Smoking at the workplace: effects of genetic and environmental causal accounts on attitudes toward smoking employees and restrictive policies

Ilan Dar-Nimrod\(^{a*}\), Miron Zuckerman\(^{b}\) and Paul Duberstein\(^{c}\)

\(^{a}\)School of Psychology, University of Sydney, Sydney, NSW, Australia; \(^{b}\)Department of Psychology, University of Rochester, Rochester, NY, USA; \(^{c}\)Department of Psychiatry, University of Rochester Medical Center, Rochester, NY, USA

(Received 13 November 2013; final version received 27 June 2014)

People hold diverse beliefs regarding the etiologies of individual and group differences in behaviors which, in turn, might affect their attitudes and behaviors. It is important to establish how perceived etiologies of smoking might affect the effectiveness of policy initiatives and prevention efforts. The present study assessed whether exposure to genetic vs. environmental accounts for smoking affects attitudes toward (a) workplace-related smoking policies and (b) smokers at the workplace. Results indicate that exposure to a genetic explanation led to stronger objections to a smoking restrictive policy compared with a non-genetic explanation. Additionally, participants in the genetic condition were more accepting of a smoker in the workplace than those in the environmental condition. Evidently, beliefs about the etiology of smoking influence a range of attitudes related to smokers and smoking-related policies.

**Keywords:** public opinion; public policy; workplace; etiology

**Introduction**

With more than a billion smokers worldwide, smoking is considered to be the leading behavioral cause of mortality globally by the World Health Organization (WHO), with a myriad of recent worrying indicators such as a single country, China, reporting over a million annual smoking-related deaths for 2012 (WHO 2012). Meta-analytic-based estimations indicate that about 1 out of 10 deaths is associated with smoking-induced diseases, a ratio that is expected to rise in the next decades (Mucha \textit{et al}. 2006).

In an attempt to address this global health threat, researchers have identified environmental factors that affect smoking initiation and behavior including advertising (Weiss \textit{et al}. 2006), peer group smoking (Urbán 2010), lower socioeconomic...
status (Reid et al. 2010), and modeling (Wen et al. 2005; Bricker et al. 2007). Research involving twins (Rende et al. 2005), adoption (Hopfer, Crowley, and Hewitt 2003), and molecular genetics (Munafo et al. 2009; Philibert et al. 2009) indicates that genes also contribute to whether or not people smoke.

The identification of etiological underpinnings for smoking plays a central role in interventions and policies directed at reducing smoking-related behaviors. For example, consider cigarette advertising, which had a substantial market share during the 1980s, accounting for 22.3% of total outdoor advertising in the USA in 1985 (Davis 1987). As cigarette advertising was implicated in the initiation of smoking (Pierce et al. 1991; Pierce et al. 1998), restrictive cigarette advertising policies were introduced in many countries. Subsequently, a number of studies documented the effectiveness of advertising bans and restrictions (Pekurinen 1989; Smee 1992).

The identification of environmental elements (such as advertising) that affect smoking behavior offers a target for policies to bring about smoking reduction. Similarly, incorporating knowledge of genetic factors to reduce tobacco consumption has been considered in discussions of smoking cessation initiatives as well (Gramling et al. 2003; Pomerleau et al. 2007).

In general, public opinion surveys indicate an increase in genetic attributions for various phenomena in the past few decades. For example, Sheldon et al. (2007) used public opinion polls to suggest a recent significant increase in endorsements of genetic explanations for homosexuality. Notably, such increases occurred despite the lack of strong scientific evidence for the genetic etiology of homosexuality. In one instance, Sheldon et al. (2007) compared two Gallup poles, indicating that “the percentage of individuals who expressed the belief that homosexuality is something people are ‘born with’ more than tripled, from 13 in 1977 to 40 in 2001” (114). A similar effect was found for the etiological perception of body weight. For example, Singer, Corning, and Lamias (1998) report that in 1979, 36% of respondents indicated that they believe heredity to be more important than environment in determining whether a person is overweight. In contrast, by 1995, 63% of the respondents perceived that being substantially overweight is largely determined by genes (Singer, Corning, and Lamias 1998).

The aforementioned changes in public perception of genetic etiology have been part of growing geneticization, the process in which people have become more likely to use genetic explanations for a range of phenomena (Lippman 1991; Richards 2010; Deister 2013). Thus, it is informative to evaluate whether the general public, particularly that portion of the public that smokes, also considers genetic attributions for smoking. If there is evidence for such consideration, it is important to evaluate its implication regarding attitudes toward smokers and smoking behavior.

Particular genes have been implicated in a variety of smoking-related phenomena, from smoking initiation and maintenance (Bierut et al. 2007; Philibert et al. 2009) to susceptibility to smoking-related disease among smokers.
In contrast to homosexuality and obesity, few people might be presumed to readily associate smoking with genetics (Houfek et al. 2008). Nonetheless, only a minority of smokers (35%) completely rejects genetic attributions for smoking (Wright et al. 2011). Furthermore, evidence suggests that in the presence of a family history for smoking, smokers are increasingly perceived as carriers of addiction genes that predispose them to smoking (Cappella et al. 2005).

Research suggests that once people are exposed to even a small amount of information that alludes to genetic correlates of smoking-related phenomena, a majority tend to endorse genetic attributions. For example, a survey among healthy adolescents (13–21 years old) attending general medical check-up showed that a majority (62%) of these individuals indicated at least a fair amount of interest in undergoing a nicotine addiction genetic susceptibility test when available, with only 16% showing no interest at all (Tercyak et al. 2006). This finding indicates an underlying belief in genetic etiology of nicotine addiction in this relevant population.

Learning about genetic attributions affects more than just a shift in etiological perceptions. For example, smokers show interest in personal tests for genetic susceptibility to smoking-related diseases when they learn of a potential connection between specific genes and such diseases (McBride et al. 2002; Hishida et al. 2010). A recent meta-analysis indicated that 60–80% of smokers show interest in such a test (Smerecnik, Grispen, and Quaak 2012). These results underline the important role of people’s genetic attributions for smoking-related phenomena.

Research on genetic underpinning of smoking-related phenomena has received attention in the public sphere as well. For example, a recent article that appeared in the Los Angeles Times (Roan 2011) quotes a leading author of a scientific journal paper as saying that anti-smoking laws and policies “may be effective in prodding social smokers with genetic resilience to quit but may do less to help genetically vulnerable smokers quit.” A similar coverage of the genetics of smoking and smoking-related diseases has appeared in other newspapers (The Associated Press, 2008). The Washington Post (2008), for instance, reported on a press release by the American Cancer Society, which indicated that researchers have identified common alleles tied to individuals with lung cancer. This press release ended on a promising note: “(b)ecause tobacco smoking is the leading preventable cause of cancer and the cancer-prone genotypes of these genetic components are relatively prevalent in the human population, our findings have important implications for the prevention of tobacco smoking-related cancers.”

Such optimism in media reports of genetic research has been discussed at length elsewhere (Nelkin and Lindee 1995; Dar-Nimrod and Heine 2011a). This “genetic optimism” (Conrad 2001) is also commonplace in genetic-focused scientific articles (Gramling et al. 2003; Pomerleau et al. 2007).

However, the introduction of genetic etiological findings to smoking cessation initiatives might lead to cognitive biases that can operate both implicitly (Gould and Heine 2012) and explicitly (Nordgren and Juengst 2009; Dar-Nimrod and
Heine 2011a). When outcomes are viewed as having a genetic etiology, they are seen as more deterministic, immutable, and natural (Dar-Nimrod and Heine 2011a). Indeed, correlational research indicates that an increase in endorsement of genetic etiological explanations is associated with more prejudiced views of various social groups based on race, ethnicity, and gender (Keller 2005; Jayaratne et al. 2006). Within the health realm, endorsing such explanations for disease is associated with an increase in perceived disease severity (Senior, Marteau, and Peters 1999) and decrease in the perceived benefit from treatment (Phelan, Cruz-Rojas, and Reiff 2002). However, correlational studies are often limited in the casual inferences that can be drawn, necessitating experimental designs.

Similar to the correlational studies, experimental research indicates that exposure to narratives that promote genetic explanations leads to a slew of essentialism-related effects, including stronger ethnic out-group aversion (Keller 2005), increased gender stereotyping (Brescoll and LaFrance 2004), reduction in intentions to exercise (Beauchamp et al. 2011), more acceptance of sexual crimes among men (Dar-Nimrod et al. 2011), and impaired women’s performance on spatial reasoning (Moë and Pazzaglia 2010) and math (Dar-Nimrod and Heine 2006). These latter findings have been explained by invoking the construct of perceived control. When any of these phenomena are given a genetic etiology, they are perceived as immune, in part or wholly, to personal control. As such, the perception of genetic etiology has far-reaching implications for smoking cessation and policies.

Genetic essentialist biases reduce perception of behavioral control among individuals who endorse them or are merely exposed to them (Dar-Nimrod and Heine 2011a; Dar-Nimrod and Lisandreli 2012; Dar-Nimrod, Zuckerman, and Duberstein 2013). The Theory of Planned Behavior (Ajzen 1991) and the Self-Regulation Model (Leventhal, Brissette, and Leventhal 2003), two empirically supported behavioral theories utilized extensively in the health field (Armitage and Conner 2001; Hartjes and Baumann 2012; Trumbo and Harper 2013), predict that reduction in perceived behavioral control decreases the likelihood that a relevant behavior will be enacted. This prediction was supported among smokers in that perception of reduced ability to control smoking behaviors (1) was associated with increases in both future smoking intentions and current smoking in a cross-sectional sample of adolescents (ter Doest et al. 2009); (2) predicted increased smoking initiation behavior longitudinally among adolescents (Armitage 2003); and (3) predicted reduced quit attempts longitudinally among young smokers (adults) who held a negative attitude about smoking (Kovac and Rise 2008). Another study (Wright et al. 2007) showed that, among smokers enrolled in a smoking cessation trial, those who endorsed genetic etiological explanations for smoking perceived lower behavioral control over their smoking compared with those who rejected genetic accounts. However, in this study, the decrease in perceived control was not associated with the quit rate. Although recent research suggests that the association between genetic etiological and reduced perception
of behavioral control are not always replicable (McClure et al. 2013), this study was arguably severely underpowered ($N = 36$) to make such a claim. Despite the increased attention to genes’ role in smoking behaviors, the effects of smoking etiological beliefs on smoking control policies and attitudes toward smokers have not been studied. Given the documented effects of etiological perceptions on attitudes, behavioral intentions, and actual behaviors, one would expect that etiological beliefs would contribute to evaluations of smokers and tobacco control policies. The present study explores these effects among a young population of college students. Because of the interest in policies and regulations designed to reduce smoking on the job or in proximity to the workplace (Lu et al. 2011; McDaniel and Malone 2012; McClure et al. 2013), this study focused on the work environment.

**Methods**

**Sample**

One hundred and thirteen undergraduate students from a northeastern American university (88 women; ages 18–35 years, $M_{age} = 20.30$, $SD = 2.2$) participated in exchange for course credit. The sample contained no regular smokers (i.e. people who smoke a cigarette a day or more).

**Procedure and measures**

Participants were invited to take part in a study on evaluations of smoking. Upon arrival, they were randomly assigned to read one of three excerpts from real articles published in the *New York Times*. One excerpt discussed psychological and genetic explanatory factors for smoking (genetic condition). A second focused on the effects of exposure to smoking on television and parental supervision on adolescence smoking (environmental condition). A third excerpt made no reference to any etiological explanation, focusing instead on health risks associated with smoking (control condition). After reading the excerpt, participants answered a few multiple-choice questions about the excerpt, the last of which served as a screening device, ensuring that the participants read the article (“what is one of the main themes discussed in the article?”).

Next, participants read the following vignette:

George has been working for the Monroe County Library Branch in Brighton for 5 years. Recently, George, who has been smoking for 8 years, was asked to abstain from smoking throughout his entire work day because the library patrons complained about the “stale smoking smell” that he emits every time he comes back from a smoking break. George was surprised and hurt as he always made sure his breaks were similar to those of other employees who did not smoke. He thought that because his smoking was a known habit, there should not be such demands placed after 5 years.
After reading the vignette, participants replied to three questions revolving around the evaluation of the library’s new policy toward smoking during work time. Specifically, these questions assessed the extent to which the policy is (1) justified (reversed scored), (2) invasive, and (3) discriminatory. Each question was answered on a four-point rating scale with higher scores indicating stronger objection to the policy. The ratings were averaged to yield a single score (Cronbach’s $\alpha = .69$). In addition, participants’ general attitudes toward a smoker at the workplace were evaluated using two questions that assessed whether participants (1) object to having George (a smoker) working in a library where they take their children, and (2) object to having George as a coworker. Two similarly phrased questions asked participants to speculate on their family and friends’ responses to the general attitude questions. Participants indicated their responses on four-point rating scales with higher scores indicating increased objection. The ratings were averaged to reflect participants’ overall attitude toward a smoker ($\alpha = .82$).

After the participants completed answering all these questions, they provided information about their gender, age, and ethnicity.

Results

Eighteen participants (eight women) were removed from the analyses because of failure to identify the main theme of the New York Times article they read, indicating a lack of adequate exposure to the manipulation. This left a final sample of 95 participants. Gender, age, and ethnicity did not significantly affect the results and will not be discussed further.

Library policy evaluations

An independent-samples $t$-test indicated that individuals exposed to the genetic account [$n = 27, M (SD) = 2.69 (0.61)$] perceived the new smoking policy as more objectionable than individuals who read the environmental explanation [$n = 31, M (SD) = 2.26 (0.37)$], $t(56) = 3.33, p = .002$, Cohen’s $d = 0.89$. To explore which of these evaluations most closely resembled people’s responses when they did not consider etiology, an analysis of variance (ANOVA) was conducted, which included the control condition (Figure 1). The results showed a significant effect for the excerpts’ content [$F(2, 92) = 4.99, p = .009$, $\eta^2 = .098$]. Follow-up Tukey’s post hoc tests revealed that the policy evaluations in the control condition [$n = 37, M (SD) = 2.66 (0.73)$] were more similar to the ones in the genetic condition ($p = .97$) than to the ones in the environmental condition ($p = .02$).

Personal and normative attitudes: An independent-samples $t$-test indicated that participants in the genetic condition [$M (SD) = 2.24 (0.60)$] had weaker personal and normative objections to having the smoking protagonist as a coworker or as a service provider at a library that serves children than individuals in the
environmental condition \[ M(\text{SD}) = 2.53 (0.42) \], \[ t(56) = -2.17, \ p = .034 \], Cohen’s \( d \) = 0.58. An ANOVA was conducted to explore which of these attitudes reflects the responses in the control condition. The excerpts’ content had no significant effect once the control condition was included \[ F(2, 92) = 1.98, \ p = .144, \ \eta^2 = .041 \]. An inspection of the data revealed that once again the responses in the control condition \[ M(\text{SD}) = 2.31 (0.70) \] were more similar to the responses in the genetic condition; however, the lack of significant results does not allow for further interpretation of these findings.

**Discussion**

There is much evidence for geneticization for varying phenomena (Nelkin and Lindee 1995; Singer, Corning, and Lamias 1998; Sheldon et al. 2007; Wright et al. 2007; Dar-Nimrod and Heine 2011a; Smerecnik et al. 2011). Media articles, many of which appear in leading American newspapers such as the New York Times, Los Angeles Times, and Washington Post (Roan 2011; The Associated Press 2008; Washington Post 2008), have consistently reported research findings connecting genes and smoking-related phenomena. Extensive research indicates that the news media exerts much of its influence on public attitudes and behavior by priming specific cognitions and closely related constructs (Price and Tewksbury 1997; Valentino, Traugott, and Hutchings 2002; Cappella et al. 2005). Given the
exposure to media accounts, it is not surprising that previous research indicates that
individuals readily accept genetic attributions for smoking-related phenomena
(Tercyak et al. 2006; Wright et al. 2011; Smerecnik, Grispen, and Quaak 2012). The present study did not investigate the underlying etiological factors for smoking. Instead, it set out to examine the potential effects of exposure to genetic (compared to environmental) attributions on attitudes toward smoking at the workplace and its consequences.

The study’s findings indicate that compared with exposure to an environmental etiological account for smoking, exposure to a genetic explanation generates more accepting attitudes toward a smoker as a coworker or a service provider, and increases objections for restrictive smoking policies at the workplace. Interestingly, the evaluations of a relevant public policy after exposure to the genetic account were similar to the evaluations of individuals who were unprompted with any etiological account. These findings imply that participants who received no information about the etiology of smoking would not have been affected much by the introduction on the genetic explanation for smoking; in other words, they already perceived smoking to be an immutable behavior. On the other hand, participants who were exposed to an environmental etiological account for smoking were less forgiving toward smoking coworkers at the workplace and more strongly supportive of restrictive policies.

Smoking is not a context-free phenomenon. It is part of a larger network of related constructs revolving around substance-based addiction (Haylett, Stephenson, and Lefever 2004). Public perceptions of the role of heredity as an explanation for addiction in the modern era can be traced back to Sir Francis Galton and his eugenic perspective on “drunkards” (Galton 1869). More recently, public opinion polls have indicated that people readily make genetic attributions for various addictions. Singer, Corning, and Lamias (1998) reported that 33% of the public at the time attributed alcoholism completely or mostly to one’s genes, whereas 20% dismissed the role of genes in such addiction altogether (similar results were reported for drug abuse). A later survey indicates that from 1996 to 2006, there was a 10% increase in the American public genetic attributions for alcohol dependence (Pescosolido et al. 2010). Such results not only indicate that most people seem to assign causal roles to both genes and other factors when it comes to substance-based addiction, in line with scientific findings (Agrawal and Lynskey 2008), but also indicate a trend for increased focus on genetic causes for addiction (Jayaratne and Gaviglio 2012).

As part of this larger context of addiction in general (Conrad and Weinberg 1996; Keller 2005; Jayaratne and Gaviglio 2012), and smoking in particular (Wright et al. 2007; Kovac and Rise 2008; ter Doest et al. 2009), genetic explanations have been commonly interpreted as indicative of reduced malleability and decreased controllability of the associated behaviors (Dar-Nimrod and Heine 2011a). In line with these assertions and the present findings, the Attribution Theory (Weiner, Perry, and Magnusson 1988) makes predictions with regard to evaluations of individuals
engaged in an undesirable behavior, which is deemed uncontrollable. According to this perspective, people judge culprits who behave out of line with acceptable social, moral, or even legal norms as less blameworthy if the behavior is viewed as uncontrollable. More specifically, Weiner, Perry, and Magnusson (1988) submit that once a somatic condition, such as a genetic predisposition to addiction, is associated with a stigmatized condition, such as being an addict (or a smoker), people are more likely to evaluate the stigmatized condition as uncontrollable. Weiner et al. also argue that once people assess reduced volition for the specific condition, they become more sympathetic toward people manifesting this condition; enhanced sympathy diminishes reprimand and condemnation. These assertions have been supported empirically in studies which demonstrated increased sympathy toward protagonists who engage in undesirable behaviors when a genetic explanation rather than psychosocial explanations were highlighted (Monterosso, Royzman, and Schwartz 2005). The present study, however, takes this research one step beyond the evaluations of individual protagonists, by explicitly testing (and finding) the effects of such causal attributions on evaluations of public policies.

Our conclusions about smoking are consistent with other findings on the consequences of providing a genetic etiology for behavior as well. In two previous studies, men evaluated sex crimes more negatively and were more punitive toward the perpetrators after learning of socio-environmental contributions to promiscuousness or rape (Dar-Nimrod et al. 2011). The unprompted responses of men in the control condition were comparable to responses following the genetic account rather than the environmental account. Considering these findings and the present results together, the implication might be that behaviors which are perceived as deeply rooted are judged as less controllable and stimulate less negative evaluations (Weiner, Perry, and Magnusson 1988). Conversely, an increased focus on environmental etiologies, which are viewed less deterministically (Dar-Nimrod and Heine 2011a, Dar-Nimrod and Heine 2011b), may increase perceptions of control and stimulate more support for punitive approaches and less tolerance of the behaviors in question.

This study adds to the growing interest in people’s attitudes toward smoking habits and policies in general and to the role of the flourishing scientific literature on its genetic correlates in specific. One notable limitation of the present study is the use of a hypothetical scenario to evaluate policy endorsement and attitudes. Future research is needed in real workplace contexts. Additionally, attitudes toward smoking policies may also be of empirical interest as a function of perceived etiological accounts. As such, the present findings may inform public policy-makers about the role of causal attributions in policy evaluations. That is, prior to the introduction of restrictive smoking policy, research-based experiential attributions may be emphasized among the relevant shareholders for which the policy would be relevant.
Acknowledgments

This work was partially supported by the National Institute of Mental Health (grant number T32MH18911) and the Australian Research Council (DP140104527). The contents of this manuscript are solely the responsibility of the authors and do not necessarily represent the official views of the National Institutes of Health.

The authors report no competing interests.

References


Manfredi, S., et al. 2007. “GSTM1, GSTT1 and CYP1A1 Detoxification Gene Polymorphisms and Susceptibility to Smoking-Related Coronary Artery Disease: A Case-Only Study.”


