THE PERCEIVED CAUSAL STRUCTURE OF CIGARETTE ADDICTION AMONG A SAMPLE OF LATE ADOLESCENT SMOKERS AND NON-SMOKERS: A NETWORK COMPARISON STUDY

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by

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ABSTRACT

Research has highlighted the roles of smoking beliefs in predicting smoking-related behaviors during adolescence. The present study used network analysis to examine the perceived causal belief structure of cigarette addiction among a sample of late adolescent, undergraduate students. Eighty-two non-smokers and seventy-nine smokers completed a questionnaire that asked them to rate the strength of the causal relationships between eight possible causes and effects of cigarette addiction: 1) smoking; 2) parental smoking; 3) stress; 4) physical health problems; 5) peer pressure; 6) being impulsive; 7) having friends who smoke; 8) cigarette addiction. A subsample of participants (N = 35) completed the measure a second time within seven days of their first session to obtain an estimate of the reliability of the network analysis method. Inductive eliminative analysis was used to produce separate networks for smokers and non-smokers consisting of the most highly rated and consensually-endorsed causal relationships. Separate networks were also created for responses at time 1 and time 2 for the subsample of participants completing the measure twice. Multidimensional scaling determined the spatial structure of the networks. The analyses resulted in highly consensual and interpretable network diagrams that conveyed the perceived causal structure of cigarette addiction for both smokers and non-smokers. In line with previous examinations of smoking-related beliefs, smokers held more optimistic beliefs about the effects of smoking and cigarette addiction relative to non-smokers. Follow-up t-tests revealed significant differences in the strength of four perceived causal relationships contained in the smoker and non-smoker networks, each indicating more optimistic beliefs about the consequences of smoking behaviors and the experience of peer pressure to smoke in smokers relative to non-smokers. A comparison of the time 1 and time 2 network diagrams revealed highly similar networks suggesting that the network analysis method is moderately reliable. By highlighting salient beliefs about the causes and effects of cigarette addiction, the network analysis method may be valuable for tailoring interventions to impact smoking beliefs during adolescence.
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1. Introduction

Smoking-related diseases remain the most preventable causes of death worldwide (American Cancer Society, 2010) and are also associated with substantial economic costs due to health care expenditures and lost productivity (Center for Disease Control, 2008). Smoking initiation is most likely to occur during adolescence (Chen & Kandel, 1995; Kandel & Logan, 1984; Lantz, 2003), and the majority of daily adult smokers start smoking by the age of 18 (USDHHS, 2012), highlighting adolescence as a critical period for smoking intervention efforts.

Adolescent smoking has proven to be a rapidly changing phenomenon. Youth cigarette use underwent a decline in recent years, beginning in the mid-1990s, although this decline has decelerated since 2010 (Johnston, O’Malley, Bachman, & Schulenberg, 2013). Changes in beliefs about smoking, including increases in perceived risk and disapproval of smoking, appear to have contributed to the decrease in cigarette use (Johnston, O’Malley, Miech, Bachman, & Schulenberg, 2014), as have many education and policy efforts that aimed to alter attitudes towards smoking (Gutman, 2011).

Given the changing landscape of cigarette use and attitudes towards smoking, the use of methodology that allows a thorough investigation of current adolescent attitudes towards smoking is integral to ensuring that intervention efforts remain relevant and effective. The present study provides an overview of the role of smoking prevention programs during adolescence, with a focus on information-deficit programs, before applying network analysis to gain novel insights into the perceived causes and effects of cigarette addiction in a late adolescent sample.

1.1. The rationale for smoking prevention programs during adolescence

Evidence suggests that adolescents are particularly susceptible to the influence of cigarettes and to developing nicotine addiction (for review see Lydon, Wilson, Child, & Geier,
under review). For example, adolescent rats exhibit enhanced sensitivity to the reinforcing effects of nicotine in conditioned place preference (Belluzzi, Lee, Oliff, & Leslie, 2004; Brielmaier, McDonald, & Smith, 2007; Shram & Le, 2010) and oral self-administration procedures (Adriani, Macri, Pacifici, & Laviola, 2002). In human subjects, initial smoking experiences during adolescence relative to adulthood result in more pleasant experiences (Buchmann et al., 2011). Such findings are important considering the role of initial reinforcement consequences in setting the stage for subsequent cigarette use. Indeed, both longitudinal and retrospective studies have demonstrated that self-reported pleasurable initial experiences are predictive of continued cigarette use (Bewley, Bland, & Harris, 1974; DiFranza et al., 2007; Pomerleau, Pomerleau, & Namenek, 1998).

As well as being more likely to have an initial smoking experience that renders them more likely to continue smoking, adolescents are also more susceptible to developing nicotine addiction than adult-onset smokers. During adolescence, there is often a rapid progression from smoking initiation to nicotine dependence (Dierker et al., 2012), with symptoms of nicotine dependence being reported within days to weeks of the onset of occasional smoking (DiFranza et al., 2000). Studies examining the acquisition of nicotine self-administration in animals have observed a faster rate of acquired nicotine self-administration among adolescent relative to adult rats (Chen, Matta, & Sharp, 2007). Furthermore, while cigarette addiction has been less extensively characterized in adolescence compared to adulthood, there is evidence that nicotine dependent adolescents, like adults, experience a heightened impulse to approach cigarettes in the context of drug-induced impairments in cognitive control (Bechara, 2005; Jacobsen et al., 2005; Lee, Lim, Wiederhold, & Graham, 2005; Musso et al., 2007; Robinson & Berridge, 1993; Rubinstein, Luks, Dryden, Rait & Simpson, 2011). Differences in the adolescent experience of nicotine addiction relative to the adult experience may occur during withdrawal with adolescents experiencing milder withdrawal symptoms (Smith, Cavallo, McFetridge, Liss, & Krishnan-Sarin, 2008), findings also observed in non-human animals (Kota, Martin, Robinson, & Damaj, 2007;
Milder withdrawal symptoms indicate that withdrawal may play a lesser role in adolescent relative to adult quit attempts (Prokhorov et al., 2001), however the lack of withdrawal symptoms may also lead adolescents to believe that the effects of nicotine are not causing harm, leading them to continue smoking.

Finally, evidence suggests that adolescent nicotine exposure results in persistent changes in affect (Slawecki, Thorsell, Khoury, Mathe, & Ehlers, 2005; Slawecki, Gilder, Roth, & Ehlers, 2003; Iniguez et al., 2009; Ribeiro-Carvalho et al., 2011), cognition (Counotte et al., 2009; Fountain, Rowan, Kelley, Willey, & Nolley, 2008; Mateos et al., 2011), drug-related behavior and responses (Bracken, Lee, Oliff, & Leslie, 2011; Brielmaier et al., 2007; Hutchison & Riley, 2008; Collins, Montano, & Izenwasser, 2004), and neurobiology (Abreu-Villaca et al., 2003; Trauth, Seidler, McCook, & Slotkin, 1999; Trauth, Seidler, & Slotkin, 2000), many of which are not found following adult-onset nicotine exposure. These persistent effects continue beyond acute withdrawal and represent a persistent abstinent syndrome characterized by negative affect, cognitive deficits, and increased reactivity towards nicotine and other drugs, all of which may undermine smoking cessation. Note that these findings have emerged primarily from the rodent literature. Dissociating the effects of smoking during adolescence from smoking during adulthood in humans remains a difficult task due to the early onset of the majority of smokers during adolescence (Chen & Miller, 1998) and due to the ethical considerations of constructing studies suited to answering these questions.

1.2. Prevention programs targeting adolescent smoking behaviors

Prevention programs designed to impact smoking behaviors during this vulnerable period of development often contain components that aim to remedy a perceived information-deficit in terms of the causes and effects of smoking (e.g., LifeSkills Training, Botvin & Griffin, 2004; Project Towards No Drug Abuse, Sussman, Dent, & Stacy, 2002; Strengthening Families
Program, Molgaard & Spoth, 2001; The Tobacco and Alcohol Prevention Project, Hansen, Malotte, & Fielding, 1988). Misperceptions such as the physical consequences of smoking displayed in advertisements are myths (Sussman et al., 1993), for example, are corrected.

The logic models of change for these programs are in line with theoretical models of health behaviors (e.g., The Theory of Planned Behavior, Ajzen, 1991; The Health Belief Model, Becker, 1974; The Common Sense Model of Health and Illness, Leventhal, Meyer, & Nerenz, 1980), which posit that one’s beliefs about the consequences of behaviors influence behavior and behavior change. For example, in the health belief model, one of the first and one of the most widely used theories of health behavior (National Cancer Institute, 2003), a perceived susceptibility of developing lung cancer due to cigarette use would lead to the avoidance or reduction of smoking behavior. In line with these models, a prevention component increasing awareness of the negative health effects of smoking would lead to reduced smoking behaviors.

Such programs have shown both short- and long-term effectiveness at reducing the rate of adolescent smoking uptake (Botvin & Griffin, 2004; Flay, 2009; Sussman et al., 2002), although due to the multi-component nature of these programs, their success cannot be attributed solely to the information-deficit components. Indeed, reviews of information-only curricula have found mixed outcomes (Hansen, 1992), with recent reports demonstrating no effects of information-only curricula on preventing smoking behaviors (Thomas, McLellan, & Perera, 2013).

This raises questions about the validity of theories emphasizing the effects of health beliefs and information on behavior. Indeed, cognitive models of adolescent decision-making which focus on risk perception and information-deficits to explain risk-taking have been criticized (Boyer, 2006; Steinberg, 2004). Contemporary cognitive-neuroscience conceptions of adolescent risk-taking emphasize “nonexistent...decision making” (Steinberg, 2004, p.52). A consideration of distinctions from the adult decision-making literature between fast and slow (Metcalfe & Mischel, 1999), or impulsive and reflective (Bechara, 2005), systems are useful in
discussing this style of decision-making. The impulsive system is supported by a widely distributed circuitry, originating in the ventral tegmental area, extending through the ventral striatum, and projecting to the medial and ventral regions of the prefrontal cortex and the anterior cingulate cortex (O’Doherty, 2004; Schultz, Tremblay, & Hollerman, 2000; Wise, 2002). This system supports reward valuation and the prediction of rewards and punishment (O’Doherty, 2004) and propels organisms to engage in incentive-motivated behaviors (Wahlstrom, Collins, White, & Luciana, 2010). This system is specialized for fast responses to stimuli. The reflective system, supported by a widely distributed circuitry, although the prefrontal cortex has received the most attention (Luna, Padmanabhan, & O’Hearn, 2010), contributes to decision-making by inhibiting prepotent responses from the impulsive system and providing the means to engage in deliberative reasoning (Knoch & Fehr, 2007). In the style of decision-making described by Steinberg (2004), the decision-making is based solely on the output of the impulsive system with adolescents acting on impulse and not stopping to think about their actions or incorporating relevant information about the behavior at hand into their decisions to act.

The experimental literature has provided evidence for this model of decision-making in adults. Increased ventral striatum activity has been observed before risky choices during financial decision-making tasks (Kuhnen & Knutson, 2005), greater fronto-parietal activity has been observed when subjects chose longer-term options relative to times in which subjects chose more immediately available rewards (McClure, Laibson, Loewenstein, & Cohen, 2004), and decreased risk-taking behavior was observed when right dorsolateral prefrontal cortex activity was upregulated through anodal direct current stimulation (Fecteau et al., 2007).

In adolescence, the configuration of these systems is thought to render them more vulnerable, relative to other ages, to act on impulse and less capable of inhibiting prepotent responses to allow the engagement of deliberative reasoning. In line with this, the brain areas comprising these systems, as well as the connections between them, undergo significant
development during adolescence (Giedd, 2008; Giedd et al., 1999; Gogtay et al., 2004). Critically, brain areas involved in functions associated with the reflective system are among the last to develop during adolescence (Gogtay et al., 2004). This delayed structural development is mirrored in the behavioral and functional magnetic resonance imaging literature with the continued development of brain processes underlying cognitive control observed through the adolescent period and into young adulthood (Stevens, Kiehl, Pearlson, & Calhoun, 2007; Velanova, Wheeler, & Luna, 2008; for reviews see Luna et al., 2010).

While the functions of the reflective system develop linearly through the adolescent period, the functioning of the impulsive system is thought to be at a peak during the adolescent period (Luciana & Collins, 2012). In line with this, adolescents demonstrate increased approach behavior towards potential rewards relative to other ages (Cauffman et al., 2010). Adolescents also show exaggerated ventral striatum responses during reward anticipation and reward receipt relative to adults (Ernst et al., 2005; Galvan et al., 2006; van Leijenhorst et al., 2010) and hypo-responsive striatal activity during the assessment of incentive value for upcoming trials (Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010). This pattern of activation suggests that adolescents may have limitations in reward assessment and a heightened reactivity in anticipation of reward, rendering them vulnerable to behavior directed by incentives when the value of the incentive has not been appropriately assessed. To account for this pattern, much attention has been directed to development changes to the dopamine system due to its role in reward processing (for reviews see Ernst, Romeo, & Andersen, 2009; Galvan, 2010; Wahlstrom et al., 2010). Dopamine tissue concentrations in both subcortical and cortical regions are at a relative high during adolescence (Goldman-Rakic & Brown, 1982; Irwin et al., 1994). Wahlstrom et al. (2010), drawing on findings suggesting an inverted u-shaped influence of dopaminergic activity on prefrontal functioning with both deficient and excessive levels impairing cognitive functions (Arnsten, 2009), hypothesize that dopamine levels in the prefrontal region, under certain circumstances, exceed optimal levels, allowing activity in subcortical regions to dominate
while prefrontal regions are “overdosed”. The greater dopaminergic activity in the nucleus accumbens is also thought to shift information flow in the nucleus accumbens towards greater limbic and less prefrontal input (Goto & Grace, 2008). This configuration is theorized to undermine the top-down modulation of approach behaviors.

The context-specificity of this style of decision-making during adolescence is key to contemporary cognitive-neuroscience theories. Adolescents demonstrate adult-like decision-making abilities in contexts during which affective responses are not triggered (Reyna & Farley, 2006). It is in contexts in which the hyper-reactive, adolescent limbic system responds to incentives in the environment that adolescent decision-making becomes compromised, relying overly on the impulsive system without the ability to consistently draw on the reflective system to inhibit prepotent responses and engage deliberative decision-making processes (Luciana & Collins, 2012). This has been elegantly demonstrated in studies involving conditions of low and high emotional arousal (e.g., Figner, Mackinlay, Wilkening, & Weber, 2009; van Duijvenvoorde, Jansen, Visser, & Huizenga, 2010).

From this perspective, it is unclear if program developers can expect adolescents to draw upon information regarding the potentially negative consequences of smoking when in the affectively-charged contexts in which smoking initiation occurs (Friedman, Lichtenstein, & Biglan, 1985; Delorme, Kreshel, & Reid, 2003). Often lacking from such cognitive-neuroscience models, however, is a conception of the course of decision-making when an impulse has been effectively inhibited. Once a response has been inhibited, an adolescent may decide, after deliberative decision-making, that their initial response to engage in a behavior remains desirable. At this point in the decision-making process, having accurate information about the consequences of the behavior is crucial and could lead the adolescent to decide not to engage in the behavior in question (see Lydon et al., under review). Thus, it is suggested that information-deficit theories are valid, although the likelihood of preventing behaviors by targeting
information-deficits alone is unlikely to be successful without the inclusion of other intervention components.

### 1.3. Smoking beliefs and smoking behaviors

While a focus on making up an information-deficit is unlikely to be a panacea for adolescent smoking, the empirical literature has confirmed the importance of considering beliefs about the consequences of smoking in predicting future smoking behavior. Beliefs about the long term consequences of smoking may include nicotine addiction (Dani & De Biasi, 2001) and lung cancer (Danaei, Vander Hoorn, Lopez, Murray, Ezzati, & Comparative Risk Assessment collaborating group, 2005). Short-term consequences may also be considered and include negative consequences, such as the production of an unpleasant odor, but also positive consequences such as the experience of a ‘buzz’ (Friedman et al, 1985; Hirschman, Leventhal, & Glynn, 1984). Beliefs about these short- and long-term consequences are predictive of future smoking behaviors. Beliefs about the stress-reducing and relaxing consequences of smoking, for example, significantly predict the smoking stages of adolescents, with experimental smokers holding more positive beliefs about smoking than non-smokers and regular smokers holding more positive beliefs than both experimental and non-smokers (Wang, Fitzhugh, Eddy, & Westerfield, 1996). Among adults, health beliefs about smoking differentiate ex-smokers and current smokers with current smokers viewing themselves as less susceptible to the health problems related to smoking (Chapman, Wong, & Smith, 1993; Weinberger, Greene, Mamlín & Jerin, 1981). Beliefs about susceptibility to smoking-related health problems also predict intentions to quit smoking (Norman, Conner, & Bell, 1999) and are significant predictors of smoking cessation success (Kaufert, Rabkin, Syrotuik, Boyko, & Shane, 1985).

The majority of studies examining the link between health beliefs and smoking behaviors have employed cross-sectional designs. This is problematic when attempting to establish the direction of influence between health beliefs and smoking behaviors. Although beliefs are
theorized to motivate behavior, beliefs may also be reflective of behavioral experiences (Festinger, 1957; Gerrard, Gibbons, Benthin, & Hessling, 1996). Thus, cross-sectional studies may be limited in the insights they provide into the association between smoking beliefs and smoking behaviors. There are a number of longitudinal studies that have been integral in demonstrating that health beliefs, including the perceptions of risk, including risk of heart attack and getting lung cancer, as well as perceptions of benefits, such as looking grown-up and becoming popular, predict smoking initiation among adolescents (Krosnick, Chang, Sherman, Chassin, & Presson, 2006; Rodriguez, Romer, & Audrain-McGovern, 2007; Song et al., 2009).

1.4. The structure of beliefs about cigarette addiction

Smoking beliefs, then, are predictive of smoking behaviors and targeting these beliefs as part of multi-component interventions may be an effective means of curbing future smoking behaviors. While the aforediscussed studies have enhanced understanding of the beliefs about the consequences of smoking that may encourage and discourage smoking behaviors, further insight may be gained by examining the perceived causal belief structures of cigarette addiction.

A person’s understanding of an event is based on the perceived location of the event within a temporally ordered network consisting of interconnected causes and effects (Kelley, 1983). This network is termed the perceived causal structure. Such belief networks can be examined through network analysis, a method that has been successfully employed to provide insight into lay perceptions of the causes of coronary heart disease (Green & McManus, 1995), lower back pain (Campbell & Muncer, 2005), and obesity (Brogan & Hevey, 2009), but also into lay perceptions of the causes of phenomena as diverse as loneliness (Lunt, 1991) and the Second Iraq War (Rafiq, Jobanuptra, & Muncer, 2006). While network analysis has been applied to investigate the perceived causes of drug use among college students (Muncer, Epro, Sidorowicz, & Campbell, 1992), no analysis has focused on the perceived causes of smoking and nicotine addiction specifically.
Unlike traditional methods which focus on atomic beliefs or aggregations of atomic beliefs, network analyses of causal belief structures provide further, complementary information about the perceived inter-connections between individual causes. Information that can be gleaned from the network includes information on the extent (distinctions between proximal and distal causes), patterning (distinctions between simple to complex connections), and direction (the temporal ordering of connections from past to future) of the causes (Brogan & Hevey, 2010; Kelley, 1983). The analysis also results in an easily interpretable, consensual diagram of the perceived causal belief structure of the sample of participants tested. A consideration of the perceived causal structure of cigarette addiction, then, may provide a greater insight into lay beliefs about the causes and effects of cigarette addiction.

If proven feasible, the method may be useful as a potential intervention aid to restructure the beliefs of intervention participants as it will highlight consensually endorsed beliefs about the causes and effects of cigarette addiction. The method may guide the construction of information-deficit intervention components, tailoring program content in response to the beliefs of program participants. The health communication literature has demonstrated the strengths of tailored over non-tailored health messages (Kreuter & Wray, 2003). Individuals are more likely to actively process information if they are motivated to do so (Petty & Cacioppo, 1986). An important determination of the motivation to process information actively is perceived relevance. Tailored health communications have been consistently perceived as more relevant, better remembered, and more effective for influencing health behaviors (Skinner, Campbell, Rimer, Curry, & Prochaska, 1999). In the context of smoking behaviors, interventions providing tailored information to program participants have shown promising effects for smoking behavior change (Dijkstra, De Vries, Roijackers, & van Breukelen, 1998; Strecher, 1999).

1.5. Generating perceived causal structures
Two main methods have been employed to generate perceived causal belief structures - the grid method and the diagram method. In the grid method, participants are presented with a
grid in which the potential causes are printed along the top and left-hand side of the grid. Participants are required to rate their perception of the presence of a link between each putative cause and the others (e.g., Gillen & Muncer, 1995; Lunt & Livingston, 1991) or, as in more recent studies (e.g., Brogan & Hevey, 2009; Rafiq et al., 2006), to rate the strength of the causal relationship between each putative cause and the others on a likert scale. A network is then constructed from the participant input. In the binary method, a network is constructed using data from an aggregated grid which combines the responses of all participants, with causes being entered into the network according to the number of individuals endorsing a link. In the likert method, the links are ranked by mean strength of the connections with the strongest links being entered first. The decision to stop network construction and to stop adding links to the network has been based on two criteria in the past. In some studies, network construction has followed the minimum systems criterion (e.g., Lunt, 1991) and stopped when all the causes have been entered at least once. The second criteria is a cause to link ratio in which network construction is stopped when the number of links required to add a cause is deemed too great (e.g., Campbell & Muncer, 1990). An alternative method of network construction has emerged, primarily in the likert method of network construction, that uses a form of inductive eliminative analysis (Muncer & Gillen, 1997). In this method, causes are added to the network until the level of endorsement of the entire network falls to a point deemed too low to be considered consensual.

In the diagram method, the target cause is presented in the center of a page and participants are asked to draw a network diagram indicating the relationships between causes (Campbell & Muncer, 2005). Participants also rate the strength of the links they draw on a scale from zero to one-hundred. Composite diagrams are then produced to indicate the mean strength of the causal paths drawn as well as the percentage of individuals endorsing the link. Strengths of the diagram method include the ability of participants to include factors that they perceive as relevant but are not included within the grid. A limitation of the method is the
tendency of participants to consider direct links to the target cause while neglecting indirect links and bidirectional links between the target cause and other causes.

The present study made use of the grid method of network construction as it was expected, based on empirical research, that cigarette addiction would be considered both an effect of other states and behaviors as well as a cause of other states and behaviors (e.g., Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Parrot, 1998). The present study incorporated recent changes to the traditional grid method. Earlier studies did not include the target behavior or state of interest (e.g., Gillen & Muncer, 1995; Lunt, 1991). Since then the inclusion of the target behavior has been advocated (Green & McManus, 1995) and the importance of doing so has been supported by a number of studies (Brogan & Hevey, 2009; Heffernan, Green, McManus, & Muncer, 1998). Doing so allows the consideration of the target state or behavior as both an effect but also a cause of other states or behaviors. Another modification of the grid method employed in the current study was to replace the input grid with a series of questions which elicited responses to every possible combination of links between potential causes (French, Marteau, Senior, & Weinman, 2002). Answering questions may be less confusing than filling out a complex matrix. Furthermore, interpretational issues are minimized as the investigator maintains greater control over the perceived relationships between the different causes respondents are asked to rate.

1.6. Aims of the current study

This study aimed to determine whether the grid method of network analysis could reveal a consensual representation of the perceived interconnections between cigarette addiction and putative causes and effects. Another aim was to determine if the relationships between the putative causes differed among non-smokers and smokers. Specifically, based on past research, it was hypothesized that smokers would perceive smoking and cigarette addiction as resulting in fewer and less severe negative consequences (Chapman et al., 1993; Weinstein,
Marcus, & Moser, 2005). Finally, as little research has been conducted into the reliability of the network analysis method (although see Green, Muncer, Heffernan, and McManus, 2003 for preliminary assessments of the psychometric properties of the network analysis method in the context of the causal structure of loneliness), a preliminary assessment of the reliability of the method in the context of the perceived causal structure of cigarette addiction was undertaken.

2. Method

2.1. Participants

Upon receipt of ethical approval, a convenience sample of 161 participants was recruited from the Psychology subject pool at the university. Eighty-two non-smokers (24 males) were defined as participants that had smoked less than 100 cigarettes in their lives and reported no current daily or occasional smoking. They ranged in age from 18 to 23 ($M = 18.76$, $SD = 1.12$). Seventy-nine smokers (30 males) were defined as participants that had smoked at least 100 cigarettes in their lives and reported current daily ($N = 28$) or someday ($N = 51$) smoking. Smokers ranged in age from 18 to 24 ($M = 19.56$, $SD = 1.46$). A random subsample of smokers completed the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). The scores on the FTND for everyday smokers ($M = 1.95$, $SD = 1.93$, $N = 20$) were not significantly different from the scores of someday smokers ($M = 1.38$, $SD = 1.71$, $N = 13$), $t (31) = 0.86$, $p = .40$. The FTND scores suggested a sample of smokers with a very low level of dependence. Everyday smokers reported a higher number of cigarettes smoked per day ($M = 6.50$, $SD = 4.71$) relative to someday smokers ($M = 2.77$, $SD = 2.31$), $t (29.34) = 3.03$, $p < .01$.

2.2. Materials

2.2.1. Questionnaire development

As the study was interested in lay beliefs about the causal structure of cigarette addiction, a pilot study was undertaken to inform the study questionnaire. Semi-structured...
interviews on the topic of smoking behaviors, including cigarette addiction, were conducted with four participants recruited from the Psychology subject pool (3 non-smokers, 1 self-identified smoker). The results of these interviews generated seven salient causal attributions for cigarette addiction which were included in the questionnaire.

2.2.2. Questionnaire
Participants completed the Perceived Causes of Cigarette Addiction questionnaire (Appendix A). On each page, participants were asked questions to determine how likely they believed a given cause was to lead to the other causes on a scale ranging from 0 (highly unlikely) to 5 (highly likely). An example of a question with smoking as the cause and physical health problems as the effect is: “How likely is it that smoking causes physical health problems?” Participants were asked to base their ratings on their personal opinions. The seven causes and their definitions were: (1) smoking - the act of smoking cigarettes; (2) parental smoking – having at least one parent who smokes cigarettes; (3) stress - a state of mental or emotional strain or tension; (4) physical health problems - experiencing problems with one’s physical (as opposed to mental) health; (5) peer pressure - influence from people that belong to the same age group or social group; (6) being impulsive - acting on impulse before thinking things through; (7) friends who smoke - having friends who smoke cigarettes; (8) cigarette addiction - being addicted to cigarettes means a person has formed a dependence on cigarettes that is hard to control. Cigarette addiction, the target state, was incorporated into the measure to allow for direct links between the other causes and cigarette addiction and also to allow for the possibility that cigarette addiction would be perceived as producing the other causes. In total, the method allowed the consideration of fifty-six links between the putative causes.
2.2.3. Reliability assessment
In order to assess the reliability of the network method, a subsample of participants ($N = 35$) completed the measure a second time in a separate session within seven days of their first session. Thirty-one smokers and 4 non-smokers completed two sessions.

2.3. Analysis
Separate networks were created for non-smokers and smokers. For each network, the mean score for each of the fifty-six links was computed. The links were ranked by mean endorsement. Each link was added to the network in a hierarchical fashion, with the highest ranked links being entered first. Inductive eliminative analysis was used to determine the overall endorsement of the networks produced. To ensure the resulting network could be deemed consensual, an item average criterion was adopted during network construction such that the mean strength of a participant’s endorsement of all links in the network must be above the criterion. In line with previous studies, the item average criterion was set at 4 on the 0 to 5 scale (Brogan & Hevey, 2009; Rafiq et al., 2006). As such, with a network of five links, a participant would require an aggregate score of 20 or above on these links to result in an item average which endorses the five-link network at the item average criterion (Reser & Muncer, 2004). To further ensure the resulting networks could be deemed consensual, network construction stopped when network endorsement at the item average criterion was not achieved by at least 70% of participants. Multidimensional scaling was used to determine the spatial structure of the network. A disagreement index (Muncer, 1995) was applied to obtain a quantitative measure of the differences between the networks constructed for smokers and non-smokers. The disagreement index is obtained by dividing the number of links that are not common to both networks by the number of links appearing on both networks. A 0 on this index would represent full agreement between networks while 1 would represent complete disagreement. Finally, t-tests were used to test differences in shared links between smoker and non-smoker networks.
To determine the reliability of the network analysis method in eliciting the perceived causal structure of cigarette addiction, separate networks were constructed for the measures completed at time 1 and time 2. The method of network construction was identical to that for smoker and non-smoker network creation.

3. Results

3.1. Non-smoker network

The mean strength of the causal links as rated by participants are presented in Table 1. Multidimensional scaling of the causal ratings revealed a two-dimensional solution with a low level of stress of .02 and a dispersion accounted for of .98, indicating an excellent fit between the data and the solution. Using inductive eliminative analysis, the first causal link to be entered into the network was *addiction to physical health problems*, with a mean rating of 4.90 and an endorsement by 98% of participants at a rating of 4 and above. The second causal link to be entered into the network was *smoking to physical health problems*, with a mean rating of 4.87 and a network endorsement of 98%. In total, 24 links were added to the network (fig. 1). Adding link 25, *impulsivity to stress*, would have resulted in a drop in network endorsement from 74% to 68% and thus network constructed was stopped when link 24 was added (Table 2).

3.2. Smoker network

The mean strength of the causal links as rated by participants are presented in Table 3. Multidimensional scaling of the causal ratings revealed a two-dimensional solution with a low level of stress of .01 and a dispersion accounted for of .99, indicating an excellent fit between the data and the solution. The first causal link to be entered into the network was *addiction to physical health issues*, with a mean rating of 4.67 and a network endorsement of 92%. The second causal link to be entered into the network was *addiction to smoking*, with a mean rating of 4.62 and a network endorsement of 92%. In total, 17 links were added to the network (fig. 2).
Adding link 18, *parental smoking* to *physical health issues*, would have resulted in a drop in network endorsement from 71% to 68% and thus network construction stopped after link 17 was added (Table 4).

### 3.3. Non-smoker and smoker network comparison

The number of incoming and outgoing connections per cause in smoker and non-smoker networks are listed in Table 5. Smoker and non-smoker networks shared 17 links. Seven links unique to the non-smoker network included: *smoking* to *peer pressure to smoke*; *peer pressure to smoke* to *physical health problems*; *addiction* to *stress*; *parental smoking* to *stress*; *addiction* to *being impulsive*; *peer pressure to smoke* to *addiction*; *addiction* to *peer pressure to smoke*.

Comparing the two networks produced a disagreement index of 0.41.

The mean strengths of shared links were compared using independent samples t-tests (Table 6). Significantly different responses for four links were observed between smokers and non-smokers at a Bonferroni-corrected p-value of .003. Non-smokers rated the following causal relationships as significantly more likely than smokers: *smoking* to *physical health problems*; *smoking* to *addiction*; *peer pressure to smoke* to *smoking*; *peer pressure to smoke* to *stress*.

The effect sizes of these differences were medium in size according to the benchmarks presented by Cohen (1988).

### 3.4. Reliability of the network construction method

The mean strength of the causal links as rated by participants at time 1 are presented in Table 7. Multidimensional scaling of the causal ratings revealed a two-dimensional solution with a low level of stress of .01 and a dispersion accounted for of .99, indicating an excellent fit between the data and the solution. The first causal link to be entered into the network was *physical health problems* to *stress*, with a mean rating of 4.69 and an endorsement by 97% of participants at a rating of 4 or above. The second causal link to be entered into the network was *cigarette addiction* to *physical health problems*, with a mean rating of 4.69 and a network
endorsement of 94%. In total, 18 links were added to the network (fig 3). Adding link 19, *peer pressure to smoke to cigarette addiction*, would have resulted in a drop in network endorsement from 71% to 69% (Table 8).

The mean strength of the causal links as rated by participants at time 2 are presented in Table 9. Multidimensional scaling of the causal ratings revealed a two-dimensional solution with a low level of stress of .02 and a dispersion accounted for of .98, indicating an excellent fit between the data and the solution. The first causal link to be entered into the network was *cigarette addiction to smoking*, with a mean rating of 4.77 and an endorsement by 94% of participants with a rating of 4 or above. The second causal link to be entered into the network was *cigarette addiction to physical health problems*, with a mean rating of 4.6 and a network endorsement of 91%. In total, 15 links were added to the network (fig 4). Adding link 16, *peer pressure to smoke to having friends who smoke*, would have resulted in a drop in network endorsement from 74% to 69% (Table 10).

Comparing the two networks revealed similar networks. The networks at time 1 and time 2 shared 15 links. Three links were present in the time 1 network that were not present in the time 2 network: *peer pressure to smoke to having friends who smoke; being impulsive to smoking; cigarette addiction to peer pressure to smoke*. This resulted in a disagreement index of 0.20. Note that the links present at time 1 but absent at time 2 would be the first, third, and fourth links to be added to the time 2 network were the item average criteria made less stringent at time 2.

4. Discussion

This study is the first application of network analysis to elicit the perceived causal structure of cigarette addiction in a sample of late adolescent smokers and non-smokers. The method resulted in highly consensual, interpretable, and moderately reliable network diagrams to convey the perceived causal structure of cigarette addiction for both smokers and non-
smokers. The networks are discussed in relation to Kelley’s (1983) properties of extent, patterning, and direction.

### 4.1. Network properties

In terms of extent, seven distal causes of cigarette addiction emerged within the non-smoker network. *Having friends who smoke, being impulsive, parental smoking, and stress* operated through the intermediate causes of *smoking* and *peer pressure to smoke* which operated as proximal causes of *cigarette addiction*. *Physical Health Issues* operated through extended intermediate connections, operating through *stress* which then caused *smoking*. As well as having proximal connections with smoking, *having friends who smoked, parental smoking*, and *peer pressure* also operated through more distal pathways through their effects on *peer pressure, stress and health issues*, and *having friends who smoke* and *health issues*, respectively. *Smoking* also operated as an indirect cause, operating through *peer pressure to smoke*.

The smoker network was similar to the non-smoker network in terms of extent. *Having friends who smoke, being impulsive, parental smoking, and stress* operated through the intermediate cause of *smoking* which operated as the sole proximal cause of *cigarette addiction*. *Physical health issues* also operated through *stress* to lead to *smoking* and then *addiction*, as in the non-smoker network. *Peer pressure* and *parental smoking* operated through fewer distal pathways in the smoker network compared to the non-smoker network.

In terms of patterning, *being impulsive* was the least complex causes in the non-smoker network while *smoking* and *cigarette addiction* represented the most complex causes with the highest number of incoming and outgoing connections. A less complex patterning was observed in the smoker network with fewer incoming connections to *being impulsive* as well as fewer outgoing connections from *smoking*, and *cigarette addiction*. *Peer pressure to smoke* and
parental smoking were also more complex cause within the non-smoker, relative to the smoker, network.

In terms of direction, a complex temporal ordering emerged in both non-smoker and smoker networks. In both non-smoker and smoker networks, smoking, the most complex, proximal cause of cigarette addiction, was precipitated by factors in the social environment including parental smoking, peer pressure to smoke, and having friends who smoke, as well the personality factor of being impulsive, and the transient state of stress. With the onset of smoking, a cyclical pattern was perceived to be produced in which smoking led to the causes which had led to smoking. This cyclical pattern was slightly more complex within the non-smoker network relative to the smoker network with one extra link from smoking to peer pressure to smoke endorsed in the non-smoker network. A similarly complex temporal relationship emerged for the cause of addiction. With the establishment of addiction in the non-smoker network through both peer pressure to smoke and smoking, addiction in turn was perceived to cause factors that were perceived to lead to its emergence. There were marked differences in the patterning of this relationship between non-smoker and smoker networks with three more effects, stress, impulsivity, and peer pressure to smoke resulting from addiction in the non-smoker, but not the smoker, network.

While the analysis revealed many commonalities between non-smoker and smoker perceived causal structures, one of the most striking findings from this analysis is the difference between smokers and non-smokers in the perceived interconnections between cigarette addiction and the other causes. Smokers perceived less negative consequences of addiction relative to non-smokers who perceived addiction causing stress and impulsivity. The finding that smokers perceived less negative consequences with smoking behaviors was further confirmed in the follow-up t-test analyses of shared links in both networks that demonstrated a perceived greater likelihood of smoking leading to physical health problems and to addiction in non-smokers relative to smokers. The findings for the effects of parental smoking concur with the
general finding of increased negative effects of smoking in non-smoker responses. Parental smoking was perceived to lead to the experience of stress in children of parental smokers by non-smokers only. Differences between the non-smoker and smoker networks that are also striking are those in relation to peer pressure. Non-smokers perceived peer pressure as a salient cause of addiction, while smokers did not. Furthermore, peer pressure to smoke was perceived as significantly more likely to lead to smoking in non-smokers compared to smokers.

4.2. Implications

That individuals of different smoking statuses differ in their perceptions of the benefits and risks of smoking has been well established in the empirical literature (Brandon & Baker, 1991; Halpern-Felsher, Biehl, Kropp, & Rubinstein, 2004; McCoy et al., 1992; Copeland, Brandon, & Quinn, 1995). Indeed, successful prevention and intervention programs targeting smoking behavior have focused on restructuring beliefs related to the causes of smoking and addiction (Guichenez et al., 2007; Sussman et al., 2002). Networks such as those produced in this study could have implications for such programs. Providing an easily interpretable visual aid, the networks could be used to highlight beliefs that are perceived as relevant to intervention participants. The networks highlight potential areas in which an intervention with the current sample could be specifically catered to address.

In this case, the absence of a link between addiction and stress as well as between addiction and impulsivity in the smoker network highlights a potential area in which an intervention with the current sample could address. Research suggests that while many smokers believe that smoking reduces anxiety (Fidler & West, 2009; Kassel, Stroud, & Paronis, 2003), smoking may result in anxiety (Hughes, Gust, Kennan, & Fenwick, 1990; Parrott, 1999) and successful quit attempts result in reduced anxiety (McDermott, Marteau, Hollands, Hankins, & Aveyard, 2013). In terms of impulsivity, while smokers sometimes report increased concentration and cognitive control with smoking (Heishma, Taylor, & Henningfield, 1994; West,
with continued smoking there is evidence for increased drive to smoke (Bradley, Field, Mogg, & De Houwer, 2004; Buhler et al., 2010) as well as smoking-related cognitive deficits (Spinella, 2002; Ward, Swan, & Jack, 2001) which may act synergistically, to diminish the ability to inhibit impulses to smoke in the future (Bechara, 2005). Providing such information during an intervention in order to remedy the information deficit revealed by the consensual network could affect future health behavior and be perceived as a relevant intervention component by intervention participants. However, as these causal relationships are viewed as less salient than others in the current sample, content that emphasizes their roles in driving smoking behaviors to increase their perceived relevance may be desirable.

The finding of reduced salience of the perceived causal relationship between peer pressure and smoking in smokers relative to non-smokers suggests another avenue through which an information-deficit curriculum may be tailored for the current sample. Many smoking prevention programs provide information on the social influences of smoking, including information on how to resist peer pressure (e.g., Botvin & Griffin, 2004; Sussman et al., 2002). However, if the smoker sample does not view peers as a salient cause of smoking, as in the current case, the content may not be deemed relevant and, as a result, may be less effective at affecting smoking behavior. Again, content that emphasizes the effects of peers on smoking behaviors preceding the delivery of the social information curriculum might be desirable.

4.2. Limitations and future research
While the current study provides insight into the feasibility and reliability of network analysis to produce diagrams of the perceived causal structures of cigarette addiction, there were a number of limitations which could be improved upon in future studies. These include the use of a relatively homogenous sample consisting of undergraduate Psychology subjects. A key step for future research is to apply the method in more diverse samples, reflective of the samples recruited into interventions. Collecting smoking-related measures and recruiting a
larger sample of smokers in future studies would allow the examination of potential differences in perceived causal structures among different subtypes of smokers (Mayhew, Flay, & Mott, 2000; Shiffman, 1989).

Limitations of the network analysis method include the lack of guidelines for the establishment of cut-off points for network construction. The minimum 70% endorsement criterion and the selection of item average criterion may be deemed arbitrary and, thus, contested. However, the endorsement criterion of 70% used in the current study ensures the construction of highly consensual network diagrams representative of the majority of participants and the selection of the item average criterion reflects levels chosen in similar studies and is appropriate considering the high endorsement of many links by participants (Brogan & Hevey, 2009; Rafi et al., 2006). As this was an exploratory study, measurement construction relied heavily on causes identified by participants in the pilot study to ensure the networks constructed were reflective of lay beliefs. Future studies could tailor the grid method to incorporate causes of interest to the question at hand or causes that are relevant to the intervention aims. Considering the differences in perception of negative effects of addiction among smokers and non-smokers observed in the current study, future studies using this technique may benefit from including more putative positive and negative effects of addiction and smoking. Finally, while this study demonstrated the feasibility of the network analysis approach to examine smoking-related beliefs and provided preliminary evidence for the reliability of this method, it will be important for future research to further examine the reliability and validity of this method.
References


Lydon, D., Wilson, S., Child, A., & Geier, C. (under review). Adolescent brain maturation and smoking: what we know and where we’re headed.


Appendices

Appendix A: Figures and Tables

Figure 1. Network of the perceived causal structure of cigarette addiction for non-smokers. Continuous lines represent direct links to and from addiction. Dashed lines represent indirect links.
Figure 2. Network of the perceived causal structure of cigarette addiction for smokers. Continuous lines represent direct links to and from addiction. Dash lines represent indirect links.
Figure 3. Network of the perceived causal structure of cigarette addiction for time 1. Continuous lines represent direct links to and from addiction. Dashed lines represent indirect links.
Figure 4. Network of the perceived causal structure of cigarette addiction for time 2. Continuous lines represent direct links to and from addiction. Dashed lines represent indirect links.
Table 1

Mean strength of the causal links as rated by non-smoking participants

<table>
<thead>
<tr>
<th></th>
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<td>4.26</td>
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<td>2.54</td>
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<td>-</td>
<td>3.24</td>
<td>2.43</td>
<td>3.23</td>
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<td>3.29</td>
<td>-</td>
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<td>-</td>
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<td>8. Cigarette Addiction</td>
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<td>3.66</td>
<td>3.46</td>
<td>1.54</td>
<td>-</td>
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</table>

Notes: N = 82. The relationships are presented from cause (row) to effect (column).
Table 2

*Mean strength of the links entered into the non-smoker network and percentage of participants endorsing the entire network at an item average criterion of at least 4 per link*

<table>
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<th>Mean strength</th>
<th>Percentage at IAC ≥ 4</th>
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*Notes: N = 82.*
Table 3

*Mean strength of the causal links as rated by smoking participants*

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*Notes: N = 79. The relationships are presented from cause (row) to effect (column).*
Table 4

Mean strengths of the links entered into the smoker network and percentage of participants endorsing the entire network at an item average criterion of at least 4 per link

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<td>77</td>
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Notes: N = 79.
Table 5

*Number of incoming and outgoing connections by cause within smoker and non-smoker networks*

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<td>1</td>
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Table 6

_T-test table of comparisons between links common to smoker and non-smoker networks_

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<td>-0.13</td>
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<tr>
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<td>4.15</td>
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<td>0.81</td>
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<td>-0.27</td>
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<tr>
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<td>3.89</td>
<td>1.11</td>
<td>0.67</td>
<td>0.10</td>
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<td>4.41</td>
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<td>3.71</td>
<td>1.26</td>
<td>3.46</td>
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Notes: *Significant at Bonferroni corrected value of _p_ < .003.
Table 7

*Mean strength of the causal links as rated by participants at time 1*

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<th>6</th>
<th>7</th>
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<td>4.11</td>
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<td>2.06</td>
<td>0.91</td>
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</tr>
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<td>1.06</td>
<td>1.09</td>
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<td>2.37</td>
<td>2.20</td>
<td>1.23</td>
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<td>4.29</td>
<td>2.49</td>
<td>-</td>
<td>4.60</td>
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<td>0.77</td>
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<tr>
<td>4. Peer Pressure</td>
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<td>3.57</td>
<td>-</td>
<td>4.03</td>
<td>2.63</td>
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<td>5. Stress</td>
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<td>-</td>
<td>3.31</td>
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<td>-</td>
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*Notes: N = 35*
Table 8

*Mean strength of links entered into the time 1 network and percentage of participants endorsing the entire network at an item average criterion of at least 4 per link*

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<td>Addiction to Smoking</td>
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<td>4</td>
<td>Smoking to Physical Health Issues</td>
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<td>91</td>
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<td>Having Friends Who Smoke to Peer Pressure</td>
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<tr>
<td>6</td>
<td>Addiction to Having Friends Who Smoke</td>
<td>4.60</td>
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<td>7</td>
<td>Stress to Physical Health Problems</td>
<td>4.40</td>
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<tr>
<td>8</td>
<td>Having Friends Who Smoke to Smoking</td>
<td>4.29</td>
<td>91</td>
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<tr>
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<td>Smoking to Addiction</td>
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<td>89</td>
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<td>10</td>
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<td>4.11</td>
<td>89</td>
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*Notes: N = 35*
Table 9

*Mean strength of the causal links as rated by participants at time 2*

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<td>1.09</td>
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<td>1.20</td>
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<td>-</td>
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<td>4.46</td>
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*Notes: N = 35*
Table 10

*Mean strength of links entered into the time 2 network and percentage of participants endorsing the entire network at an item average criterion of at least 4 per link*

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<th>Percentage at IAC ≥ 4</th>
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<tr>
<td>2</td>
<td>Addiction to Physical Health Issues</td>
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<td>Physical Health Issues to Stress</td>
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<td>5</td>
<td>Cigarette Addiction to Having Friends Who Smoke</td>
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<td>6</td>
<td>Smoking to Addiction</td>
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<td>Having Friends Who Smoke to Peer Pressure to Smoke</td>
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<td>13</td>
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<td>Stress to Smoking</td>
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<td>16</td>
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<td>Being Impulsive to Smoking</td>
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<tr>
<td>20</td>
<td>Peer Pressure to Smoke to Addiction</td>
<td>3.11</td>
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</tbody>
</table>

*Notes: N = 35*
Appendix B: Perceived Causal Structure of Cigarette Addiction Measure

Perceived Causes of Cigarette Addiction

We are interested in what people believe are the causes and effects of cigarette addiction. In this booklet we would like you to consider how likely it is that potential causes lead to potential effects.

Here are definitions of the potential causes and effects:

**Smoking:** the act of smoking cigarettes

**Parental smoking:** at least one parent who smokes cigarettes

**Stress:** a state of mental or emotional strain or tension

**Physical health problems:** experiencing problems with one’s physical (as opposed to mental) health

**Peers:** people that belong to the same age group or social group

**Being impulsive:** acting on impulse before thinking things through

**Cigarette addiction:** being addicted to cigarettes means a person has formed a dependence on cigarettes that is hard to control.

*Please base your answers to the questions in this booklet on your own opinion*
1. Smoking

Q.1. How likely is it that smoking causes physical health problems?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.2. How likely is it that smoking causes cigarette addiction?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.3. How likely is it that a child who smokes causes their parents to smoke?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.4. How likely is it that smoking causes people to feel stressed?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.5. How likely is it that smoking causes people to experience peer pressure to continue smoking?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.6. How likely is it that smoking causes people to become friends with peers who smoke?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely

Q.7. How likely is it that smoking causes people to become impulsive?

Highly Unlikely | 0 | 1 | 2 | 3 | 4 | 5 | Highly Likely
2. Parental Smoking

Q.1. How likely is it that having a parent who smokes causes the child to experience physical health problems?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.2. How likely is it that having a parent who smokes causes the child to smoke cigarettes?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.3. How likely is it that having a parent who smokes causes the child to experience stress?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.4. How likely is it that having a parent who smokes causes the child to experience peer pressure to smoke?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.5. How likely is it that having a parent who smokes causes the child to become friends with peers who smoke?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.6. How likely is it that having a parent who smokes causes the child to become addicted to cigarettes?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.7. How likely is it that having a parent who smokes causes the child to be impulsive?

Highly Unlikely 0 1 2 3 4 5 Highly Likely
3. Stress

Q.1. How likely is it that feeling stressed causes physical health problems?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.2. How likely is it that feeling stressed causes smoking?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.3. How likely is it that feeling stressed causes people to experience peer pressure to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.4. How likely is it that feeling stressed causes people to become friends with peers who smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.5. How likely is it that a child’s stress causes that child’s parents to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.6. How likely is it that feeling stressed causes cigarette addiction?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.7. How likely is it that feeling stressed causes people to be impulsive?

Highly Unlikely  0  1  2  3  4  5  Highly Likely
4. Physical Health Problems

Q.1. How likely is it that physical health problems causes smoking?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.2. How likely is it that physical health problems causes people to experience peer pressure to smoke?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.3. How likely is it that physical health problems causes people to become friends with peers who smoke?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.4. How likely is it that a child experiencing physical health problems causes their parents to smoke?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.5. How likely is it that physical health problems causes cigarette addiction?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.6. How likely is it that physical health problems causes stress?

Highly Unlikely 0 1 2 3 4 5 Highly Likely

Q.7. How likely is it that physical health problems causes people to be impulsive?

Highly Unlikely 0 1 2 3 4 5 Highly Likely
5. Having Friends Who Smoke

Q.1. How likely is it that having friends who smoke causes people to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.2. How likely is it that having friends who smoke causes people to experience peer pressure to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.3. How likely is it that having a child with friends who smoke causes that child’s parents to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.4. How likely is it that having friends who smoke causes people to experience stress?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.5. How likely is it that having friends who smoke causes people to experience physical health problems?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.6. How likely is it that having friends who smoke causes cigarette addiction?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.7. How likely is it that having friends who smoke causes the people to be impulsive?

Highly Unlikely  0  1  2  3  4  5  Highly Likely
6. Experiencing Peer Pressure to Smoke

Q.1. How likely is it that experiencing peer pressure to smoke causes people to smoke?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.2. How likely is it that experiencing peer pressure to smoke causes people to become friends with peers who smoke?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.3. How likely is it that a child experiencing peer pressure to smoke causes that child’s parents to smoke?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.4. How likely is it that experiencing peer pressure to smoke causes physical health problems?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.5. How likely is it that experiencing peer pressure to smoke causes cigarette addiction?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.6. How likely is it that experiencing peer pressure to smoke causes people to feel stressed?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.7. How likely is it that experiencing peer pressure to smoke causes people to be impulsive?

<table>
<thead>
<tr>
<th>Highly Unlikely</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>
7. Cigarette Addiction

Q.1. How likely is it that cigarette addiction causes people to smoke?

Highly Unlikely

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<th>0</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.2. How likely is it that cigarette addiction causes people to become friends with peers who smoke?

Highly Unlikely

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<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.3. How likely is it that a child’s cigarette addiction causes that child’s parents to smoke?

Highly Unlikely

<table>
<thead>
<tr>
<th></th>
<th>0</th>
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<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.4. How likely is it that cigarette addiction causes physical health problems?

Highly Unlikely

<table>
<thead>
<tr>
<th></th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.5. How likely is it that cigarette addiction causes people to feel stressed?

Highly Unlikely

<table>
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<tr>
<th></th>
<th>0</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>

Q.6. How likely is it that cigarette addiction causes people to experience peer pressure to smoke?

Highly Unlikely

<table>
<thead>
<tr>
<th></th>
<th>0</th>
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<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
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</thead>
</table>

Q.7. How likely is it that cigarette addiction causes people to be impulsive?

Highly Unlikely

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>Highly Likely</th>
</tr>
</thead>
</table>
8. Being Impulsive

Q.1. How likely is it that being impulsive causes people to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.2. How likely is it that being impulsive causes people to become friends with peers who smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.3. How likely is it that being impulsive causes that child’s parents to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.4. How likely is it that being impulsive causes physical health problems?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.5. How likely is it that being impulsive causes people to feel stressed?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.6. How likely is it that being impulsive causes people to experience peer pressure to smoke?

Highly Unlikely  0  1  2  3  4  5  Highly Likely

Q.7. How likely is it that being impulsive causes cigarette addiction?

Highly Unlikely  0  1  2  3  4  5  Highly Likely