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# Concerns about unintended negative consequences of informing the public about multifactorial risks may be premature for young adult smokers

**Erika A. Waters**,
Department of Surgery (Division of Public Health Sciences), Washington University School of Medicine, Saint Louis, MO

**Caroline Kincaid**,
Department of Surgery (Division of Public Health Sciences), Washington University School of Medicine, Saint Louis, MO

**Annette R. Kaufman**,
Division of Cancer Control and Population Sciences, National Cancer Institute, Rockville, MD

**Michelle L. Stock**,
Department of Psychology, George Washington University, Washington, DC

**Laurel M. Peterson**,
Department of Psychiatry, University of Pittsburgh, Pittsburgh PA

**Nicole L. Muscanell**, and
Department of Psychology, The University of Alabama, Tuscaloosa, AL

**Rosanna E. Guadagno**
Department of Psychology, The University of Alabama, Tuscaloosa, AL

## Abstract

**Background—**Many health risks are associated with both genetic and behavioral factors. Concerns have been raised that learning about such *multifactorial* risks might have detrimental effects on health-related beliefs, cognitions and affect. However, experimental evidence is sparse.

**Objective—**To explore the effects of reading an online news article about the discovery of a genetic basis for nicotine addiction.

**Methods—**Smokers (N=333) were recruited from the psychology subject pools of two major universities. Participants were randomly assigned to read one of three news articles: one describing a genetic basis for nicotine addiction and lung cancer obtained from a national news source, one altered to indicate no genetic basis for nicotine addiction and lung cancer, or one unrelated attention control. Participants then completed an online questionnaire, which assessed smoking-related cognitions and affect, and beliefs about nicotine addiction, quitting smoking, and whether the harms of tobacco use are delayed.

Corresponding Author: Erika A. Waters, Department of Surgery (Division of Public Health Sciences), 660 S. Euclid, Campus Box 8100, St. Louis, MO 63110; Phone: 314-747-5705; Fax: 314-454-7941; waterse@wudosis.wustl.edu.

**Results—**There was no statistically significant influence of experimental condition on smoking-related cognitions/affect (ps>.05, $\eta^2$<.002), beliefs about addiction and quitting (Wilks' $\lambda$=0.98, $p$=.66, $\eta^2$=.01), or delayed harm (ps>.05, $\eta^2$<.002).

**Conclusion—**Reading an online news article about the presence or absence of a genetic basis for nicotine addiction was not found to change smoking-related cognitions/affect or beliefs among young adult smokers. Concerns about negative effects of multifactorial risk information on health beliefs may be premature. Nevertheless, to effectively translate basic genomics research into effective public health practice, further research should investigate these issues in different populations, via different communication modalities, and for different health outcomes.

### Keywords

tobacco use; smoking; multifactorial; genetic; gene-by-environment; behavior; attitudes

## Background

In the last two decades, society has witnessed an explosion of genetics research. What began as specific tests that identified a small number of relatively rare, high-risk genes, such as those related to cystic fibrosis and breast cancer (Lerman, 1995; Livingstone, Axton, Mennie, Gilfillan, & Brock, 1993), has expanded greatly. Recent research has identified genetic variations that operate in concert with other genes, environmental factors, and behaviors to affect mental and physical health, personality, and behavior (Khoury, Gwinn, Bowen, & Dotson, 2012; Miller & O'Callaghan, 2013). Such *multifactorial* genetic risks occur more often in the population than single-gene or other high-penetrance allelic variations, but the risk increase (or reduction) that they confer is relatively small, and the clinical validity and utility of finding these low penetrance variations is limited at this time (Khoury, 2010; Khoury et al., 2009). Nevertheless, experts in public health genomics are hopeful that research that identifies multifactorial risks can be used to improve public health by encouraging health behavior change (Collins, Green, Guttmacher, & Guyer, 2003; McBride et al., 2010).

Although most research related to providing genetic information to the public focuses on providing an individual with his or her personal genetic test results (e.g., Sanderson et al., 2009), for reasons described below, this paper will focus on a lesser-studied but likely more common experience: learning about the discovery of a multifactorial genetic risk via an article published in the news media.

### The Mass Media as a Primary Source of Information

Although personalized tests for multifactorial genetic risks are available, their use is not widespread. In addition to concerns about the validity and utility of testing, the considerable barriers that many people experience in accessing basic healthcare also act—and may continue to act—as barriers to genetic testing (Institute of Medicine, 2009). Even the least expensive direct-to-consumer tests, which examine approximately one million allelic variations across the genome (23andMe, 2007), may be out of reach for individuals with limited incomes. Consequently, the mass media (e.g., newspaper, television, Internet) currently holds the greatest role in disseminating genomics research findings to the public.

The mass media is already a primary source of information about cancer, health, and genetics for the public (Bates, 2005; Caulfield & Condit, 2012; Rutten, Moser, Beckjord, Hesse, & Croyle, 2007; Viswanath, 2005). In addition to increasing awareness about an issue, the media can shape the public's perceptions of risk (Brennan, Durkin, Cotter, Harper, & Wakefield, 2011; Morton & Duck, 2001) and influence engagement in health behaviors (Valente & Saba, 1998; Wakefield et al., 2012). However, the media have been criticized for failing to convey scientific results accurately (Brechman, Lee, & Cappella, 2011; Condit, 2007), for overemphasizing the genetic aspect of a disease that has both genetic and behavioral risk factors (Cheng, Condit, & Flannery, 2008), and for overstating the importance of genomics research in general (Caulfield & Condit, 2012). Although research suggests that news headlines such as “Can't quit smoking? Blame your genes”(Associated Press, 2008) and “Are your genes making you fat?” (Jones, 2013) may not increase endorsement of genetic determinism (Condit et al., 2001), it is important to understand how exposure to such information affects other health cognitions, beliefs, and behavior. As coverage of scientific news expands to new media (e.g., news aggregation websites, blogs), and information on these websites is shared with friends, family, and acquaintances via social networking websites (Enda & Mitchell, 2013), any consequences—positive or negative—of learning about the discovery of a multifactorial genetic risk may affect ever-larger numbers of people (Lea, Kaphingst, Bowen, Lipkus, & Hadley, 2011; Viswanath et al., 2012).

## Preliminary Evidence of Potential Unintended Consequences

Many laypeople recognize that the risk of developing a disease (e.g., cancer) or of having a phenotype that could lead to disease (e.g., obesity) is dependent upon both genetic and environmental or behavioral causes (Condit & Shen, 2011; Sanderson, Waller, Humphries, & Wardle, 2011). However, it is difficult for laypeople to articulate the idea of a multifactorial risk (Condit et al., 2009), and beliefs that genes determine outcomes (Bates, Templeton, Achter, Harris, & Condit, 2003) and that genes represent the essential nature of individuals (Keller, 2005; Parrott, Kahl, Ndiaye, & Traeder, 2012) are prevalent. This suggests that learning about a multifactorial genetic risk might prompt counter-adaptive changes in health behavior constructs (e.g., lower perceived behavioral control for changing health behavior). There is preliminary evidence from three related yet distinct categories of research supporting this assertion. Each category focuses on an abstract representation of genetic causation, which is the more likely experience for the majority of the public for the next several years. Responses to receiving real genetic test results, which provide more concrete information, will be reviewed in the discussion section.

Several studies asked participants to imagine receiving genetic test results. In one study, smokers indicated that testing positive (*versus* negative) for a genetic variation associated with nicotine dependence would encourage them to choose medication to help them quit, but they would also be less likely to use their own abilities (Wright, Weinman, & Marteau, 2003). This is concerning because, although medication can improve the odds of making a *successful* quit attempt, being willing to expend personal effort is also important (Fiore, Jaén, Baker, & al., 2008). Asking people to imagine that they had a genetic variation associated with an increased risk of obesity has had mixed effects: lower perceived

behavioral control in one study (Frosch, Mello, & Lerman, 2005), but no change in the similar construct of self-efficacy in a different study (Sanderson, Persky, & Michie, 2010). There were also mixed effects on intention to eat a healthy diet (Frosch et al., 2005; Sanderson et al., 2010). Perceptions of risk of becoming obese were also higher among individuals who were asked to imagine they had a genetic variation associated with obesity (Sanderson et al., 2010).

One study reported that smokers who spontaneously attributed their smoking to genetic causes reported lower self-efficacy of abstaining from smoking (Wright et al., 2007). Although genetic attributions were not associated with actual quit success, participants in this study were enrolled in a smoking cessation trial that included evidence-based social support and nicotine replacement therapy. Thus, any association might have been overwhelmed by the other components of the cessation intervention. Support for this idea resides in a population-based survey, which reported that ever smokers who endorsed (*versus* did not endorse) a genetic attribution for lung cancer were more likely to be current smokers (Kaphingst, Lachance, & Condit, 2009). Unfortunately, these cross-sectional and observational studies do not allow for drawing conclusions about the directionality of the effect or to infer causation.

To date, few studies have manipulated causal attributions by asking people to read a passage describing the potential role of genetics in behavior-dependent outcomes. One, which asked participants to read a news article describing either a genetic or behavioral basis for obesity, found that genetic attributions reduced perceptions of controllability, but increased willingness to help an obese person (Jeong, 2007). Similarly, news articles asserting a genetic (*versus* nongenetic) basis for salt-sensitive hypertension elicited lower perceptions of susceptibility and lower intentions to restrict salt intake among individuals who had never heard of the association previously (Smerecnik, Mesters, de Vries, & de Vries, 2009). Similar findings were reported for individuals who read information about hypercholesterolemia.

Overall, these three lines of research suggest that attributing a health condition to genetics, even in part, might increase endorsement of potentially detrimental health beliefs. However, a dearth of experimental research and limitations in the number of health cognitions evaluated limits our understanding of the phenomenon. Furthermore, none of the studies conveyed information about a multifactorial risk using an article that was actually published by a major media organization. If such detrimental responses to multifactorial genetic risk information were to occur systematically for certain population subgroups, it will be necessary to develop communication strategies that prevent or ameliorate such effects.

It is important to note that spontaneously attributing one's own behavior or disease to genetic factors (e.g., by observing one's family history) may be different from learning about the discovery of a genetic variation that is associated with the behavior or disease in the population. In particular, attributions that individuals make for themselves are already components of their causal models of disease and therefore are more likely to influence related beliefs and behaviors (Cameron, Marteau, Brown, Klein, & Sherman, 2011). In contrast, the extent to which being informed about a multifactorial genetic risk prompts

people to incorporate genetics into their causal models is unclear. Regardless of the exact process, the wide reach of the mass media highlights the importance of understanding how health-related cognitions, affect, beliefs, and behavior are affected by multifactorial genetic risk information.

## Objective, Rationale, and Hypotheses

Our objective was to examine how reading a news article about the genetic basis for nicotine addiction and lung cancer affected smoking-related beliefs, cognitions, and affect among young adult smokers. We had a particular interest in beliefs about nicotine addiction and the ease of quitting for reasons described below.

We focused on tobacco use because it accounts for immense suffering and death worldwide (US Department of Health and Human Services, 2004). Our choice of study population was based on the U.S. Surgeon General's assertion that young adults are at high risk of escalating their current smoking behavior, which increases their vulnerability to experiencing tobacco-related morbility and mortality (US Department of Health and Human Services, 2012). This increased risk of excalation is due, in part, to adolescents' underestimation of the addictiveness of nicotine (Slovic, 2001; US Department of Health and Human Services, 2012; Weinstein, Slovic, & Gibson, 2004). They also display unrealistic optimism regarding their personal risk of addiction and their personal ability to quit. For example, in one study 71% of adolescent smokers agreed that “most people who smoke for a few years become addicted and cannot stop”(Arnett, 2000, p. 628). However, 60% of those same smokers indicated that they personally *would* be able to smoke for a few years and then stop. This is notable because, among high school seniors who smoked *at all* in the previous 30 days, over one quarter reported having at least one unsuccessful quit attempt. Furthermore, the percentage of those interested in quitting “now” fell from 42.7% in 1995 to 34.4% in 2009 (US Department of Health and Human Services, 2012). This latter example is not a manifestation of unrealistic optimism, but the combination of diverse types of counter-adaptive risk beliefs suggests that many young adults do not realize their addiction or make plans to quit smoking until it is too late to quit easily.

Another reason young adults are at risk of escalating their smoking behavior is that they may believe that the harms of cigarette smoking are delayed (Weinstein, Marcus, & Moser, 2005). Although the carcinogens in cigarettes do have a cumulative effect over time, no amount of smoking is safe. Short-term and light smoking increase the risk of a host of short-term and immediate health problems including impaired healing and lower respiratory tract infections (Schane, Ling, & Glantz, 2010). Nevertheless, one study reported that 32% of adolescent smokers believed that there were *no* risks associated with smoking for only a few years (Slovic, 2000). Another study reported that smokers who believed that they had smoked for a shorter time period than the typical smoker believed that they were at low personal risk of developing lung cancer (Hahn & Renner, 1998). Emphasizing the addictiveness of nicotine and the immediacy of health risks might be a useful intervention message; one study reported that adolescents who recognized that quitting smoking was exceptionally difficult *and* believed that smoking had immediate harms were less likely to smoke (Gerking & Khaddaria, 2012).

Based on the literatures describing beliefs about multifactorial risks, the associations between genetic attributions and counter-adaptive health cognitions, and misperceptions of addiction and health risks among young adults, we hypothesized that, among young adult smokers, genetic vs. non-genetic attributions for nicotine addiction would:

1. Increase acknowledgement that addiction to nicotine occurs quickly and is difficult to avoid, and that quitting smoking is exceptionally difficult (Bates et al., 2003; Parrott et al., 2012).
2. Decrease self-efficacy of quitting, perceptions of risk, and intentions to quit (Jeong, 2007; Smerecnik et al., 2009).

We also formulated an exploratory research question based on more tentative data from the smoking perceptions literature: Do news articles with genetic attributions increase smokers' beliefs about the immediacy of the harms of smoking (Gerking & Khaddaria, 2012)?

## Methods

### Participants

Participants (N=746) were recruited from undergraduate psychology classes in exchange for course credit at the University of Alabama in Tuscaloosa (UA) and The George Washington University (GWU) during the Fall 2010 and Spring 2011 semesters, including 278 students who reported having not smoked even a puff of a cigarette in the last 30 days (i.e., never or former smokers). Because this study focuses only on smokers, never and former smokers were excluded from the analyses. Of the 392 smokers who began the survey, 333 (85.0%) were eligible for analysis. Participants were excluded because they did not check the box indicating they consented to participate (n=17), had participated in the study previously (n=6), and/or answered the comprehension verification item inaccurately (n=43).

### Procedure

Participants completed the study online. After providing consent, participants responded to questionnaire items assessing demographics and past smoking behavior. Participants were randomly assigned to read one of three news articles (described in Experimental Conditions section). Randomization was carried out by computer code that activated when participants clicked on the link to the study; study personnel were blinded to experimental condition. After reading the article, participants completed survey items assessing the dependent variables (see Table 1). The debriefing included informing participants that a genetic basis for nicotine addiction did indeed exist, but a genetic link did not mean that it would be impossible to quit. We also provided participants with smoking cessation resources. Participation took approximately 40 minutes. The complete survey can be obtained from the corresponding author.

### Experimental Conditions

The *genetic basis* condition was an abbreviated version of an actual article that was posted on the website of a large news organization (Associated Press, 2008; Jones, 2013). The article described the discovery of a genetic variation associated with increased risk of both

severe nicotine dependence and lung cancer. We elected to keep the description of both risks in the article to increase the ecological validity of the present research in comparison with other similar studies (i.e., individuals who read the article during the course of their everyday lives, rather than an experimental setting, read about both risks). The *no genetic basis* condition was created by altering the *genetic basis* condition to assert there was no scientific evidence of a link between a particular genetic variant and nicotine addiction or lung cancer. The *attention control* condition described a study about the tendency of dogs to imitate human behavior. It was chosen because, like the study reporting the genetic basis for nicotine addiction, it was found in the science section of the news website. Including an article unrelated to health also provided a baseline for comparison regarding smokers' cognitions, beliefs, and affect in the absence of any multifactorial risk information.

## Measures

Before reading the article, participants answered items assessing standard *demographic characteristics* for studies conducted in the U.S. (i.e., age, race, gender), school attended, and *smoking behavior* (i.e., "Within the last 30 days did you smoke…? [Not at all, not even a puff/Some days/Every day]"; "Have you smoked at least 100 cigarettes in your entire life? [Yes/No]"; "On average, how many cigarettes do you now smoke per day?"). After reading the article, participants answered questions to verify their comprehension of the information, to assess whether they did or did not believe that a genetic basis for nicotine addiction exists, and agreement or disagreement with items assessing genetic determinism in relation to nicotine addiction (e.g., "If a person smokes cigarettes and has a gene for addiction to nicotine, he or she will definitely get addicted to nicotine [5-point Likert-type scale, Strongly disagree—Strongly agree]"). They also completed the items described in Table 2.

## Analysis Plan

**Preliminary Analyses—**Individuals were considered smokers if they reported smoking even a puff on some or every day of the 30 days prior to the experiment. Smokers were further categorized according to whether they had smoked fewer than 100 cigarettes in their lifetimes ("experimenters") or least 100 cigarettes in their lifetimes ("establishedsmokers") (Bondy, Victor, & Diemert, 2009).

Exploratory factor analysis (EFA) with varimax rotation was conducted for the 19 variables related to beliefs about nicotine addiction and quitting. The EFA was restricted to five factors due to the small percentage of variance explained by each of the eight factors that an EFA yielded based on setting the Eigenvalue criterion to >1. The results of the EFA were used to create subscales of addiction and quitting beliefs. Separate EFAs were also conducted for self-efficacy, worry, feelings of risk, and delayed harm items to ensure that they represented unified constructs. Scales for each construct were then compiled from multiple items. Cronbach α coefficients were calculated for each scale and subscale.

Chi-square tests and Pearson correlation coefficients were examined to identify potential confounding variables (data not shown but are available from the first author). When demographic variables were correlated with outcome variables, they were included in the

analyses as covariates. Dependent variables that did not meet the normality assumption were transformed (see Table 2 note).

**Main Analyses—**Multivariate analysis of covariance (MANCOVA) was used to examine whether the type of news article affected beliefs about nicotine addiction and quitting. Experimental condition was the independent variable, the EFA-produced subscales (see "Factors" below) were the predictors, and variables that were related to the outcome of interest and might have therefore biased results (e.g., school attended, gender, race/ethnicity, smoking *versus* not smoking at least one cigarette per day) were the covariates. The remaining outcome variables of interest, for which MANCOVA was not appropriate, were analyzed using analyses of covariance (ANCOVA).

## Results

### Sample Characteristics

Of the 333 individuals who completed the study, most (97%; n=322) were aged 18-22 (M=19.3, SD=2.0), identified as having a White racial background (85%; n=283), and were female (66.4%; n=221). The sample was comprised primarily of people who smoked relatively few cigarettes per day (M=2.7, SD=4.3). Less than half of individuals reported smoking at least one cigarette per day (41.4%; n=138). The majority of participants were classified as "established" smokers (53%; n=177). Approximately one-third of participants (34.5%, n=115) had made at least one prior quit attempt. Smoker status (established *versus* experimental) and quit attempts (at least one attempt *versus* never attempted) were explored as potential moderators of the relationship between experimental condition and the outcomes of interest. We planned to examine nicotine dependence and craving as additional potential moderators, but floor effects yielded too little variance for analysis.

A greater proportion of participants from UA than GWU reported being White (92.9% vs. 80.7%, respectively, $\chi^2(1)=10.5$, $p=.001$), smoking at least one cigarette per day (52.3% vs. 21.8%, $\chi^2(1)=29.3$, $p<.0001$), being established smokers (63.6% vs. 34.5%, $\chi^2(1)=26.0$, $p<.0001$), and having made at least one quit attempt, (39.7% vs. 25.2%, $\chi^2(1)=7.1$, $p=.008$). Although school attended, gender, experimenter/established smoker status, and average number of cigarettes smoked per day were not associated with experimental condition, slightly more women were randomized to the *no genetic basis* condition than the other two experimental conditions, $p=.045$. Consequently, gender and recruitment location were controlled in all analyses. The study was powered to detect a small main effect of experimental condition (Cohen's d=0.3, equivalent to $\eta^2=.022$) (Cohen, 1988).

### Factors

The EFA yielded five factors: *Amount to Addiction*; *Can Avoid Addiction*; *Judgment*; *Deterministic Addiction*; and *Agentic Quitting* (see Table 1). Factor loadings and variance accounted can be obtained from the corresponding author.

### Main Analysis

The MANCOVA showed no significant relationship between experimental condition and the five nicotine addiction and quitting belief subscales (Wilks' $\lambda$=0.98, F(10, 620)=0.7, *p*=.66, $\eta^2$=.01). Because the overall MANCOVA was not significant, follow-up ANCOVAs on each subscale were not examined (Stevens, 2002). The ANCOVAs conducted on the remaining constructs indicated that experimental condition did not alter any of the remaining smoking-related beliefs, cognitions, or affect (see Table 2).

We conducted four sets of supplemental analyses to examine possible moderators of the influence of experimental condition on the outcome variables: 1) endorsement *versus* non-endorsement of the belief that a genetic basis for nicotine addiction existed, 2) slight/strong endorsement *versus* non-endorsement of genetic determinism, 3) at least one *versus* no prior quit attempt, and 4) established *versus* experimenter smoker status. None of the moderators yielded significant findings, all *p*s=.07-.98; all effect sizes $\eta^2$=.000-.017.

## Discussion

Prior research suggested that being informed of a genetic (*versus* non-genetic) risk for diseases and health conditions with multiple causal factors might be associated with counter-adaptive health cognitions (Jeong, 2007; Smerecnik et al., 2009). This study explored the extent to which such concerns should extend to young adult smokers who read about the discovery of a genetic basis for nicotine addiction and lung cancer. If counter-adaptive changes occurred, it would be necessary to develop communication strategies to counteract them. It should be noted that this study was not designed to examine actual quitting behavior, nor do we argue that a news article reporting a genetic basis for nicotine addiction will improve cessation outcomes. However, focusing on factors that influence whether people even *attempt* to quit (i.e., beliefs, cognitions, affect) is reasonable (Ajzen, 1985; Fiore, Jaén, Baker, & al., 2008; Fishbein, 2008; Rogers & Prentice-Dunn, 1997).

Mass media-based communications of multifactorial genetic risk information are important because, until much more basic genomics research provides truly reliable and valid personalized genetic and genomic tests (Collins et al., 2003; Khoury et al., 2011), the media will be the primary source of such information for most of the lay public. Because the media shapes public perceptions of genetics (Bates, 2005), it is important to understand whether individuals who encounter such information experience changes in their perceptions and attitudes that are potentially adaptive (e.g., increased acknowledgement of the addictiveness of nicotine) or counter-adaptive (e.g., reduced perceptions of risk of negative smoking-related outcomes). Any changes in attitudes could provide one additional element that might, in conjunction with other intrapersonal, social, and institutional factors, prompt changes in smoking behavior.

We found that reading a news article about a genetic basis for nicotine addiction did not influence young adult smokers' smoking-related beliefs, cognitions, or affect. Responses were not moderated by believing (or not believing) that there was a genetic basis for nicotine addiction, endorsing genetic determinism, or having ever made a prior quit attempt. Responses were also unaffected by being an established smoker *versus* experimenting with

smoking. We had the statistical power to detect a small main effect ($\eta^2$=.022). All except one of our observed effect sizes were much smaller, which raises doubt about the practical importance of such differences, even if they had been found to be statistically significant. Consequently, it seems reasonable to conclude that these results reflect an approximation of how young adult smokers might respond to reading a news article about the genetic basis for nicotine addiction and lung cancer.

These results contrast with published experimental and observational studies that examined cognitive and affective responses to learning about the existence of interactions among genes, behavior, the environment, and health (Jeong, 2007; Kaphingst et al., 2009; Smerecnik et al., 2009; Wright et al., 2007). It is possible that this study's findings differed from others because smokers are often reminded of the dangers of tobacco use and, consequently, they may have been less likely to process the message in a careful and systematic way (Slovic, 2001). Another possibility is that they responded defensively to the information and produced counter arguments (Kessels, Ruiter, & Jansma, 2010), or did not perceive the information to be personally relevant (Docherty et al., 2011). It could also be that a brief news article is not sufficient to change an individual's beliefs, which may formulate and evolve over time from a multitude of information sources. Future research should explore these issues, as well as whether continuous inundation with the same type of information (rather than a single encounter), or discussions generated via social interactions, could elicit changes (Pidgeon, Kasperson, & Slovic, 2003).

## Personal Genetic Testing

It is important to consider these results in the context of and in contrast to personal genetic testing. Genetic counselors and other clinicians and researchers have worked diligently for many years to convey personal genetic test results in a way that is meaningful and useful to patients without producing long-lasting detrimental psychological effects like depression or clinically-relevant levels of distress. BRCA1/2 testing is an excellent example of such efforts (Hilgart, Coles, & Iredale, 2012; Hilgart, Hayward, Coles, & Iredale, 2012; Howard, Balneaves, & Bottorff, 2009; Katapodi et al., 2011; Lerman, Biesecker, et al., 1997; Schwartz et al., 2012; van Oostrom et al., 2003), which emphasize the importance of educating patients about basic genetic information, working with patients who may have limited numeracy and health literacy, and facilitating informed and clinically-appropriate decision making (Portnoy, Roter, & Erby, 2010).

Whereas a BRCA and other similar high penetrance genetic variations confer an extremely high level of risk, genetic variations that interact with behavior and other environmental factors tend to increase risk to a lesser extent that may or may not be clinically relevant (McBride et al., 2008). These multifactorial risks may have a more limited influence on medical decision making and motivation to change behavior. For example, providing personalized genetic risk results to smokers may increase perceptions of risk of developing lung cancer (Lerman, Gold, et al., 1997), but may not motivate smoking cessation (Audrain et al., 1997; Lipkus, McBride, Pollak, Lyna, & Bepler, 2004; Sanderson et al., 2009). The reasons for this should be explored further.

Viewed in this context, it is perhaps unsurprising that a single exposure to a news media article was insufficient to change smoking-related beliefs, cognitions, and affect. Nevertheless, the contrast between the findings of this study and the findings of work conducted by others who also examined the influence of non-personalized genetic risk information (i.e., informing participants about the discovery of a multifactorial risk (e.g., Smerecnik et al., 2009)) highlights the need for further research to determine the conditions under which genomic information—either provided on an individual or a mass media level—does and does not motivate attitude and behavior change (Collins et al., 2003; McBride et al., 2008). For example, do responses vary according to whether a health hazard is perceived to be under more (*versus* less) personal control (e.g., tobacco use *versus* breast cancer)? If so, communications could be designed to target such beliefs.

## Ethical Considerations

There may be ethical concerns surrounding research that informs the public about a multifactorial genetic risk, whether such information is provided via the mass media in a nonspecific manner or it is provided as a personal genetic test result. The extent of the concern is likely proportional to the degree of genetic determinism endorsed by the target population. Genetic determinism is by no means universal (Bates et al., 2003; Condit et al., 2009), but studies that examine this issue should develop a debriefing process that alleviates any potential misunderstandings. This is especially important for studies that examine outcomes that have multiple individual and social causes, in addition to genetics (e.g., obesity, tobacco use).

## Limitations and Future Research

It should be noted that the Cronbach alpha levels for the deterministic addiction and agentic beliefs subscales were modest. This might have obscured effects that may have been present in a more cohesive scale. However, sensitivity analyses found that using individual items, rather than the subscale scores, did not alter the overall results; the overall MANCOVA results for the effect of experimental condition on addiction and quitting beliefs remained statistically nonsignificant.

Another factor that might limit our findings is that nicotine dependence, as measured by time to first cigarette, was insufficiently variable to include in the models. However, our sensitivity analyses examining differences in responding according to smoking status (established *versus* experimenter) and past quit attempt alleviate these concerns somewhat. Future work would benefit by recruiting participants with a broader range of nicotine dependence, including those who may be interested in joining a smoking cessation program.

This study recruited college students as participants, who were younger and more educated than smokers in the general population. Nevertheless, their vulnerability to becoming long-term smokers, relatively lower levels of nicotine addiction, potentially long lifespan, erroneous beliefs about the addictiveness of nicotine, and sometimes unrealistically optimistic beliefs about quitting make young adults an ideal population to target with tobacco control efforts (US Department of Health and Human Services, 2012). Research should determine the extent to which the results of this study extends to young adults outside

the university system, members of racial and ethnic minority groups, older and long-term smokers, and those with low health literacy. The information processing and persuasion literatures suggest that people may also respond differently based on the method of exposure (e.g., television, newspaper, internet) and based on the source of information (e.g., government agency, pharmaceutical company, physician) (e.g., Chen & Chaiken, 1999).

### Implications

It could also be that providing laypeople with generic genetic information, such as that presented in this study, is not an effective strategy for changing attitudes or motivating behavior change. Yet, at the present time, this research domain contains too many questions that need to be addressed before stating with certainty that this is the case. Therefore, identifying whether counter-adaptive changes in health beliefs and cognitions occur in different populations and via different sources of multifactorial genetic risk information is necessary to facilitate translation and dissemination of genomic risk information to the public in a way that is both useful and meaningful to laypeople.

## References


23andMe. 23andMe. 2007. Retrieved May 3 2013, from www.23andme.com

Ajzen, I. From intentions to action: A theory of planned behavior. In: Kuhl, J.; Beckman, J., editors. Action Control: From Cognitions to Behaviors. New York: Springer; 1985. p. 11-39.

Arnett JJ. Optimistic bias in adolescent and adult smokers and nonsmokers. Addict Behav. 2000; 25(4):625–632. [PubMed: 10972456]

Associated Press. Can't quit smoking? Blame your genes. 2008. http://www.nbcnews.com/id/23919596/ns/health-addictions/t/cant-quit-smoking-blame-your-genes/

Audrain J, Boyd NR, Roth J, Main D, Caporaso NF, Lerman C. Genetic susceptibility testing in smoking-cessation treatment: one-year outcomes of a randomized trial. Addict Behav. 1997; 22(6): 741–751. [PubMed: 9426791]

Bates, Benjamin R. Public culture and public understanding of genetics: A focus group study. Public Understanding of Science. 2005; 14:47–65. [PubMed: 15822248]

Bates, Benjamin R.; Templeton, Alan; Achter, Paul J.; Harris, Tina M.; Condit, Celeste M. What does “A gene for heart disease” mean? A focus group study of public understandings of genetic risk factors. American Journal of Medical Genetics. 2003; 119A:156–161. [PubMed: 12749055]

Bondy SJ, Victor JC, Diemert LM. Origin and use of the 100 cigarette criterion in tobacco surveys. Tob Control. 2009; 18(4):317–323.10.1136/tc.2008.027276 [PubMed: 19491091]

Brechman JM, Lee CJ, Cappella JN. Distorting genetic research about cancer: From bench science to press release to published news. Journal of Communication. 2011; 61(3):496–513. [PubMed: 25580022]

Brennan E, Durkin SJ, Cotter T, Harper T, Wakefield MA. Mass media campaigns designed to support new pictorial health warnings on cigarette packets: evidence of a complementary relationship. Tob Control. 2011; 20(6):412–418.10.1136/tc.2010.039321 [PubMed: 21474501]

Cameron, Linda D.; Marteau, TM.; Brown, PM.; Klein, WM.; Sherman, KA. Communication strategies for enhancing understanding of the behavioral implications of genetic and biomarker tests for disease risk: The role of coherence. J Behav Med. 201110.1007/s10865-011-9361-5

Caulfield T, Condit Celeste M. Science and the sources of hype. Public Health Genomics. 2012; 15(3-4):209–217.10.1159/000336533 [PubMed: 22488464]

Chen, Serena; Chaiken, Shelly. The heuristic-systematic model in its broader context. In: Chaiken, S.; Trope, Y., editors. Dual-Process Theories in Social Psychology. New York, NY: The Guilford Press; 1999. p. 73-96.

Cheng Y, Condit Celeste M, Flannery D. Depiction of gene-environment relationships in online medical recommendations. Genet Med. 2008; 10(6):450–456. doi:10.1097GIM.0b013e31817701a8. [PubMed: 18496221]

Cohen, Jacob. Statistical power analysis for the behavioral sciences. 2nd. Hillsdale, NJ: Erlbaum; 1988.

Collins FS, Green ED, Guttmacher AE, Guyer MS. A vision for the future of genomics research. Nature. 2003; 422(6934):835–847. [PubMed: 12695777]

Condit, Celeste M. How geneticists can help reporters to get their story right. Nature Reviews Genetics. 2007; 8(10):815–820.

Condit, Celeste M.; Ferfuson, A.; Kassel, R.; Thadhani, C.; Gooding, C.; Parrott, R. An exploratory study of the impact of news headlines on genetic determinism. Science Communication. 2001; 22:379–395.

Condit, Celeste M.; Gronnvoll, Marita; Landau, Jamie; Shen, Lijiang; Wright, Lanelle; Harris, Tina M. Believing in both genetic determinism and behavioral action: a materialist framework and implications. Public Understanding of Science. 2009; 18(6):730–746.

Condit, Celeste M.; Shen, L. Public understanding of risks from gene-environment interaction in common diseases: implications for public communications. Public Health Genomics. 2011; 14(2): 115–124.10.1159/000314915 [PubMed: 20714109]

Docherty SL, McBride CM, Sanderson SC, O'Neill SC, Shepperd JA, Lipkus IM. Young smokers' views of genetic susceptibility testing for lung cancer risk: minding unintended consequences. J Community Genet. 2011; 2(3):165–172.10.1007/s12687-011-0053-1 [PubMed: 21860660]

Enda, Jodi; Mitchell, Amy. Friends and Family – Important Drivers of News. 2013. http://stateofthemedia.org/2013/special-reports-landing-page/friends-and-family-important-drivers-of-news/

Fiore, MC.; Jaén, CR.; Baker, TB. Treating Tobacco Use and Dependence: 2008 Update Clinical Practice Guideline. Rockville, MD: United States Department of Health and Human Services, Public Health Service; 2008.

Fishbein, Martin. A reasoned action approach to health promotion. Medical Decision Making. 2008; 28:834–844. [PubMed: 19015289]

Gerking S, Khaddaria R. Perceptions of health risk and smoking decisions of young people. Health Econ. 2012; 21(7):865–877.10.1002/hec.1760 [PubMed: 21796724]

Hahn A, Renner B. Perception of health risks: How smoker status affects defensive optimism. Anxiety, Stress, and Coping. 1998; 11(2):93–112.

Hilgart JS, Coles B, Iredale R. Cancer genetic risk assessment for individuals at risk of familial breast cancer. Cochrane Database Syst Rev. 2012; 2:CD003721.10.1002/14651858.CD003721.pub3 [PubMed: 22336791]

Hilgart JS, Hayward JA, Coles B, Iredale R. Telegenetics: a systematic review of telemedicine in genetics services. Genet Med. 2012; 14(9):765–776.10.1038/gim.2012.40

Howard AF, Balneaves LG, Bottorff JL. Women's decision making about risk-reducing strategies in the context of hereditary breast and ovarian cancer: a systematic review. J Genet Couns. 2009; 18(6):578–597.10.1007/s10897-009-9245-9 [PubMed: 19802692]

Institute of Medicine. America's Uninsured Crisis: Consequences for Health and Health Care. Washington, DC: National Academies Press; 2009.

Jeong, Se-Hoon. Effects of news about genetics and obesity on controllability attribution and helping behavior. Health Communication. 2007; 22(3):221–228. [PubMed: 17967144]

Jones, Caroline. Are your genes making you fat?. 2013. http://www.dailyrecord.co.uk/news/health/your-genes-making-you-fat-1852525

Kaphingst, Kimberly A.; Lachance, CR.; Condit, Celeste M. Beliefs about heritability of cancer and health information seeking and preventive behaviors. Journal of Cancer Education. 2009; 24(4): 351–356. [PubMed: 19838898]

Katapodi MC, Northouse L, Pierce P, Milliron KJ, Liu G, Merajver SD. Differences between women who pursued genetic testing for hereditary breast and ovarian cancer and their at-risk relatives who did not. Oncol Nurs Forum. 2011; 38(5):572–581.10.1188/11.ONF.572-581 [PubMed: 21875844]

Keller, Jonannes. In genes we trust: The biological component of psychological essentialism and its relationship to mechanisms of motivated social cognition. Journal of Personality and Social Psychology. 2005; 88(4):686–702. [PubMed: 15796668]

Kessels LT, Ruiter RA, Jansma BM. Increased attention but more efficient disengagement: neuroscientific evidence for defensive processing of threatening health information. Health Psychol. 2010; 29(4):346–354.10.1037/a0019372 [PubMed: 20658820]

Khoury, Muin J. Dealing with the evidence dilemma in genomics and personalized medicine. Clin Pharmacol Ther. 2010; 87(6):635–638.10.1038/clpt.2010.4 [PubMed: 20485318]

Khoury, Muin J.; Clauser, SB.; Freedman, AN.; Gillanders, EM.; Glasgow, RE.; Klein, WM.; Schully, SD. Population sciences, translational research, and the opportunities and challenges for genomics to reduce the burden of cancer in the 21st century. Cancer Epidemiol Biomarkers Prev. 2011; 20(10):2105–2114.10.1158/1055-9965.EPI-11-0481 [PubMed: 21795499]

Khoury, Muin J.; Gwinn, M.; Bowen, MS.; Dotson, WD. Beyond base pairs to bedside: a population perspective on how genomics can improve health. Am J Public Health. 2012; 102(1):34–37.10.2105/AJPH.2011.300299 [PubMed: 22095352]

Khoury, Muin J.; McBride, Colleen; Schully, Sheri D.; Ioannidis, John PA.; Feero, W Gregory; Janssens, A Cecile JW., et al. The scientific foundation for personal genomics: Recommendations from a National Institutes of Health--Centers for Disease Control and Prevention multidisciplinary workshop. Genetics in Medicine. 2009; 11(8):559–567. [PubMed: 19617843]

Lea DH, Kaphingst KA, Bowen D, Lipkus I, Hadley DW. Communicating genetic and genomic information: health literacy and numeracy considerations. Public Health Genomics. 2011; 14(4-5):279–289.10.1159/000294191 [PubMed: 20407217]

Lerman, Caryn. Effects of individualized breast cancer risk counseling: A randomized trial. Journal of the National Cancer Institute. 1995; 87:286–292. [PubMed: 7707420]

Lerman, Caryn; Biesecker, B.; Benkendorf, JL.; Kerner, J.; Gomez-Caminero, A.; Hughes, C.; Reed, M. Controlled trial of pretest education approaches to enhance informed decision-making for BRCA1 gene testing. Journal of the National Cancer Institute. 1997; 89(2):148–157. [PubMed: 8998184]

Lerman, Caryn; Gold, K.; Audrain, J.; Lin, TH.; Boyd, NR.; Orleans, CT.; et al. Caporaso, N. Incorporating biomarkers of exposure and genetic susceptibility into smoking cessation treatment: effects on smoking-related cognitions, emotions, and behavior change. Health Psychol. 1997; 16(1):87–99. [PubMed: 9028818]

Lipkus, Isaac M.; McBride, Colleen M.; Pollak, Kathryn I.; Lyna, Pauline; Bepler, Gerold. Interpretation of genetic risk feedback among African American smokers with low socioeconomic status. Health Psychology. 2004; 23(2):178–188. [PubMed: 15008663]

Livingstone J, Axton RA, Mennie M, Gilfillan A, Brock DJ. A preliminary trial of couple screening for cystic fibrosis: designing an appropriate information leaflet. Clin Genet. 1993; 43(2):57–62. [PubMed: 8448901]

McBride CM, Bowen D, Brody LC, Condit CM, Croyle RT, Gwinn M, et al. Valente TW. Future health applications of genomics: priorities for communication, behavioral, and social sciences research. Am J Prev Med. 2010; 38(5):556–565.10.1016/j.amepre.2010.01.027 [PubMed: 20409503]

McBride CM, Hensley-Alford S, Reid RJ, Larson EB, Baxevanis AD, Brody LC. Putting science over supposition in the arena of personalized genomics. Nature Genetics. 2008; 40(8):939–942. [PubMed: 18665132]

Miller DB, O'Callaghan JP. Personalized medicine in major depressive disorder -- opportunities and pitfalls. Metabolism. 2013; 621(Suppl):S34–39.10.1016/j.metabol.2012.08.021 [PubMed: 23021040]

Morton, Thomas A.; Duck, Julie M. Mass and interpersonal influences on perceptions of risk to self and others. Communication Research. 2001; 28(5):602–626.

Parrott R, Kahl ML, Ndiaye K, Traeder T. Health communication, genetic determinism, and perceived control: the roles of beliefs about susceptibility and severity versus disease essentialism. J Health Commun. 2012; 17(7):762–778.10.1080/10810730.2012.677301 [PubMed: 22574711]

Pidgeon, Nick; Kasperson, Roger E.; Slovic, Paul, editors. The social amplification of risk. Cambridge, UK: Cambridge University Press; 2003.

Portnoy DB, Roter D, Erby LH. The role of numeracy on client knowledge in BRCA genetic counseling. Patient Educ Couns. 2010; 81(1):131–136.10.1016/j.pec.2009.09.036 [PubMed: 19854023]

Rogers, RW.; Prentice-Dunn, S. Protection motivation theory. In: Gochman, DS., editor. Handbook of Health Behavior Research I: Personal and Social Determinants. New York: Plenum Press; 1997. p. 113-132.

Rutten, LF.; Moser, RP.; Beckjord, EB.; Hesse, BW.; Croyle, RT. Cancer Communication: Health Information National Trends Survey. Washington DC; 2007. NIH Pub. No. 07-6214

Sanderson SC, O'Neill SC, White DB, Bepler G, Bastian L, Lipkus IM, McBride CM. Responses to online GSTM1 genetic test results among smokers related to patients with lung cancer: a pilot study. Cancer Epidemiology, Biomarkers, and Prevention. 2009; 18(7):1953–1961.

Sanderson SC, Waller J, Humphries SE, Wardle J. Public awareness of genetic influence on chronic disease risk: are genetic and lifestyle causal beliefs compatible? Public Health Genomics. 2011; 14(4-5):290–297.10.1159/000294280 [PubMed: 20431275]

Schane RE, Ling PM, Glantz SA. Health effects of light and intermittent smoking: a review. Circulation. 2010; 121(13):1518–1522.10.1161/CIRCULATIONAHA.109.904235 [PubMed: 20368531]

Schwartz MD, Isaacs C, Graves KD, Poggi E, Peshkin BN, Gell C, et al. Perley L. Long-term outcomes of BRCA1/BRCA2 testing: risk reduction and surveillance. Cancer. 2012; 118(2):510–517.10.1002/cncr.26294 [PubMed: 21717445]

Slovic, Paul. What does it mean to know a cumulative risk? Adolescents' perceptions of short-term and long-term consequences of smoking. Journal of Behavioral Decision Making. 2000; 13:259–266.

Slovic, Paul, editor. Smoking: Risk, perception, & policy. Thousand Oaks, CA: Sage Publications, Inc; 2001.

Smerecnik, Chris MR.; Mesters, Ilse; de Vries, Nanne K.; de Vries, Hein. Alerting the general population to genetic risks: The value of health messages communicating the existence of genetic risk factors for public health promotion. Health Psychology. 2009; 28(6):734–745. [PubMed: 19916642]

Stevens, John P. Applied multivariate statistics for the social sciences. 4th. Mahwah, NJ: Lawrence Erlbaum Associates; 2002.

US Department of Health and Human Services. The health consequences of smoking: a report of the Surgeon General. Atlanta, GA: 2004. Retrieved from http://www.cdc.gov/tobacco/data_statistics/sgr/sgr_2004/index.htm

US Department of Health and Human Services. Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2012.

Valente TW, Saba WP. Mass Media and Interpersonal Influence in a Reproductive Health Communication Campaign in Bolivia. Communication Research. 1998; 25(1):96–124.

van Oostrom I, Meijers-Heijboer H, Lodder LN, Duivenvoorden HJ, van Gool AR, Seynaeve C, et al. Tibben A. Long-term psychological impact of carrying a BRCA1/2 mutation and prophylactic surgery: a 5-year follow-up study. J Clin Oncol. 2003; 21(20):3867–3874.10.1200/JCO.2003.10.100 [PubMed: 14551306]

Viswanath K. Science and society: the communications revolution and cancer control. Nat Rev Cancer. 2005; 5(10):828–835. doi:nrc1718[pii]10.1038/nrc1718. [PubMed: 16195753]

Viswanath K, Nagler RH, Bigman-Galimore CA, McCauley MP, Jung M, Ramanadhan S. The communications revolution and health inequalities in the 21st century: implications for cancer control. Cancer Epidemiol Biomarkers Prev. 2012; 21(10):1701–1708.10.1158/1055-9965.EPI-12-0852 [PubMed: 23045545]

Wakefield MA, Bowe SJ, Durkin SJ, Yong HH, Spittal MJ, Simpson JA, Borland R. Does Tobacco-Control Mass Media Campaign Exposure Prevent Relapse Among Recent Quitters? Nicotine Tob Res. 201210.1093/ntr/nts134

Weinstein, Neil D.; Marcus, SE.; Moser, RP. Smokers' unrealistic optimism about their risk. Tob Control. 2005; 14(1):55–59.10.1136/tc.2004.008375 [PubMed: 15735301]

Weinstein, Neil D.; Slovic, Paul; Gibson, Ginger. Accuracy and optimism in smokers' beliefs about quitting. Nicotine & Tobacco Research. 2004; 6(Supplement 3):S375–S380. [PubMed: 15799601]

Wright, Alison J.; Aveyard, Paul; Guo, Boliang; Murphy, Michael; Brown, Karen; Marteau, Theresa M. Is attributing smoking to genetic causes associated with a reduced probability of quit attempt success? A cohort study. Addiction. 2007; 102(10):1657–1664. [PubMed: 17854342]

Wright, Alison J.; Weinman, J.; Marteau, Theresa M. The impact of learning of a genetic predisposition to nicotine dependence: an analogue study. Tob Control. 2003; 12(2):227–230. [PubMed: 12773736]

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**Table 1**

**Survey items, organized by construct (in BOLD CAPITALS) and name of subscale**

| Scale | Question |
|---|---|
| **ADDICTION AND QUITTING**[a] | |
| Amount to addiction (α=.82) | "People can become addicted to cigarettes even if they only smoke one or two cigarettes a day." |
| | "People can become addicted to cigarettes even if they only smoke one or two cigarettes a week." |
| | "People can become addicted to cigarettes even if they only smoke occasionally with friends." |
| Can avoid addiction (α=.62) | "If I have not yet become addicted to nicotine then I never will." |
| | "If signs of nicotine addiction haven't appeared by the time someone smokes a TOTAL of 5 packs of cigarettes, they will never become addicted." |
| | "If people who smoke have good genes, they won't get addicted to nicotine." |
| | "There's not much risk In smoking during college because people have plenty of time to quit." |
| Judgment (α=.64) | "People who say they are addicted to nicotine just don't have the willpower to quit smoking." |
| | "People who are addicted to nicotine are emotionally weak." |
| | "People don't become addicted to nicotine unless they enjoy smoking." |
| | "People who are addicted to nicotine are not in control of their lives." |
| Deterministic addiction (α=.46) | "Some people will never become addicted to nicotine, no matter how much or how long they smoke." |
| | "Addiction to nicotine is an unavoidable consequence of smoking cigarettes." |
| Agentic quitting (α=.55) | "People can stop smoking if they really want to." |
| | "Willpower is the best way to quit smoking." |
| | "People who smoke have complete control over their nicotine addiction." |
| **DELAYED HARM**[a] | "There is usually no risk to this person's health at all for the first few years." |
| | "Although smoking may eventually harm this person's health, there is really no harm from smoking the very next cigarette." |
| | "There is usually no risk of nicotine addiction for the first few years." |
| **QUITTING SELF-EFFICACY**[*acdefg*] | "Whether or not I quit smoking cigarettes completely in the next month would be entirely up to me" |
| | "How much personal control would you feel you have over quitting smoking completely in the next month?" |
| | "Quitting cigarettes in the next month would be…" |
| | "How confident would you be that you could quit smoking completely in the next month if you really wanted to?" |
| | "How certain would you be that you could quit smoking completely in the next month if you really wanted to?" |
| | "How difficult would it be for you to quit smoking cigarettes in the next month?" |
| **WORRY**[*h*] | "How much do you worry about…getting lung cancer in the future? |
| | "How much do you worry about…having a heart attack in the future?" |

| Scale | Question |
|---|---|
| **FEELINGS OF RISK**[a] | "How much do you worry about…becoming addicted to nicotine within in the next year?" |
| | "I feel that I will develop lung cancer in the future if I continue to smoke." |
| | "I feel that I will have a heart attack in the future if I continue to smoke." |
| | "I feel that I will get addicted to nicotine in the next year if I continue to smoke." |

*Note*: Response options were as follows:

[a] Strongly disagree, Slightly disagree, Neither agree or disagree, Slightly agree, Strongly agree.

[b] Not at all, A Little, Somewhat, Very, Extremely.

[c] No control, A little control, Some control, A lot of control, Total control.

[d] Not at all possible,A little possible,Somewhat possible, Very possible, Completely possible.

[e] Not at all confident, A little confident, Somewhat confident, Very confident, Completely confident.

[f] Not at all certain, A little certain, Somewhat certain, Mostly certain, Completely certain.

[g] Not at all difficult, A little difficult, Somewhat difficult, Very difficult, Extremely difficult.

[h] Not at all worried, A little worried, Somewhat worried, Very worried,Extremely worried.

**Table 2**

**Effects of experimental condition on attitudes**

| | | | | | | Experimental Condition | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Genetic Basis | | No Genetic Basis | | Attention Control | |
| **Outcome Variable** | **Cronbach α** | **F** | **df** | **p** | **$\eta^2$** | **Mean** | **SD** | **Mean** | **SD** | **Mean** | **SD** |
| Addiction and Quitting Beliefs Subscales | | | | | | | | | | | |
| Amount to addiction | 0.82 | 0.5 | 313 | .62 | 0.003 | 3.4 | 0.9 | 3.2 | 1.1 | 3.3 | 1.1 |
| Can avoid addiction | 0.62 | 0.3 | 313 | .77 | 0.002 | 1.9 | 1.4 | 1.8 | 1.4 | 1.8 | 1.4 |
| Judgment | 0.64 | 0.3 | 313 | .71 | 0.002 | 1.5 | 0.3 | 1.5 | 0.3 | 1.5 | 0.3 |
| Deterministic addiction | 0.46 | 0.3 | 313 | .73 | 0.002 | 3.2 | 1.0 | 3.1 | 1.0 | 3.1 | 1.0 |
| Agentic quitting | 0.55 | 0.7 | 313 | .47 | 0.005 | 3.5 | 0.8 | 3.6 | 0.8 | 3.6 | 0.7 |
| Delayed harm | 0.76 | 0.6 | 325 | .53 | 0.004 | 1.2 | 1.0 | 1.2 | 1.0 | 1.2 | 1.0 |
| Quitting self-efficacy | 0.86 | 0.4 | 328 | .67 | 0.002 | 4.3 | 3.9 | 4.3 | 3.9 | 4.2 | 3.9 |
| Worry | 0.87 | 0.1 | 325 | .93 | 0.000 | 2.0 | 1.6 | 2.1 | 1.7 | 2.0 | 1.7 |
| Feelings of risk | 0.81 | 2.5 | 326 | .08 | 0.015 | 2.9 | 1.1 | 3.2 | 1.0 | 3.1 | 1.1 |
| Quitting intentions | -- | 0.2 | 326 | .79 | 0.001 | 4.3 | 4.1 | 4.3 | 4.1 | 4.2 | 4.2 |

*Note*. No means were statistically significantly different from each other. All means and SDs presented here were backtransformed from the transformation used to construct a more normal distribution. Log10 transformation was performed on can avoid addiction, worry, and delayed harm. Square root and exponential transformations were performed on judgment and quitting self-efficacy, respectively. The remaining variables were not transformed.