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The perceived causal structures of smoking: Smoker and non-smoker comparisons

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Abstract

Despite the detrimental impact of smoking on health, its prevalence remains high. Empirical research has provided insight into the many causes and effects of smoking, yet lay perceptions of smoking remain relatively understudied. This study used a form of network analysis to gain insight into the causal attributions for smoking of both smoking and non-smoking college students. The analyses resulted in highly endorsed, complex network diagrams that conveyed the perceived causal structures of smoking. Differences in smoker and non-smoker networks emerged with smokers attributing less negative consequences to smoking behaviors. Implications for intervention are discussed.

Keywords

beliefs, cause, health behavior, nicotine dependence, perception

Introduction

Despite the well-publicized evidence for the adverse health consequences of smoking, rates of cigarette use among adolescents remain high (Johnston et al., 2012). Furthermore, rates of smoking initiation at age 18 or older increased from 623,000 in 2002 to 1.1 million in 2012 (Substance Abuse and Mental Health Services Administration (SAMHSA), 2013). Empirical research has provided insight into the many causes of smoking behaviors (Lydon et al., 2014; Tyas and Pederson, 1998 for reviews), yet lay perceptions of the causes of smoking behaviors remain unclear.

Theoretical models emphasize the role of causal attributions, or people's explanation for the causes of behaviors or states, in influencing health behaviors (e.g. Ajzen, 1991; Leventhal et al., 1980). Causal attributions can be classified along three principal dimensions—locus (internal–external), controllability, and stability (Weiner, 1986). From this perspective, causal attributions have received the greatest interest in addiction research in the form of the abstinence violation effect (e.g. Curry et al., 1987; Shiffman et al., 1997), the phenomenon whereby abstinent

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drug users experiencing a lapse holding more internal and stable, potentially less controllable, causal attributions for their lapse are more likely to return to regular drug use than users who attribute the lapse to situational and temporary causes. Such attributions are thought to influence behavior by motivating efforts to change behavior, with perceptions that the causes of events reflect stable, uncontrollable causes, for example, undermining efforts to enact change (Sensky, 1997; Weiner, 1994).

The work investigating causal attributions for smoking has mostly focused on adults (e.g. Jenks, 1994; Kleinke et al., 1983) and early adolescents (e.g. Guo et al., 2010; Rugkasa et al., 2001), although one study has focused on young adults in the Navy (Cronan et al., 1991). Previous studies on causal attributions for smoking have observed many causal attributions for smoking, including peer pressure, having friends who smoke, and as a way of coping with negative affect (Cronan et al., 1991; Guo et al., 2010), with recent research demonstrating a role for causal attributions of smoking in the initiation and progression of smoking behaviors (Guo et al., 2012).

While limited studies have compared causal attributions among smokers and non-smokers, there is evidence for a self-serving attributional bias among smokers, with non-smokers being more likely than smokers to suggest that smoking causes detrimental outcomes such as health problems (Kleinke et al., 1983; Weinstein et al., 2005). The presence of other attributional biases seems to be less consistent. In line with the view in attribution theory that actors tend to explain their behaviors in terms of external factors while observers tend to attribute the same behavior to internal characteristics of the actors (Jones and Nisbett, 1972), researchers studying causal attributions in smoking have hypothesized that smokers will provide external attributions for smoking while non-smokers will provide internal attributions (e.g. Eiser et al., 1978; Kleinke et al., 1983).

While studies have provided some support for this hypothesis (Eiser et al., 1978; Kleinke et al., 1983), later work in smoking research (Jenks, 1994; Sadava and Weithe, 1985), and in attribution research more generally (Malle, 2006), has not supported the presence of an actor–observer bias. In contrast, more recent theory suggests that labeling one's drug use as resulting from internal (e.g. addiction) causes may be self-serving as it implies that drug use is a relatively uncontrollable condition, thus removing personal responsibility, diminishing blame, and protecting self-esteem (Davies, 1997; Monk and Heim, 2011).

Few studies have focused on young adult, college-aged students (although see Cronan et al., 1991 for an exception). This is a limitation given the recent increases in smoking initiation observed during this period. Furthermore, all studies to date that examined smoking attributions focused on single, cause–effect relationships. For example, a study examining the effects of smoking beliefs on smoking onset created an aggregate item focusing on the perceived effects of smoking (cause) on health (effect) using multiple items assessing the perceived effect of smoking on various health consequences, for example, "If I smoke cigarettes, I will get lung cancer" (Krosnick et al., 2006: S35). An alternative way of conceptualizing causal attributions is to contextualize single, cause–effect relationships within a broader attribution network that explicitly models the interconnections between multiple causal relationships. This conceptualization of causal attributions was advocated in early work on causal attribution with Kelley (1983), for example, suggesting that a person's understanding of an event is based on the perceived location of the event within a temporally ordered network consisting of interconnected nodes termed the perceived causal structure. The need to distinguish between simple attributions, involving single cause–effect relationships, and complex attributions, involving interdependence between multiple cause–effect relationships, with regard to health behaviors has been urged by Sensky (1997), who suggested that even the simplest models of illness and treatment are likely to involve complex, interdependent attributions.

Alongside these theoretical considerations, network analysis techniques have emerged and have been employed to provide insight into lay perceptions of the causes of a range of health-related outcomes including coronary heart disease (Green and McManus, 1995) and obesity (Brogan and Hevey, 2009). Information on the extent (proximal vs distal causes) and patterning (simple vs complex connections) of the causes can be gleaned from these methods (Brogan and Hevey, 2010). The analyses also results in group-level representations of the perceived causal belief structure that may act as convenient visual aids for cognitive structuring. Studies employing these methods have highlighted the complex representations of health behaviors held by the participants (e.g. Campbell and Muncer, 2005; French et al., 2002) confirming earlier theoretical work.

This study employed a variation on the grid method of network analysis, the most commonly used method of network analysis employed in studies of perceived causal structures of health beliefs (e.g. Brogan and Hevey, 2009; Gillen and Muncer, 1995), to determine the perceived causal structures of smoking. In this 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 the strength of the causal relationship between each putative cause and the other causes (i.e. its effects) on a Likert scale (Rafiq et al., 2006). A network is then constructed from the participant input using a form of inductive eliminative analysis (Muncer and Gillen, 1997) in which 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. This study replaced the input grid with a series of questions which elicited responses to every possible combination of links between potential causes (French et al., 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.

This exploratory study aimed to generate the perceived causal structures of smoking. Another aim was to determine whether the perceived causal structures differed across smoking statuses. Given the tendency for non-smokers to rate more causes than smokers (Stanton et al., 1993), smoker causal networks were hypothesized to be sparser (containing less links) than non-smoker networks. In line with attribution theory as well as previous research (e.g. Kleinke et al., 1983), it was hypothesized that smokers would demonstrate a self-serving bias in their attributions, primarily in the causal relationships between smoking and undesirable outcomes such as health problems. Given the conflicting results of the presence of other attributional biases on internal and external causes of addiction (e.g. Jenks, 1994; Kleinke et al., 1983) as well as research suggesting that internal attributions for drug use may play a self-serving function for drug users (Monk and Heim, 2011), it was expected that no simple internal versus external pattern of attributions for smoking would be evident across smokers and non-smokers as might be expected by the actor–observer bias phenomenon. Given the public health significance of the rise in young adult smoking in recent years, this study was undertaken in a young adult sample of college students.

Method

Participants

Upon receipt of ethical approval, a sample of 161 participants was recruited from the Psychology subject pool at a large university in the Northeast of the United States. A total of 82 non-smokers (24 males) were defined as participants who 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 years ($M = 18.76$, standard deviation (SD) = 1.12). A total of 79 smokers (30 males) were defined as participants who had smoked at least 100 nicotine cigarettes in their lives and self-reported as current daily ($N = 28$) or

someday ($N=51$) smokers. Smokers ranged in age from 18 to 24 years ($M=19.56$, $SD=1.46$).

Participants undertook the study in a research laboratory setting. Upon entering the laboratory, participants read and signed an informed consent form. Participants then completed a questionnaire developed from the pilot study documented in the "Measures" section, as well as a questionnaire detailing their smoking status.

Measures

Questionnaire development. Semi-structured interviews on the topic of smoking were conducted with four participants recruited from the Psychology subject pool. The results of these interviews, as well as a review of the literature, generated salient causal attributions for smoking, from which eight were selected for inclusion in the questionnaire. Given the exploratory nature of the study, the selection process aimed to produce a wide range of salient causes that spanned numerous biopsychosocial domains. The causes and their definitions were as follows: (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 health; (5) *peer pressure*—influence from people who 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.

Questionnaire. This study used a series of questions that elicited responses to every possible combination of links between potential causes (French et al., 2002). 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 cigarette addiction as the effect is, "How likely is it that smoking causes cigarette addiction?" An example of a question with cigarette addiction as the cause and smoking as the effect is, "How likely is it that cigarette addiction causes smoking?" In total, the method allowed the consideration of 56 links.

Data analysis

Separate networks were created for non-smokers and smokers. For each network, the mean score for each of the 56 links was computed. The links were ranked by mean endorsement. Each link was added to the network in a hierarchical fashion, with the highest rated links 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 (IAC) was adopted 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 IAC was set at 4 on the 0–5 scale (e.g. 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 IAC. To further ensure the resulting networks could be deemed consensual, network construction stopped when network endorsement at the IAC was not achieved by at least 70 percent of participants. Multidimensional scaling was used to determine the spatial structure of the network. Finally, *t*-tests were used to test differences in shared links between smoker and non-smoker networks.

Results

Non-smoker network

The mean strengths of the causal links are presented in Supplemental Table 1. Multidimensional scaling of the 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. Using inductive eliminative analysis,

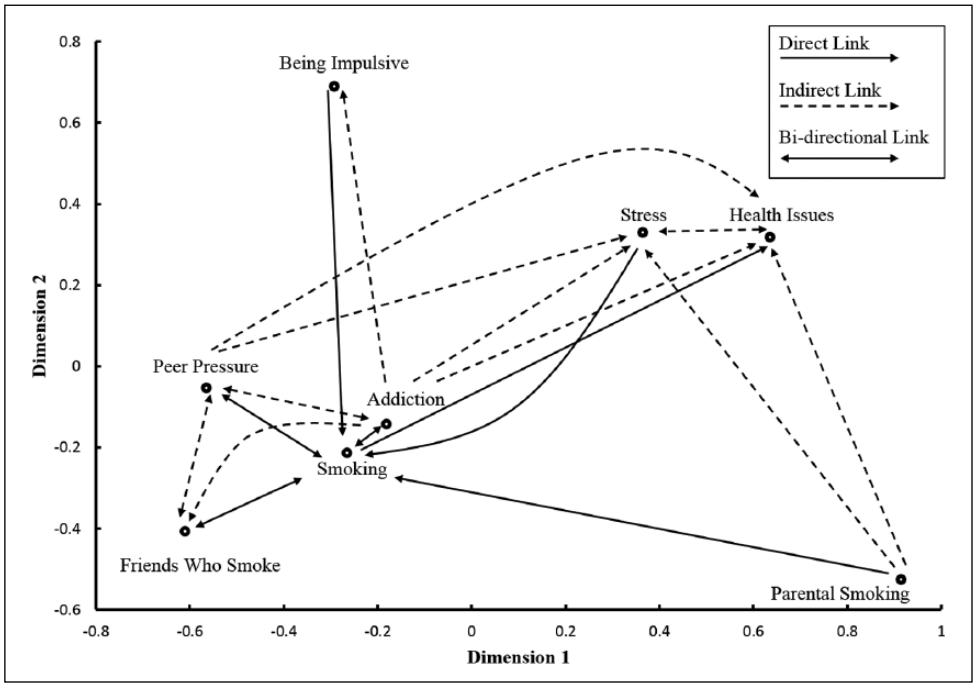

Figure 1. Network of the perceived causal structure of smoking for non-smokers. The placement of the causes was determined by multidimensional scaling and provides information on the perceived proximity of the causal relationships. Direct links (links containing smoking as a cause or an effect) are differentiated from indirect links (links not containing smoking as a cause or effect) to increase interpretability of the figure.

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 percent of participants at a rating of 4 and above. In total, 24 links were added to the network (Figure 1). Adding link 25, *impulsivity* to *stress*, would have resulted in a drop in network endorsement from 74 to 68 percent (Supplemental Table 2). Thus, the network constructed stopped when link 24 was added.

Smoker network

The mean strengths of the causal links are presented in Supplemental Table 3. Multidimensional scaling of the 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. 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 percent. In total, 17 links were added to the network (Figure 2). Adding link 18, *peer pressure* to *addiction*, would have resulted in a drop in network endorsement from 71 to 68 percent (Supplemental Table 4). Thus, network construction stopped after link 17 was added.

Non-smoker and smoker network comparison

Smoker and non-smoker networks shared 17 links. Seven links unique to the non-smoker network included the following: *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*.

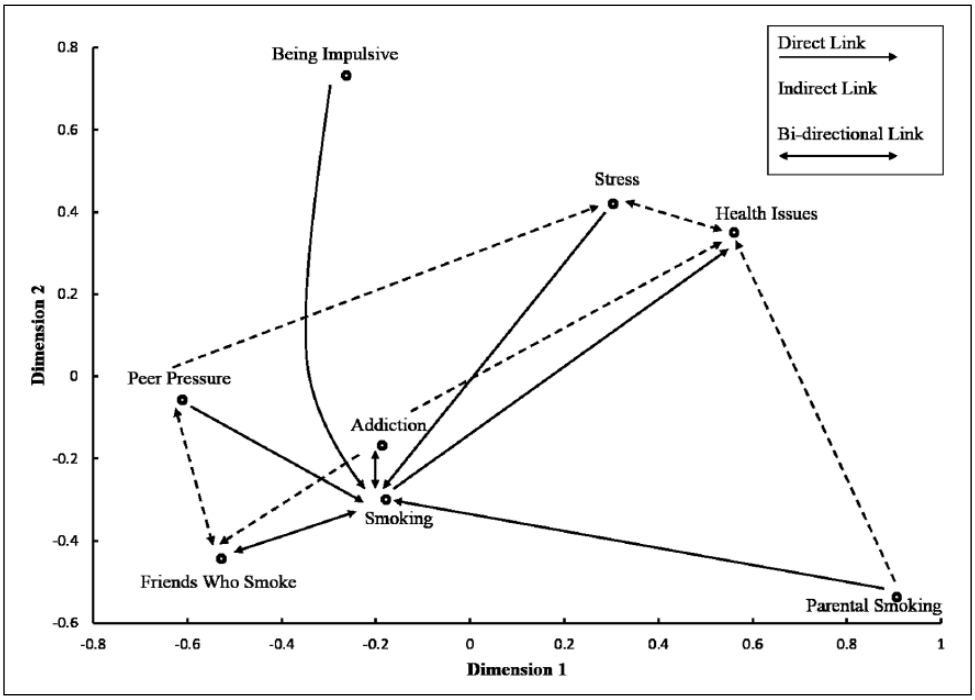

Figure 2. Network of the perceived causal structure of smoking for smokers. The placement of the causes was determined by multidimensional scaling and provides information on the perceived proximity of the causal relationships. Direct links (links containing smoking as a cause or an effect) are differentiated from indirect links (links not containing smoking as a cause or effect) to increase interpretability of the figure.

The mean strengths of shared links were compared using independent samples *t*-tests. 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,* $t(119.26)=3.91$, $p<.001$, $d=0.62$; *smoking* to *addiction,* $t(134.08)=4.17$, $p<.001$, $d=0.66$; *peer pressure to smoke* to *smoking,* $t(159)=4.57$, $p<.001$, $d=0.73$; *peer pressure to smoke* to *stress,* $t(159)=3.46$, $p=.001$, $d=0.55$.

Discussion

This study is the first application of network analysis to elicit the perceived causal structure of smoking. The method provided a rich representation of causal attributions with distal causes (e.g. *being impulsive; parental smoking*) of smoking emerging in both non-smoker and smoker networks that operated through more proximal causes (e.g. *addiction*) of smoking. Complex temporal orderings also emerged in the networks. For example, in both networks, *parental smoking* was perceived to cause *smoking* which in turn caused *health issues* which operated through *stress* to cause *smoking*. Both stable (e.g. the personality trait of *being impulsive*) and temporary (e.g. the state of *stress*) processes emerged in both networks. Both external (e.g. *peer pressure* and *parental smoking*) and internal (e.g. *addiction* and *being impulsive*) attributions were present in both networks.

While the analyses revealed many commonalities between non-smoker and smoker perceived causal structures, a number of differences

in smoking attributions emerged among the two groups. At the gross network level, non-smokers endorsed more causal links than smokers. This is similar to comparisons of smoker and non-smoker attributions for smoking in a previous study among early adolescents in which non-smokers were more likely to endorse more causes of smoking (Stanton et al., 1993). This relative sparseness of links for smokers may result from a self-serving bias. This interpretation is plausible given that three of seven links absent from the smoker network may be conceived of as undesirable consequences of smoking and addiction: *addiction* causes *stress, addiction* causes a person to *be impulsive*, and *parental smoking* causes *stress*. The finding that smokers attributed less negative consequences with smoking behaviors was further confirmed in the follow-up *t*-test analyses of shared links in both networks. Results demonstrated a perceived greater likelihood of *smoking* causing *physical health problems* and *addiction* in non-smokers relative to smokers.

While this pattern of findings has been described in previous research as a self-serving bias (Kleinke et al., 1983; Weinstein et al., 2005), an alternative, though not mutually exclusive, interpretation of these findings is provided by cognitive dissonance theory (Festinger, 1957). Despite agreeing that smoking is causally implicated in serious health outcomes, many smokers continue to smoke, creating a discrepancy between their behavior and their beliefs (McMaster and Lee, 1991). This discrepancy is theorized to lead to dissonance, an unpleasant psychological state, which individuals are motivated to reduce by altering their beliefs to justify their behavior. Evidence for cognitive dissonance in smokers has been reported in previous studies (e.g. Fotuhi et al., 2014) and may be reflected in the fewer negative causal links observed in the smoker relative to non-smoker networks in this study.

The extent to which a self-serving bias or cognitive dissonance in smokers can explain the absence of four of the seven links, all relating to peer pressure, is unclear. These links are qualitatively different as they may carry less of the negative connotations and less of a discrepancy between the knowledge that smoking leads to poor health outcomes and continued smoking behavior, although they may reflect an unwillingness to admit to being coerced into smoking by their peers. Alternatively it may be due to smokers' actual experiences. It is a widely held assumption that people initiate smoking in response to peer pressure, yet this may not reflect actual experience, with peer smoking rather than peer pressure influencing young adult smoking (Harakeh and Vollebergh, 2012). Indeed, while the role of *peer pressure* in causing *smoking* was not salient to smokers, *having friends who smoke* was identified as a salient cause.

In terms of the actor–observer attribution bias, smokers and non-smokers gave identical attributions for *smoking* with both internal (e.g. *addiction* and *being impulsive*) and external (e.g. *peer pressure* and *parental smoking*) causes present in both networks. The only difference in perception of internal versus external attributions that emerged was the perception that *peer pressure*, an external attribution, was perceived to be a greater cause of *smoking* in the non-smoker network. These findings are not in line with the actor–observer bias, but they are similar to previous findings (e.g. Sadava and Weithe, 1985) and in line with suggestions that internal attributions in drug use may be self-serving, absolving the drug user of personal responsibility for his or her actions by implying that drug use is a relatively uncontrollable condition (e.g. Monk and Heim, 2011).

Given that smokers did not emphasize internal causes of smoking to a greater extent than non-smokers, alternative explanations for the current pattern of results may be necessary. A consideration of the larger perceived causal structure rather than single cause–effect relationships may give context to these findings. While it may be expected based on previous literature that smokers may attribute their smoking to internal causes to absolve themselves of blame, the lower endorsement of negative consequences of smoking and addiction previously described in the smoker network suggests that

smokers do not consider smoking to be as detrimental as non-smokers. As a result, smokers in this study may not experience the same motivation to emphasize uncontrollable attributions such as addiction in order to excuse their behaviors, resulting in similar internal and external attributions in both smoker and non-smoker networks. This is in line with suggestions that the attribution of drug use behaviors to addiction is more likely when drug use is deemed problematic, thus requiring an explanation of uncontrollability to assuage feelings of guilt (Davies, 1997).

Furthermore, longitudinal research will be required to determine the development of these differences in causal attributions in smokers and the extent to which they may relate to attribution biases. Despite this need for future research, networks such as those produced in this study could have implications for programs targeting smoking behaviors that focus on restructuring beliefs related to the causes of smoking and addiction (Lai et al., 2010; Sussman et al., 2002). Providing a visual aid, the networks may guide the tailoring of program content in response to the beliefs of program participants.

In this case, for example, the absence of a link between *addiction* and *being impulsive* in the smoker network may be a potential area in which an intervention with the current sample could be specifically catered to address. While smokers sometimes report increased concentration and cognitive control with smoking (e.g. Heishma et al., 1994), with continued smoking there is evidence for increased drive to smoke (Bradley et al., 2004) as well as smoking-related cognitive deficits (Spinella, 2002) which may act synergistically, to diminish the ability to inhibit impulses to smoke in the future (Bechara, 2005). However, biased causal attributions are not necessarily dysfunctional. Indeed, biased attributions may result in better health outcomes under certain conditions (Sensky, 1997), and thus, longitudinal studies involving both smokers and non-smokers will be required to identify perceived causal structures that render non-smokers vulnerable to smoking initiation and smokers less likely to quit.

This study demonstrated the feasibility of using network analysis to generate perceived causal networks of smoking in both smokers and non-smokers. In line with previous studies, differences emerged in non-smoker and smoker networks, especially in terms of the self-serving attribution bias. This study extended this literature by examining causal attributions in college-aged smokers, a population with limited exploration in previous studies on causal attributions of smoking. Collecting more detailed smoking-related information and recruiting a larger sample of smokers in future studies will also allow the examination of potential differences in perceived causal structures among different subtypes of smokers (e.g. Mayhew et al., 2000). A key direction for future research will be to examine the perceived causal structures longitudinally and to investigate their effects on smoking behaviors.

Declaration of conflicting interests

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