants were asked to provide dimension ratings taking into consideration *all* causes of smoking together. As such, it is reasonable to expect some difference between these two measures. Furthermore, participants provided ratings on each dimension prior to being presented with the list of individual causes. Reviewing the list may have increased the cognitive accessibility of factors that were not initially reported, but later deemed important or applicable. As a result, the dimension ratings may have been made with reference to a somewhat different set of causes than causal type ratings. Future research should examine whether presentation of the IPQ-R before the CDSII yields different findings. Future research should also examine whether there is greater correspondence with dimension ratings for the single most important cause, and whether such ratings are of similar predictive value compared to dimension ratings for all causes combined.

Though little research has examined and compared ratings on individual causes with ratings on causal dimensions (Hall et al., 2003), a lack of concordance between these two methods of assessing attributions has been reported previously. As discussed earlier, Krantz and Rude (1984) found that when undergraduate students were asked to imagine that they had received a low exam grade, there was poor convergence between

ratings of the influence of specific causes (effort, ability, task difficulty, bad luck) and

causal dimensions (stability, locus, globality). Furthermore, participants' dimension

ratings did not always agree with theoretical predictions, highlighting the importance of

asking participants directly to rate causes on underlying dimensions versus using judges

to assign dimension scores based on open-ended responses. However, in contrast with

findings here, ratings on both individual causes and causal dimensions made a unique

contribution to the prediction of depression scores and the optimal prediction of

depression included both causes and dimensions (Krantz & Rude, 1984). The lack of

concordance between the two types of measures here and in previous research, and the

finding that at least in some cases both have unique predictive value, points to the

importance of assessing attributions using both ratings for individual causes and

underlying dimensions in attributional research.

Associations Between Causal Attributions and Perceived Control, Perceived Treatment

Effectiveness, and Intentions to Quit Smoking and Seek Smoking Cessation Treatment

   Causal types. Contrary to hypotheses, type of causal attribution (biological,

psychological, social, and stress) was not a significant predictor of perceived behavioural

control or perceived treatment control in the causal model tested. While causal types

exhibited weak correlations with perceived effectiveness and intentions to use

psychosocial treatments, the sign and strength of each correlation was similar across each

type of attribution, suggesting the particular type of attribution was not predictive but

simply endorsing any type of attribution was. Types of attribution exhibited very minimal

or non-significant correlations with other variables in the model, as a result, no

alternative hypotheses or models were formulated or tested.

Several possibilities are considered here for why causal types exhibited non-significant or weak associations with other study variables. Firstly, many participants were in agreement with several causes that represented different causal types. This finding may be in part owing to the type of methodology used to assess type of attribution. The findings in the current study replicate the findings of Gudmundsdóttir et al. (2001), who found that types of attributions made were very similar across different methods of assessing causal attributions and the most commonly mentioned causes were the same, but a greater number of causes were endorsed when cued attribution lists were used than when they were asked openly. Reasons for increased endorsement of causes may include acquiescence bias, the tendency to agree with questionnaire items (Aday & Cornelius, 2006). As discussed previously, causes endorsed on rating scales may not have been as cognitively accessible during response to open-ended questions because they are not as important or salient a cause for that individual, and this may account for weak associations with outcomes. If ratings were inflated for any of the above reasons, this would have decreased the power to detect any true effects.

However, even in open-ended responses, many participants listed more than one cause, and often these causes represented different causal types. Thus, it is evident that many participants viewed their smoking as being influenced by multiple types of causes. In fact, there were relatively high inter-correlations between psychological, stress, and social causal type scores. As a result, any one causal type may not have had as much predictive power in statistical analysis, in that it could not account for as much unique variance. An additional methodological concern when using factor analysis with questionnaire measures of causal attributions is common method variance (French et al.,

2005), the "variance that is attributable to the measurement method rather than to the constructs the measures represent" (Podsakoff, MacKenzie, Lee & Podsakoff, 2003, p. 879). In the absence of alternative measurement techniques to address this psychometric issue, French et al. suggested avoiding principal components analysis to extract factors, and this suggestion was observed in the current study.

It is of interest to compare the current findings with that of Hilbert et al. (2009), who used a similar methodology as used here, having adapted the Causes subscale of the IPQ-R for obesity. Though not equivalent to nicotine dependence, many comparisons have been made between excessive or compulsive food intake (or 'food addiction') and drug addiction (DiLeone, Taylor, & Picciotto, 2012; Ziauddeen & Fletcher, 2013). Hilbert et al. asked participants to rate a total of 15 potential causes of obesity, which were analyzed using principal components analysis with orthogonal rotation and five factors were extracted (Genetic/Biological, Behavioural, Psychological, Lack of Knowledge, and Other). While Genetic/Biological, Lack of Knowledge, and Other attributions were unrelated to self-efficacy, perceived control over weight, restrained/restrictive eating behaviour, and physical activity, as well as change in BMI at 6-month follow-up, Behavioural and Psychological Attributions were both associated with most indicators of weight regulation beliefs and behaviour in both cross-sectional and longitudinal analyses. While Hilbert and colleagues found that Genetic/Biological and Behavioural Attributions were endorsed by the majority of the sample (77% and 64% respectively), the proportion who endorsed the other types of attributions were much lower, ranging from 20% to 31%. Thus, it appears that attributions for obesity were less complex and multi-determined than those reported for smoking here, possibly accounting

for why attribution type factors were more predictive of related beliefs and behaviour for obesity in this previous study than for smoking in the current study.

Weiner (1985) points out that the same cause mentioned by two different people may have very different meanings. One way this would be apparent would be different ratings on underlying causal dimensions for the same cause. For example, for an individual who suffers from persistent and/or severe depressive episodes 'depressed mood' may be seen as a stable cause of their smoking with biological underpinnings over which they have little control. Another individual however may view depressed mood as transient and under their control. Thus, we may see very different outcomes for two individuals who endorse the same cause. If the same cause has a different underlying meaning across participants, different outcomes could follow for individuals who endorse the same cause, diminishing the predictive ability of ratings on that variable. Furthermore, this may also account for why few associations were identified between causal type and causal dimension scores in the current study.

An important tenet of Weiner's theory is that the specific attribution made (e.g., genetics) is less influential than the causal dimensions underlying that attribution. The findings of the current study are consistent with this theory. Thus, it is also possible that the lack of association between type of causal attribution and other study variables was accurate and in line with Weiner's theory and not due to methodological shortcomings. The lack of prior investigation of causal attributions for current smoking does not allow for considerable comparison with past findings. However, Wright et al. (2007) found that attributions to genetic causes were associated with perceived control over smoking when ratings were dichotomized so those who believed that genetics were not at all an

important cause of smoking were compared to those who believed it was possibly important. Thus it appears that type of attribution may be significant under some circumstances. Further research is necessary to address above-mentioned methodological concerns prior to concluding that type of attribution for current smoking, particularly genetics, is not associated with levels of perceived control or other motivational and behavioural outcomes.

   Causal dimensions. As hypothesized, causal dimensions (internal control, external control, stability, and locus of causality) did significantly predict levels of perceived behavioural control and perceived treatment control, and with a few modifications the hypothesized model was a good fit to the data. Based on the pattern of associations in the model, we can expect that individuals who attribute their smoking to causes they rate high on internal control are expected to have increased perceptions of personal control over smoking and stronger plans to quit, but weaker intentions to use various smoking cessation treatments to assist with quitting. Individuals who attribute their smoking to causes they rate high on external control are also expected to have stronger plans to quit smoking, but via a stronger belief that treatment is able to control smoking. As a result of this increased perceived treatment control, these same individuals are more likely to believe that pharmacological and psychosocial smoking cessation treatments are effective and intend to use them. Those who believe that their smoking is caused by stable factors are less likely to believe that they have control over their smoking, whether personally or via treatment seeking, and as such are less likely to intend to quit or to use smoking cessation treatment. Finally, judging the locus of causes

as internal was associated with perceived behavioural control and thus may be associated with greater intention to quit without assistance.

Several hypothesized paths in the model were not significant and deleted from the final model. First, both internal control and external control were positively associated with only one type of perceived control, but not negatively associated with the other type of perceived control as hypothesized. This was consistent with the finding that the two types of perceived control were not negatively associated; thus, a high score on one type of perceived control was not associated with a low score on the other type of perceived control. Second, while locus of causality (internal) was positively associated with internal control, it was not negatively associated with external control as hypothesized. Thus, while internal locus is associated with increased control by oneself, external causes are not always perceived to be controllable by other people.

Third, perceived behavioural control was not associated with perceived effectiveness of treatment, although it was associated with intentions to use treatment. Thus, perceived behavioural control appears to modify intentions to use treatment for reasons other than beliefs about treatment effectiveness. Individuals who feel a sense of personal control over their smoking may simply not feel the need to use any form of assistance to quit, and perhaps also anticipate a greater sense of accomplishment if they achieve abstinence on their own. Fourth, perceived treatment control was not directly associated with intention to use treatment, instead there was an indirect association via perceived effectiveness. This suggests that perceived effectiveness fully mediates this relationship.

Additionally, intention to quit was not a predictor of intention to use treatment. Thus, the finding of Vogt et al. (2008, 2010) that intention to stop smoking was a significant, but weak, predictor of intentions to use behavioural support and cessation medication (except NRT) was not replicated in the current study. While intention to quit may be considered a necessary prior condition for using treatment, it is not sufficient. That is, while individuals intend to use treatment in order to quit smoking, some individuals who intend to quit do not intend to use treatment. In fact, those with high levels of perceived behavioural control who intend to quit are less likely to intend to use treatment. This may account for the absence of a significant association between intention to quit and intention to use treatment in the current study. Vogt et al. did not assess perceived treatment control, which was indirectly associated with both quit intentions and treatment intentions in the current study. Thus, it is plausible that the association between intention to quit and intention to seek treatment found by Vogt et al. was owing to their shared association with perceived treatment control; that is, the finding may have been spurious.

Finally, after modification of the model, the path from locus of causality to perceived treatment control was no longer significant and was deleted. While it was thought that internal causes would be more amenable to treatment (compared to external causes), this hypothesis was not supported. Thus, causes external to the self are considered no more or less amenable to treatment, presuming they can be controlled.

It was also hypothesized that perceived treatment control would be more strongly associated with perceived effectiveness and intentions to use pharmacological treatment versus psychosocial treatment, given the greater behavioural demands of counselling and

self-help treatments. While the size of the standardized path coefficients did support this, the difference was minimal. Thus it appeared that where causal beliefs were associated with increased perceived treatment control, this was associated with treatment seeking in general, rather than a strong preference for one type of treatment in particular.

The unique pathways from internal control and external control appear consistent with the hypothesis of Marteau et al. (2004) that overall level of perceived control over a health threat is not altered by genetic risk information, but rather views regarding how this control can be achieved does shift, presuming that genetic risk information alters causal beliefs. Thus, if genetic risk information alters causal beliefs such that causes of a health threat are consequently rated higher on external control, we can expect from findings here a change in how control over that threat is achieved (via treatment versus personal behaviour alone) and subsequent treatment seeking behaviour would differ as a result. In contrast, a decrease in overall perceived control would occur if genetic risk information prompted lower ratings on both internal and external control, and/or high ratings on stability. However, a longitudinal or experimental study is required to adequately test this hypothesis and determine if causal beliefs do change following genetic risk feedback.

If genetic risk information were to shift perceptions of control toward treatment, based on the current findings we would not expect a strong preference for biological based treatment among smokers. Yet, past studies have reported a preference for biologically based treatments after individuals are provided with real or hypothetical positive genetic test results (indicating greater risk). For example, Wright et al. (2003) found individuals were more likely to report that they would use Zyban if they received a

positive genetic test result (hypothetically) and would be less likely to quit unassisted (rely on willpower). No significant differences were found for likelihood of using other types of treatment, including counselling and nicotine replacement therapy (NRT). Being less likely to quit unassisted would be consistent with a decrease in perceived behavioural control. While participants did report an increased likelihood of using Zyban, they did not report an increased likelihood of using NRT, another form of pharmacological aid. Thus the findings do not point to a generalized preference for biological based treatment. That the treatment preference was specific to Zyban may have followed from the wording of the vignettes, as they clearly linked genetic susceptibility to low levels of dopamine ("This gene is found in people with low levels of dopamine") and described Zyban as "a drug that reduces cravings for nicotine by increasing dopamine levels". Similarly, Marteau et al. (2004) found that individuals whose familial hypercholesterolemia was confirmed by genetic testing believed more strongly that their cholesterol levels were determined by genetics, believed less strongly in the efficacy of lowering dietary fat intake and believed somewhat more strongly in the efficacy of medication to reduce cholesterol level. Because lowering dietary fat intake relies largely on the individual exerting control over their own behaviour, it is likely to be associated with perceived behavioural control more so than perceived treatment control. Therefore, findings in these previous studies may suggest a preference for treatment due to increased perceived treatment control and a decreased reliance on unassisted behaviour change due to lower levels of perceived behavioural control. Clearly, more research is needed to elucidate the impact that genetic testing and perceived treatment control may have on beliefs and preferences for various types of treatment and behavioural strategies to manage health

threats. Thus, it remains to be seen if genetic risk information enhances preference for biologically based treatment, or treatment in general.

If genetic testing and genetic causal attributions do in fact enhance perceptions of treatment control, then the current study would suggest that these attributions are higher on external control, as it was the only dimension positively associated with perceived treatment control. In fact, in the current study there was a positive association between biological attributions and external control scores, and a negative correlation between biological attributions and internal control scores. Though statistically significant, the strength of these correlations was weak. Additionally though, biological attributions were positively correlated with stability, which was negatively associated with perceived treatment control. Unfortunately, too few participants listed genetics in open-ended responses to permit a comparison of causal dimension scores. Future research would benefit from asking participants directly about the underlying causal dimensions for genetic attributions specifically, in order to determine where individuals place them on each dimension, and whether their placement is consistent across individuals.

An unexpected finding in the current study was the addition of the path from perceived treatment control to perceived behavioural control. While this may seem somewhat contradictory given that the results reveal very different outcomes for perceived behavioural control and perceived treatment control on intentions to use treatment, the perceived behavioural control items do not exclude the possibility that individuals can personally exert control over their smoking by choosing to seek treatment. For example, "My actions will have no effect on whether I quit smoking" and "I am able to quit smoking" are two perceived behavioural control items, and individuals

who believe that treatment can control their smoking will likely rate higher on these items than individuals who do not have a sense that either their personal actions or treatment can control their smoking. On the other hand, there are some perceived behavioural control items that should not correlate as highly with perceived treatment control, such as "Whether or not I smoke depends entirely on me". Individuals who feel that counselling or medication is necessary to quit may not rate this item as highly. Revision of perceived behavioural control items is advisable in future research to make it more distinct from perceived treatment control. For example, "I am able to quit smoking" may be revised to read "I am able to quit smoking on my own without assistance".

Overall, findings in the current study are consistent with and extend various past research findings. Firstly, as predicted, several findings were consistent with those of Vogt et al. (2008, 2010), who had examined predictors of intentions to use various smoking cessation treatments among smokers in England. Vogt and colleagues found that perceived effectiveness of both pharmacological and nonpharmacological smoking cessation treatments strongly predicted intentions to use these same types of treatment. Additionally, they found that smokers high in self-efficacy to stop smoking, part of the perceived behavioural control construct in the current study, had lower intentions to use both pharmacological and nonpharmacological treatments.

The current study also suggests that one antecedent of these perceptions of treatment effectiveness is perceived treatment control, which in is part determined by causal attributions. This is consistent with certain findings by Wright et al. (2012), who examined mediators of the impact of genetic causal information on perceived treatment effectiveness using hypothetical vignettes about heart disease, depression, and obesity.

While findings differed according to type of health condition and severity of the condition, findings indicated for heart disease that attributions and perceived control were mediators between the presentation of genetic causal information and perceived effectiveness of treatment. For severe depression, only perceived control was a mediator. The current study extends some of these findings by demonstrating that among smokers, perceived treatment control was not only a predictor of perceived treatment effectiveness, but it mediated the association between causal attributions and perceived treatment effectiveness. However, the current study did not find an association between perceived behavioural control and ratings of perceived treatment effectiveness. Thus, the current study points to the importance of differentiating between perceived behavioural control and perceived treatment control, as only perceived treatment control mediated the association between attributions and perceived treatment effectiveness.

It is unknown why attributions for specific types of causes (e.g., genetics, diet) were significant predictors of perceived treatment effectiveness for select health conditions in the Wright et al. (2012) study, but not for all health conditions and not for smoking in the current study. Possible reasons include the nature of the particular health condition being studied or methodological factors. For example, Wright et al. raise possible issues with order effects with presentation of each vignette. In comparing the Wright et al. study with the current study, there are some differences worthy of consideration. For example, in order to measure strength of causal attributions, Wright et al. asked participants (after reading the hypothetical vignette) "How important do you think each of the following might have been in causing Sam's problem?", after which causes such as "genes" and "exercise" were rated on a 7-point scale. In the current study,

the corresponding question asked, "Please indicate how strongly you agree or disagree

that the following are causes of your current smoking", and each cause was rated on a 5-

point scale. It is possible that asking how important a cause is may be more predictive of

outcomes versus simply asking if a cause is applicable, particularly with conditions for

which many different possible causes may apply. This may also be accomplished by

asking participants to rank how important each cause is, and the rankings can be

analyzed. While the current study permitted participants to provide identical ratings for

multiple causes, ranking would require that participants prioritize causes. In addition, a 7-

point scale may allow for greater precision than a 5-point scale, which may be

particularly beneficial when there is agreement on several different causes. While Wright

et al. asked respondents to provide attributions for a hypothetical other person, the current

study asked for attributions for the self. This may have an impact on the types of

attributions participants report, as research has shown that people tend to make different

attributions depending on whether they are the actor or observer (Jones & Nisbett, 1972).

Such variations in the wording of items to measure attributions should be investigated in

future research. However, as already mentioned, the difference may lie simply in the

nature of causal beliefs for the health behaviour or condition being studied and may not

be related to methodological differences.

The association found between perceived treatment control and intentions to use

treatment, though indirect and mediated via perceived treatment effectiveness, is

consistent with previous research. For example, Bradley et al. (1987) found that levels of

perceived medical control predicted the selection of insulin pump over conventional

injection regimens among individuals with type 1 diabetes. A positive association has

also been documented in the past between perceived treatment control and seeking

mental health services (Vanheusden et al., 2009). Again, the current study extends these

findings by identifying perceived effectiveness of treatment as a mediator of this

association.

Overall, the current findings were consistent with Weiner's attribution theory.

According to Weiner (1985), causal attributions have a direct impact on expectancy of

successful goal attainment. In the current study, perceived control and perceived

effectiveness reflect expectancies that behaviour will influence the outcome of interest

(quitting smoking). In turn, expectancy is expected to have a direct role in determining

whether efforts are made to manage or eliminate a health threat or condition, and if so,

which types of behaviour, or coping methods, are utilized. In particular, Weiner (1985)

believed that expectancy of success in the future was based on the perceived degree of

stability of the cause, whereas the other causal dimensions were purported to impact

affect, which in turn would also play a role in determining behaviour along with outcome

expectancies. However, the current study found that all three causal dimensions were

associated with expectancy of success, though stability was the strongest predictor of

perceived behavioural control and perceived treatment control compared to the other

causal dimensions. Furthermore, outcome expectancies predicted whether participants

intended to manage or eliminate the health threat (quit smoking) and what type of

behaviour would be utilized to do so (via treatment seeking or unassisted). Affect was not

assessed in the current study but likely plays a role in predicting outcomes as well.

While actual behaviour performance was not measured in the current study, a

previous prospective study by Eiser and colleagues (Eiser et al., 1985; Eiser & van der

Pligt, 1986) found that greater perceived stability of the cause of other smokers' failed

quit attempts was negatively related to personal confidence in ability to quit smoking,

which in turn was associated with intentions to quit, and intention predicted actual

abstinence attempts made during follow-up. Locus of causality did not predict confidence

in ability to quit and controllability was not assessed. Thus, the findings are both

consistent with what the current study found with respect to the stability dimension, and

support the prediction that causal attributions and intentions to quit will be associated

with actual future quit attempts.

Limitations

Several limitations of the current study should be taken into consideration prior to

making any conclusions. First of all, while the data was consistent with the hypothesized

causal relationships, the study design was both correlational and cross-sectional and

therefore causal effects cannot be inferred from the findings. Future longitudinal or

experimental research is necessary to demonstrate causation.

While the sample was diverse on most demographic and smoking characteristics,

the majority of the sample was Caucasian and classified as having a moderate level of

nicotine dependence. Therefore, the findings may not generalize to other ethnic groups or

those with higher or lower levels of nicotine dependence, particularly a high level of

nicotine dependence, as only a small percentage of the sample fell into this category.

However, the proportion of participants classified as having a low, moderate, or high

level of nicotine dependence according to the Heaviness of Smoking Index was similar to

the proportion found in the general population of smokers in Ontario (Health Canada,

2010).

Another potential threat to the generalizability of the findings is the fact that participants were recruited and data was collected exclusively online. While recent statistics show that 81% of households in Ontario have access to the Internet (Statistics Canada, 2011), access varies according to income and only 54% of households in the lowest income group ($30,000 or less) have home Internet access. As such, the current study may under-represent lower socioeconomic groups. This is of particular importance given the finding that lower socioeconomic status groups have higher rates of tobacco use (Health Canada, 2012; Jarvis & Wardle, 2006) and are less likely to intend to quit, make a quit attempt, or achieve abstinence from smoking (Reid, Hammond, Boudreau, Fong, & Siahpush, 2010). While the current study did not collect data on income, level of education, one indicator of socioeconomic status, was not skewed toward the more highly educated; the proportion of participants who reported a high school diploma as their highest level of education was slightly higher than found among the general population aged 25-64 in Canada, and the proportion who reported a university degree was slightly lower (Statistics Canada, 2013). Future research in this area should incorporate the use of other data collection methods to ensure that findings are replicated across diverse and representative samples.

The internal consistency of the measures of biological attributions, stability, locus of causality, and level of nicotine dependence were below what is conventionally considered acceptable (Nunnally, 1978). Thus, findings pertaining to these variables should be interpreted with caution. However, each of these measures was comprised of only two to three items, and given that Cronbach's alpha increases with the number of items in a scale, the reliability of these measures may be underestimated due to their

brevity. Further, as noted earlier, Kline (2011) states that somewhat lower levels of score reliability can be tolerated in latent variables methods compared with observed variable methods.

An additional potential issue with measurement was the lack of clarity between attributions made for smoking in the past and attributions for smoking in the present. Even though instructions asked for responses to be made for current smoking, in open-ended questions it appeared that attributions to social factors were primarily referring to initial smoking uptake, but may not have had a similar (if any) causal influence on continued smoking. It is unknown whether this occurred with other causes as well. Any lack of differentiation between past and current reasons for smoking when responding may have diminished the predictive ability of these measures, as each may have a distinct (and dissimilar) relationship with outcomes. Some modification of the phrasing may enhance the clarity of these instructions, such as asking individuals to only list or endorse factors that cause them to *continue* to smoke or reasons why they have not yet stopped smoking, but not causes for initiation of smoking. Alternatively, it may be helpful in future research to devise separate questions about attributions for initiation of smoking versus continued smoking. This second option would permit a comparison of these two sets of attributions, including their associations with other variables of interest. For example, genetics may be viewed as more relevant for initiation versus continuation of smoking, or vice versa, and whether it is endorsed at either time point may have different implications for future plans to quit and to seek treatment.

Additionally, the list of putative causes in the IPQ-R Causes subscale in the current study may require revision as well. Though responses to the 'other' option in the

list of causes revealed that almost all causes were covered by the list, as few respondents listed additional causes, some items may have lacked conceptual clarity and this may account for weak associations with other study variables; however, this is speculative. A qualitative analysis of causal beliefs among individuals who smoke may indicate whether changes are necessary, as well as provide the basis for such revisions, to ensure that the causes listed are both clearly defined and pertinent.

Finally, the potential mediating or moderating effects of demographic or smoking characteristics such as level of education, level of nicotine dependence, and previous number of quit attempts were not tested in the interest of scientific parsimony and due to concern over lack of power to test more complex models. Variables were selected with a focus on examining the hypothesized causal pathways through which causal attributions may exert an influence on behavioural intentions. Larger sample sizes in future research may afford the power necessary to examine more complex models.

Implications

The results of the current study have practical implications for clinical and public health settings. Many participants in the current study identified both physiological addiction to nicotine along with other psychological and social factors as causes underlying their smoking. This is consistent with tobacco control research, which has identified that while cigarette smoking is primarily a manifestation of nicotine addiction, pharmacological factors are not the sole determinants of smoking behaviour (Jarvis, 2004). This further confirms the need for interventions to address both psychosocial and pharmacological factors in order to most effectively help individuals achieve and maintain abstinence (Jarvis, 2004).

Despite the existence of effective interventions, the current study reiterates earlier findings that currently available smoking cessation interventions are not considered effective by a majority of smokers (Hammond et al., 2004). Given that perceived treatment effectiveness was a strong predictor of treatment intentions here and in previous research (Vogt et al., 2008, 2010), and has also predicted subsequent use of cessation assistance during follow-up (Hammond et al., 2004), efforts to improve quit rates by increasing treatment seeking among smokers should target beliefs about treatment effectiveness. One such strategy could be to provide evidence regarding the effectiveness of various treatments to smokers. A survey of high school students who smoked found that approximately half said they would be persuaded to use self-help or group stop-smoking interventions if provided with proof of effectiveness (Lawrance, 2001). While a survey of smokers in Ontario found that the vast majority wanted additional information on where to get help to quit smoking, many were not aware of the options available to them (Hammond et al., 2004). Thus, greater dissemination of information about the types of support available to help quit smoking, in addition to evidence of their effectiveness, are recommended strategies to increase treatment seeking among smokers.

Additionally, the current study established that causal beliefs are indirectly associated with perceived treatment effectiveness and treatment seeking intentions among individuals who smoke, as well as strength of intentions to quit. As such, efforts to enhance intentions to quit smoking and to seek treatment might include attention to what type of causal information is presented to smokers. For example, many smoking cessation interventions make some reference to causation when encouraging smokers to identify

their smoking triggers, or "things that can cause you to want a cigarette" (Canadian Cancer Society, 2013). Previous research has demonstrated that both method of treatment presentation and the presence or absence of different treatment components can influence the attributions made by smokers (Harackiewicz et al., l987). More intensive counselling presents a greater opportunity to assess and discuss causal attributions and to reframe them so that they are more adaptive. Such *attributional retraining* methods attempt to alter attributional thoughts so that they are more adaptive, and have been consistently successful in academic settings, resulting in modest increases in motivation and performance (for a review see Försterling, 1985). Though attributional retraining has not been sufficiently examined in a clinical context, it is compatible with cognitive therapy, as both share an underlying assumption that maladaptive behavioural and emotional outcomes can be modified by changing preceding cognitions (Försterling, 1985). Furthermore, restructuring of maladaptive attributions has already been incorporated into interventions for smoking relapse prevention (Marlatt & Gordon, 1985).

The current findings suggest that messages are best framed to highlight how smokers might exert some degree of control over causal influences, as well as their variability or changeability. Given that genetic makeup is fixed, dissemination of information about links between genetics and nicotine dependence may lead to greater perceived stability of the causes of smoking if it prompts an individual to form an attribution to genetics. In turn, the current study suggests that increased stability will be associated with less perceived control over smoking and weaker intentions to quit or to seek smoking cessation treatment. While genetics are fixed, if smokers are provided with information about how genetics exert an impact on nicotine dependence via alterations in

brain chemistry, and how these processes in turn can be modified with treatment such as medication, this may diminish the perceived stability of genetics or counteract any negative impact on stability by simultaneously enhancing perceived control.

In addition, the interaction between genetics and environment in determining behaviour can also be addressed. According to Dar-Nimrod and Heine (2011), when people encounter information that genetics are relevant for a behaviour or condition, a cognitive bias that they refer to as *genetic essentialism* is elicited that leads to a particular set of thoughts about those behaviours or conditions. Among these is the belief that genetically influenced outcomes are immutable (stable) and determined, that is, if the gene is present the outcome is expected to occur and to be independent of any environmental influence or personal control. They contend that reinforcing these beliefs are oversimplified reporting of genetics-related research findings in the media, as well as the fact that education about genetics in middle/high school is also oversimplified (e.g., Mendel's pea experiments). Educational efforts can help individuals better appreciate the complexity of how behaviours and conditions are influenced by genetics, including how genetics interact with environmental factors, which may help diminish genetic determinist or essentialist beliefs (Dar-Nimrod & Heine, 2011). Thus, if evidence of genetic contributions to nicotine dependence are to be communicated to smokers, such as during genetic testing, it would be beneficial to incorporate an educational component to improve genetics knowledge. Appreciating the interaction between genetics and environmental factors may encourage smokers to form attributions to factors other than genetics, which may influence the overall perceived stability and controllability of underlying causes.

Future Directions for Research

The current study was unique in that it conducted a comprehensive examination of causal attributions for daily smoking. As such, these findings need to be replicated in other samples. One task for future research is to examine possible methodological factors that may have led to non-significant findings prior to concluding that the types of causes to which smoking is attributed has no influence on smoking cessation behaviour. Suggestions reviewed earlier include asking participants to rate or rank how *important* each cause is, using a 7- or 9-point rating scale instead of a 5-point scale, and improving the clarity of instructions and items and possibly separately assessing attributions for initiation versus continued smoking. Future research should continue to assess and compare ratings for causes and dimensions, as research suggests that they may both play a unique role in determining outcomes (Krantz & Rude, 1984).

Further research is necessary before more conclusive statements can be made in response to questions raised about the impact of genetic testing and causal attributions on smoking behaviour. The current study suggests that future research may not reveal any significant associations between genetic attributions and outcomes without an examination of underlying causal dimensions. The influence of individual differences such as personality traits (e.g., optimism), attributional style, or affective state (e.g., depression) may play a role in why people rate the same cause differentially on Weiner's (1985) dimensions, and is also an area requiring further study. For example, individuals who are depressed are more likely to make attributions for negative events that are stable, internal, and global (e.g., Ball, McGuffin, & Farmer, 2008); they are also less likely to successfully quit smoking (Cinciripini et al., 2003).

As mentioned earlier, the current study design does not permit causal statements to be made about the relationships between variables, and study variables cannot truly be considered predictors and outcomes. Hence, longitudinal or experimental research is needed to examine whether a change in causal attributions predicts changes in motivational and behavioural variables. Until genetic testing is more accessible or attributions to genetics are more widespread among smokers, research using hypothetical vignettes may be most feasible, though external validity is weakened.

Additionally, further research is needed to track behavioural outcomes such as number of quit attempts, treatment seeking, and short- and long-term quit success. This will be necessary in order to establish whether the associations between attributions and intentions actually translate into differences in behaviour, and to determine whether these associations are clinically significant prior to investing resources to apply these findings to interventions.

Finally, one of the more novel aspects of the current study was the assessment of both perceived treatment control and perceived behavioural control. The current findings suggest that in order to motivate a quit attempt, perceived control does not necessarily need to take the form of a personal sense of control over behaviour, but that the perception that treatment can control smoking also motivates behaviour. However, further research is needed to determine whether each type of perceived control is equally as likely to translate into actual quit attempts and long-term quit success. Thus, future research would benefit from a greater examination of perceived treatment control, and the manner in which it is distinct from perceived behavioural control, in terms of both antecedents and sequelae.

Conclusion

To the extent that genetic testing feedback prompts a change in beliefs about the causation of a physical or behavioural outcome, an understanding of how causal attributions for smoking influence smoking behaviour and attempts to quit has implications for the process of providing genetic testing feedback to smokers in clinical settings. Furthermore, this knowledge can also be applied to understanding how predominant views and information communicated to smokers about the causal factors underlying smoking may have an impact on their motivation and success with quitting. The current study found that individuals who smoke daily in Ontario, Canada, most commonly attribute their smoking to habit, addiction, and stress, and often attribute their smoking to multiple different causes concurrently. Attributions to genetics were much less common, and knowledge that genetics play a role in determining level of addiction to nicotine was not pervasive. Results revealed that the causal dimensions underlying attributions for smoking were predictive of level of perceived control over smoking, which in turn predicted perceived effectiveness of cessation treatment, as well as intentions to quit and to seek treatment. Overall, the current study has made a contribution to this area of research by conducting a comprehensive assessment of causal attributions for current smoking, as well as developing and establishing support for a model of the pathways between causal attributions and intentions to quit smoking and to seek treatment for cessation. Current findings can be applied to future research on the effects of providing genetic testing feedback to smokers in clinical settings, and may have wider applicability to other health threats as well.

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Appendix A: **Glossary of Terms**

Behavioural intention:	an individual's plan or perceived likelihood of engaging in a given behaviour; assumed to be an immediate antecedent of behaviour and to indicate the amount of effort one is willing to exert to perform the behaviour.
Causal attribution:	an explanation made for the cause of an event, including our own or other people's behaviour.
Controllability:	the degree to which a cause is perceived to be under volitional control by the actor.
External control:	the degree to which a cause is perceived to be controllable by other people.
Internal control (personal control):	the degree to which a cause is perceived to be controllable by the self.
Locus of causality:	whether an individual perceives a cause to be internal or external; an internal locus of causality implies the behaviour was a result of volitional or dispositional factors originating from the actor while an external locus of causality implies that contextual factors compelled or constrained the actor to behave in a particular way.
Outcome expectation (outcome expectancy):	estimate that a given behaviour will bring about certain outcomes.
Perceived behavioural control:	an individual's subjective belief in the degree of control they have over performance of a behaviour and the perceived ease or difficulty of performing the behaviour.
Perceived treatment control:	the degree to which a condition is believed to be controllable by treatment.
Perceived treatment effectiveness:	an outcome expectancy regarding whether a particular treatment is able to manage or cure a condition.
Stability:	the perceived degree of permanence of a cause over time.

Appendix B: **Screening Question**

How often do you smoke cigarettes?

- ○ Every day
- ○ Occasionally
- ○ Not at all
- ○ Prefer not to answer

Appendix C: Notice of Ineligibility

Unfortunately, based on your initial response you do not meet criteria for participation in this study. If you chose not to answer the first question, your eligibility for the study could not be determined.

Thank you for your time and interest in this study.

Appendix D: Revised Causal Dimension Scale (CDSII)

People have different ideas about the reasons why they started smoking and the reasons why they smoke now. What do think are the causes or reasons for your smoking now?

Please keep in mind that we are interested in what YOU think causes YOUR smoking, not what other people such as doctors or family may have suggested to you or what you think causes other people to smoke.

The items below concern your impressions or opinions of the overall cause or causes of your smoking. Thinking about all of the reasons you listed for the previous question, please rate the items below.

If you prefer not to answer any question, please select 'N/A'.

Is the cause of your smoking something…		
1.That reflects an aspect of the situation	1 2 3 4 5 6 7 8 9 N/A	That reflects an aspect of yourself
2.Not manageable by you	1 2 3 4 5 6 7 8 9 N/A	Manageable by you
3.Temporary	1 2 3 4 5 6 7 8 9 N/A	Permanent
4.You cannot regulate	1 2 3 4 5 6 7 8 9 N/A	You can regulate
5.Over which others have no control	1 2 3 4 5 6 7 8 9 N/A	Over which others have control
6.Outside of you	1 2 3 4 5 6 7 8 9 N/A	Inside of you
7.Variable over time	1 2 3 4 5 6 7 8 9 N/A	Stable over time
8.Not under the power of other people	1 2 3 4 5 6 7 8 9 N/A	Under the power of other people
9.Something about others	1 2 3 4 5 6 7 8 9 N/A	Something about you
10.Over which you have no power	1 2 3 4 5 6 7 8 9 N/A	Over which you have power
11.Changeable	1 2 3 4 5 6 7 8 9 N/A	Unchangeable
12.Other people cannot regulate	1 2 3 4 5 6 7 8 9 N/A	Other people can regulate

Note. The total score for each dimension is obtained by summing the items as follows: Locus of Causality = 1, 6, 9; Stability = 3, 7, 11; Personal Control (Controllability) = 2, 4, 10; External Control (Controllability) = 5, 8, 12.

Appendix E: Causal Attributions for Current Smoking

Below is a list of possible causes. Please indicate how strongly you agree or disagree that the following are causes of your current smoking.

	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree	Prefer not to answer
Heredity (runs in my family)*						
Seeing other people smoke						
Stress						
Brain chemistry						
Work/school problems						
My mental attitude (e.g., thinking about life negatively)*						
Lack of willpower						
Habit						
My emotional state*						
Feeling down (e.g., sad, lonely, empty, depressed)						
Anxiety and worrying						
Physiological addiction to nicotine						
Social pressure						
Family/relationship problems*						
My personality or character*						
Financial stress/worries						
Overwork*						
Genetics						

*Original or revised IPQ-R item

If there are any additional causes of your smoking not included above, please list them below: _____

_____

Please list what is the ONE most important cause for your current smoking. You may use any of the causes listed in the table above or a cause of your own: _____

　　❍ Prefer not to answer

Appendix F: Perceived Treatment Control

We are interested in your personal views of how you see your smoking. Please indicate how much you agree or disagree with the following statements about your smoking by clicking the appropriate box.

	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree	Prefer not to answer
There is very little treatment can do to stop my smoking *(There is very little that can be done to improve my illness)*						
Treatment for quitting smoking will be effective for me *(My treatment will be effective in curing my illness)*						
The negative effects of my smoking can be prevented with treatment *(The negative effects of my illness can be prevented (avoided) by my treatment)*						
Treatment can control my smoking *(My treatment can control my illness)*						
There is no treatment that can help my smoking *(There is nothing which can help my condition)*						

Note. Original IPQ-R items are in parentheses.

Appendix G: Perceived Behavioural Control – Personal Control

Please indicate how much you agree or disagree with the following statements about your smoking by clicking the appropriate box.

	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree	Prefer not to answer
There is a lot which I can do to control my cravings and withdrawal symptoms *(There is a lot which I can do to control my symptoms)*						
What I do can determine whether I stop smoking *(What I do can determine whether my illness gets better or worse)*						
Whether or not I smoke depends entirely on me *(The course of my illness depends on me)*						
Nothing I do will affect my smoking *(Nothing I do will affect my illness)*						
I have the power to influence my smoking *(I have the power to influence my illness)*						
My actions will have no effect on whether I quit smoking *(My actions will have no affect on the outcome of my illness)*						

Note. Original IPQ-R items are in parentheses.

Appendix H: Perceived Behavioural Control – Self-Efficacy

How confident are you that you could stop smoking if you wanted to?

Not at all   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Extremely*   ❍ Prefer not
confident   *confident*   to answer

How easy would it be for you to stop smoking if you wanted to?

Not at all   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Extremely*   ❍ Prefer not
easy   *easy*   to answer

I am able to quit smoking

Strongly   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Strongly*   ❍ Prefer not
disagree   *agree*   to answer

How confident are you that you cannot smoke, even under difficult circumstances (e.g., when stressed or bored, when drinking alcohol or coffee, or when with smokers)?

Not at all   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Extremely*   ❍ Prefer not
confident   *confident*   to answer

Appendix I: Perceived Treatment Effectiveness

How effective do you think each of these treatments would be to help you stop smoking?

	Not effective at all 1	2	3	4	5	6	Very effective 7	Prefer not to answer
Nicotine Replacement Therapy (patch, gum, etc.)								
Bupropion (Zyban)								
Varenicline (Champix)								
One-on-one advice/counselling from a healthcare professional								
Group counselling/support groups								
Quitline/telephone counselling								
Internet-based support (e.g., message boards, instant messaging)								
Self-help manuals / Internet-based information & tools								
Alternative therapies (hypnosis, acupuncture, herbal remedies, etc.)								

To what extent do you believe the following treatments would increase your chances of quitting smoking?

	Strongly disagree 1	2	3	Neither agree nor disagree 4	5	6	Strongly agree 7	Prefer not to answer
Nicotine Replacement Therapy (patch, gum, etc.)								
Bupropion (Zyban)								
Varenicline (Champix/Chantix)								
One-on-one advice/counselling from a healthcare professional								
Group counselling/support groups								
Quitline/telephone counselling								
Internet-based support (e.g., message boards, instant messaging)								
Self-help manuals / Internet-based information & tools								
Alternative therapies (hypnosis, acupuncture, herbal remedies, etc.)								

Appendix J: Behavioural Intentions

Do you intend to stop smoking in the next month?

Definitely   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Definitely*   ❍ Prefer not
do not   *do*   to answer

How likely is it that you will stop smoking in the next month?

Extremely   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Extremely*   ❍ Prefer not
unlikely   *likely*   to answer

Do you intend to stop smoking in the next 6 months?

Definitely   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Definitely*   ❍ Prefer not
do not   *do*   to answer

How likely is it that you will stop smoking in the next 6 months?

Extremely   ❍ 1   ❍ 2   ❍ 3   ❍ 4   ❍ 5   ❍ 6   ❍ 7   *Extremely*   ❍ Prefer not
unlikely   *likely*   to answer

Which of the following statements best describes whether you plan to quit smoking?

- ❍ I am making a quit attempt right now (I have not smoked for at least 24 hours)
- ❍ I plan on quitting within the next week
- ❍ I plan on quitting within the next month
- ❍ I plan on quitting within the next 6 months
- ❍ I plan on quitting within the next 5 years
- ❍ I plan on quitting eventually but not within the next 5 years
- ❍ I do not plan on quitting smoking ever
- ❍ Prefer not to answer

Please indicate whether you intend to use each type of treatment the next time you try to stop smoking. If you do not intend to make a quit attempt, please imagine what treatment you would be likely to use if you were planning to quit.

	Definitely do not 1	2	3	4	5	6	Definitely do 7	Prefer not to answer
Nicotine Replacement Therapy (patch, gum, etc.)								
Bupropion (Zyban)								
Varenicline (Champix/Chantix)								
One-on-one advice/counselling from a healthcare professional								
Group counselling/support groups								
Quitline/telephone counselling								
Internet-based support (e.g., message boards, instant messaging)								
Self-help manuals / Internet-based information & tools								
Alternative therapies (hypnosis, acupuncture, herbal remedies, etc.)								

How likely is it that you will use each of the following types of treatment for your next quit attempt? Please imagine that you plan to stop smoking if you are not planning to.

	Extremely Unlikely 1	2	3	4	5	6	Extremely Likely 7	Prefer not to answer
Nicotine Replacement Therapy (patch, gum, etc.)								
Bupropion (Zyban)								
Varenicline (Champix/Chantix)								
One-on-one advice/counselling from a healthcare professional								
Group counselling/support groups								
Quitline/telephone counselling								
Internet-based support (e.g., message boards, instant messaging)								
Self-help manuals / Internet-based information & tools								
Alternative therapies (hypnosis, acupuncture, herbal remedies, etc.)								

Appendix K: **Heaviness of Smoking Index (HSI)**

How soon after you wake up do you smoke your first cigarette?

- ○ Within 5 minutes
- ○ 6–30 minutes
- ○ 31–60 minutes
- ○ After 60 minutes
- ○ Prefer not to answer

Appendix L: Genetics Knowledge

Please indicate whether each statement below is true or false. Please respond even if you are not sure of the correct answer.

	True	**False**	**Prefer not to answer**
The chance to become addicted to smoking is influenced by the presence of certain hereditary traits (genes).			
Genes exist that increase the chance to become addicted to smoking.			
A parent with a genetic predisposition to get addicted to smoking will transfer this predisposition to its children, even when the parent doesn't smoke or has never smoked.			
Genes exist that decrease the chance to become addicted to smoking.			

Appendix M: Smoking Cessation Treatment History

Please indicate whether you have used the following methods to try to help you quit smoking in the past.

	Yes	No	Not sure	Prefer not to answer
Nicotine Replacement Therapy – patch				
Nicotine Replacement Therapy – gum				
Nicotine Replacement Therapy – inhaler				
Nicotine Replacement Therapy – lozenge				
Nicotine Replacement Therapy – nasal spray				
Bupropion (Zyban)				
Varenicline (Champix/Chantix)				
Brief advice from a health care professional				
One-on-one counselling				
Group counselling/support groups				
Quitline/telephone counselling				
Internet-based support and advice (e.g., message boards, instant messaging)				
Internet-based information and tools				
Self-help books or manuals				
Alternative therapies (hypnosis, acupuncture, herbal remedies, etc.)				

Please list any other methods you have used not listed above:

Appendix N: Smoking Characteristics and Background

How many cigarettes do you smoke per day on average? [drop down menu: 1 to 75+ and "prefer not to answer"]

How old were you when you started smoking **daily**? _____

    ❍ Prefer not to answer

Have you ever tried and succeeded in stopping smoking for 24 hours or more?

    ❍ Yes
    ❍ No
    ❍ Prefer not to answer

How many times have you succeeded in stopping smoking for 24 hours or more? [drop down choice from 1 to 11+ and "prefer not to answer"]

What was the longest period of time you quit smoking?

    ❍ More than 1 day but less than 1 week
    ❍ At least 1 week but less than 1 month
    ❍ At least 1 month but less than 6 months
    ❍ At least 6 months but less than 1 year
    ❍ At least 1 year but less than 2 years
    ❍ At least 2 years but less than 5 years
    ❍ 5 years or more
    ❍ Prefer not to answer

Which of the following statements best describes whether you plan to quit smoking?

    ❍ I am making a quit attempt right now (I have not smoked for at least 24 hours)
    ❍ I plan on quitting within the next week
    ❍ I plan on quitting within the next month
    ❍ I plan on quitting within the next 6 months
    ❍ I plan on quitting within the next 5 years
    ❍ I plan on quitting eventually but not within the next 5 years
    ❍ I do not plan on quitting smoking ever
    ❍ Prefer not to answer

Appendix O: Sociodemographic Questions

In what year were you born? [drop down menu: 1911 to 1994 and "prefer not to answer"]

What is your gender?

- ❍ Female
- ❍ Male
- ❍ Prefer not to answer

What ethnic background do you most identify with?

- ❍ Aboriginal (e.g. First Nations, Métis, Inuit)
- ❍ Arab (e.g. Egypt, Iraq, Jordan, Lebanon)
- ❍ Black (African or Caribbean descent)
- ❍ Chinese
- ❍ Filipino
- ❍ Japanese
- ❍ Korean
- ❍ Latin American/Hispanic
- ❍ South Asian (e.g. India, Sri Lanka, Pakistan, Bangladesh)
- ❍ Southeast Asian (e.g. Malaysia, Indonesia, Vietnam, Cambodia, Laos)
- ❍ West Asian (e.g. Turkey, Iran, Afghanistan)
- ❍ White (European descent)
- ❍ Other ethnic group (not listed above)
- ❍ Prefer not to answer

What is your current relationship status? (please check the one that applies best to you)

- ❍ Married and/or living with a partner
- ❍ Divorced
- ❍ Separated
- ❍ Widowed
- ❍ Single, never married
- ❍ Prefer not to answer

What is your highest level of education?

- ❍ Elementary School
- ❍ High School
- ❍ Trade, technical or vocation school, apprenticeship training or technical CEGEP
- ❍ Diploma from a community college, pre-university CEGEP or non-university certificate
- ❍ University certificate below Bachelor's level
- ❍ Bachelor's degree
- ❍ Graduate degree (MSc, MBA, MD, PhD, etc.)
- ❍ None
- ❍ Prefer not to answer

Please indicate which best describes your current employment status:

- ❍ Employed full-time
- ❍ Employed part-time
- ❍ Unemployed
- ❍ Homemaker
- ❍ Doing unpaid or volunteer work
- ❍ Retired
- ❍ Disability
- ❍ Student
- ❍ Other (please specify):_____
- ❍ Prefer not to answer

Appendix P: Survey Invitation

Dear [participant first name],

We are contacting you to let you know about a University of Windsor study that may interest you. This study is investigating factors that influence thoughts and opinions about treatments designed to help you quit smoking (e.g., medication, counselling). You are being invited to take part in this study because when you joined the Ontario Health Study, you agreed to let us contact you about other research studies that have been approved by the Ontario Health Study.

Taking part in this smoking study requires completing an online survey that will take approximately 30-40 minutes. We ask that you complete the survey by December 10. When filling out the online questionnaire, you will be able to save your responses and return to the survey at a later time to complete it.

Completing this questionnaire is entirely voluntary. You may continue to participate in the Ontario Health Study even if you choose not to complete this survey. However, you may be interested to know that your participation in this study also benefits the Ontario Health Study as we will be able to link the information you provide in this survey back to information you have provided in your Ontario Health Study questionnaire.

The study you are being invited to participate in is being conducted by Sabrina Voci (PhD student) and Dr. Ken Cramer (Professor, Faculty Supervisor) of the Department of Psychology at the University of Windsor, and has received clearance from both the University of Windsor Research Ethics Board and the Ontario Health Study's Research Ethics Board at the University of Toronto.

To learn more about this smoking study and decide whether you want to participate, please click on the link below:

[unique URL with invite code]

If you have any questions about the survey, please email Sabrina Voci at smokingstudy@uwindsor.ca.

Sincerely,

Dr. Lyle Palmer
Executive Scientific Director
Ontario Health Study

Appendix Q: **Letter of Information**

LETTER OF INFORMATION FOR CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: Attributed causes of current smoking and treatment seeking among persons who smoke

You are being asked to participate in a research study conducted by Sabrina Voci, from the Department of Psychology at the University of Windsor. The study is being conducted in fulfilment of Sabrina Voci's doctoral dissertation requirements, under the supervision of Dr. Ken Cramer.

Please note that only those who received an email invitation from the Ontario Health Study are eligible to participate in this study. **This study is secondary to the Ontario Health Study – you can continue to participate in the Ontario Health Study even if you choose not to complete this questionnaire.**

If you have any questions or concerns about the research, please feel free to contact Sabrina Voci at smokingstudy@uwindsor.ca or Dr. Ken Cramer at 519-253-3000, ext. 2239.

PURPOSE OF THE STUDY

The purpose of this study is to investigate thoughts that persons who smoke have about what factors contribute to their smoking (such as their genetic makeup, stress levels, etc.), and how this might relate to other thoughts and beliefs about their smoking and plans to seek treatment if they want to quit.

PROCEDURES

If you volunteer to participate in this study, you will be asked to complete an online survey. The online survey includes questions about your current and past smoking history and attempts to quit, how effective you think various treatments are for helping you to quit smoking, thoughts and ideas about your reasons for smoking, how much control you feel you have over your smoking and quitting, your future plans to try to quit and to use treatment or assistance to help quit, as well as questions relating to your overall health and well-being, and general demographic and background information. The survey should take approximately 30-40 minutes to complete.

POTENTIAL RISKS AND DISCOMFORTS

There are no serious anticipated risks associated with participating in this study. Some people may experience mild distress or discomfort as they focus on issues surrounding their smoking and attempts to quit.

POTENTIAL BENEFITS TO PARTICIPANTS AND/OR TO SOCIETY

No direct benefits are anticipated from participating in this study. Your participation in this study will help generate a better understanding of factors that contribute to decisions to quit smoking or seek treatment to help quit. This knowledge may also be used to improve education and treatment programs for people who smoke.

COMPENSATION FOR PARTICIPATION

No compensation is offered for participating in this study.

CONFIDENTIALITY

Any information that is obtained in connection with this study and that can identify you will remain confidential and will be disclosed only with your permission. Anything containing personal information, such as emails to the researchers, will be stored in a place that is secure. Any reports on the findings of this study will contain information that reflects group results and not information about specific individuals in order to protect anonymity. Data may be stored indefinitely; any data that is disposed of will be done so in a secure manner.

The survey you are invited to complete as part of this study is hosted on a secure encrypted FluidSurveys website, a Canadian online survey service. Survey data will be encrypted and stored in a secure location. The survey does not include any questions that ask you to provide identifying information such as your name. Only the researchers will have access to the survey responses until the completion of the study. At the end of this study, the data will be shared with the Ontario Health Study. While you will remain anonymous to the researchers conducting this study, the responses you provide will be identifiable to the staff and researchers at the Ontario Health Study through a unique 5-digit code at the end of the website link emailed to you, which is different for every person invited to participate in this study.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time by clicking the "Withdraw from Study & Discard Responses" link located on each page of the survey. When you click on "Withdraw from Study & Discard Responses," you will be withdrawn from the study and all responses you provided on previous pages of the survey will be deleted. Except for the first survey question, which is required to determine your eligibility for the study, you may refuse to answer any questions you do not want to answer and still remain in the study. You may also withdraw from the study within 30 days of completing the survey by contacting the researchers at smokingstudy@uwindsor.ca.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE PARTICIPANTS

A brief report on the findings of this study will be made available online at http://www.uwindsor.ca/reb/study-results upon completion of the study by June 2013.

SUBSEQUENT USE OF DATA

These data may be used in subsequent studies. The responses you provide as part of this study will be linked with other data and samples you provided in the past or will provide in the future as a participant in the Ontario Health Study. As part of the Ontario Health Study database, the data you provide in the current study will be available to researchers based at recognized institutions in Canada and abroad. The data and samples also may be accessed through an application to the Canadian Partnership for Tomorrow Project, which the Ontario Health Study is a part of (www.partnershipfortomorrow.ca). Access to the data and samples is governed by strict guidelines that protect the confidentiality of participants. All researchers using data from the Ontario Health Study must receive approval from a Research Ethics Board or similar committee before access is permitted. When you completed the baseline questionnaire, you may have consented to allow the OHS to link information that you provide with other health information held by other organizations. This linkage would only take place under very specific data sharing agreements. These are legal agreements that will protect the data and will outline exactly how the data are to be used. Researchers granted access to the data by the OHS will not be able to access any information that would allow them to identify you or any other participant. Only anonymous information will be provided to the researcher for the project analysis. Researchers will not be able to identify individual people but will analyze the linked information by groups of people with similar experiences or health conditions.

RIGHTS OF RESEARCH PARTICIPANTS

If you have questions regarding your rights as a research participant, contact: Research Ethics Coordinator, University of Windsor, Windsor, Ontario N9B 3P4; Telephone: 519-253-3000, ext. 3948; email: ethics@uwindsor.ca.

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

Sabrina Voci
November 1, 2012

You may print out a copy of this letter of information for your records.

To acknowledge that you have read and understood this information and agree to participate in this study, click on 'I agree to participate' below. If you do not wish to participate, click on 'I do not wish to participate' to exit the survey.

❍ I agree to participate
❍ I do not wish to participate

Appendix R: Survey Instructions

Thank you for taking part in this study!

This questionnaire is about your own personal views about your smoking and treatments available for stopping smoking. There are no right or wrong answers to the questions; it is what **you** think that counts.

Some questions may seem very similar to one another. Please complete all questions, even if you think you've answered them already. This helps us to get the most accurate picture of your views. Some demographic questions are the same as questions asked in the Ontario Health Study (OHS) questionnaire. You are being asked to complete these questions again to limit the amount of information the OHS shares with the researchers conducting the current study.

If you wish to skip a question at any time, you may do so by selecting the "Prefer not to answer" option included for each question

This survey is a multi-page survey. On each page you can either click

- **Withdraw from Study & Discard Responses** -- to withdraw from the study and delete all your responses

- **Save and Continue Later** -- to save your responses and return to the survey at a later time to complete it

- **Next** -- to submit your responses and go to the next page of the survey

Appendix S: Smoking Cessation Resource List

Below is a list of free resources available to you if you would like assistance with quitting or reducing smoking now or in the future. This study is not affiliated with any of these services.

Canadian Cancer Society Smoker HelpLine
http://www.smokershelpline.ca/

-- Online access to a supportive community and self-help program.
-- Telephone support for confidential, one-to-one support with a Quit Specialist.
-- Interactive text messaging support.

Stop Smoking Center
http://www.stopsmokingcenter.net/

An eHealth program offering online community support, tools and resources, and access to online Health Educators.

Smoking Treatment for Ontario Patients (STOP) Study
http://www.stopstudy.ca/

The Smoking Treatment for Ontario Patients (STOP) Study is a research project that aims to discover the most effective methods of delivering free smoking cessation medication and counselling support to smokers across Ontario. Since its inception in 2005, the STOP Study has provided free smoking cessation medication and counselling support to over 65,000 Ontario smokers who wanted to quit smoking.

Thank you for your participation in this study!

VITA AUCTORIS

Name:	Sabrina Concetta Voci
Place of Birth:	Toronto, Ontario
Year of Birth:	1977
Education:	Honours Bachelor of Science (with distinction), Psychology University of Toronto, 2001
	Masters of Arts, Applied Social Psychology University of Windsor, 2007
	Doctor of Philosophy, Applied Social Psychology University of Windsor, 2013