The effects of learning about one’s own genetic susceptibility to alcoholism: a randomized experiment

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Purpose: Increased accessibility of direct-to-consumer personalized genetic reports raises the question: how are people affected by information about their own genetic predispositions?

Methods: Participants were led to believe that they had entered a study on the genetics of alcoholism and sleep disorders. Participants provided a saliva sample purportedly to be tested for the presence of relevant genes. While awaiting the results, they completed a questionnaire assessing their emotional state. They subsequently received a bogus report about their genetic susceptibility and completed a questionnaire about their emotional state and items assessing perceived control over drinking, relevant future drinking-related intentions, and intervention-related motivation and behavior.

Results: Participants who were led to believe that they had a gene associated with alcoholism showed an increase in negative affect, decrease in positive affect, and reduced perceived personal control over drinking. Reported intentions for alcohol consumption in the near future were not affected; however, individuals were more likely to enroll in a “responsible drinking” workshop after learning of their alleged genetic susceptibility.

Conclusion: The first complete randomized experiment to examine the psychological and behavioral effects of receiving personalized genetic susceptibility information indicates some potential perils and benefits of direct-to-consumer genetic tests.

Key Words: alcoholism; direct-to-consumer; personalized genetics; randomized experiment; susceptibility information

INTRODUCTION

In recent decades, much interest has been directed toward identifying specific genes as potential causes of particular conditions, behaviors, and diseases. During this period, the cost of genotyping has decreased considerably and the number of companies offering testing services has dramatically increased. Despite these advances, no randomized experiments have examined psychological and behavioral effects of receiving personalized genetic susceptibility information. This is the first such experiment.

More than 1,600 tests designed to identify genes associated with specific disorders are available.1 The continuous reduction in the cost of these ever-expanding tests may lead to the incorporation of genomic information as part of personalized preventative care.2 However, the clinical value of such information has been challenged.3 Even single-gene disorders, once thought to allow easy risk assessment, have proven to be less tractable than anticipated.4

Despite such disappointments, genetic research has captured public attention. Relevant findings have been featured by the media with notable zeal, proclaiming breakthrough evidence that links specific genes, markers, or chromosomal abnormalities with a variety of human diseases and disorders (e.g., schizophrenia),5 tendencies (e.g., criminality),6 and behaviors (e.g., novelty seeking).7 Unfortunately, these findings were often followed by replication failures.8 Scientists have developed a more intricate understanding of the nature of the relationship between genotypes and phenotypes, whereas lay people have not, reflecting their largely inaccurate mental representations of genetics.9,10

The perception of genetic etiology engenders cognitive fallacies, termed genetic essentialist biases. Specifically, when a particular disease or behavioral tendency is considered “genetic,” essentialist biases lead people to view these phenomena as more immutable, predetermined, and natural.9 Essentialist biases also decrease the acknowledgment of environmental causes and personal choice.9 With regard to health-related information, research indicates that individuals who make genetic attributions for health-related conditions perceive cardiovascular conditions as more life threatening,11 manifest greater desire for social distance from a person with mental illness,12 and expect poorer prognosis with respect to medical conditions.13 Thus, perception of genetic etiology is linked to a variety of determinism-enhancing beliefs.

Stronger causal inferences regarding the effects of genetic attributions come from experimental designs. Exposure to narratives that introduce genetic explanations has been shown to prompt a slew of undesirable effects, including an increase in ethnic out-group dislike,14 a decrease in intentions to exercise,15 an increase in acceptance of sexual crimes,16 and a decrease in...
women's performance on math. However, these studies manipulated people's general etiological beliefs. Indeed, no prior study has used a complete randomized experimental design to examine the effects of receiving personalized genetic information.

Quasi-experimental and randomized block experimental design research has been conducted but produced mixed results. Addressing emotional responses, some studies indicate elevation in general or disease-specific distress in individuals after learning about their genetic susceptibility. These effects have been found for various time spans ranging from immediately after the results are given, to weeks and months later. However, other studies found no such effects. Behavioral measures similarly show mixed findings, with some studies indicating an increase in vigilant health behaviors following carrier indication and some that do not. These mixed results may reflect, in part, variation in sampling (e.g., patient populations vs. nonpatient), designs (quasi-experimental vs. randomized block), targeted disease (e.g., those with and without a known environmental component), or time of outcome measurement (e.g., short vs. long term). Taken together, they reveal the complex effects of learning about one's genetic susceptibility to diseases. Despite these differences, the common element in all these studies was that the susceptibility information was delivered by trained professionals. Nonetheless, direct-to-consumer (DTC) genomic analysis companies offer susceptibility information to individuals without the interpretive support of trained personnel.

This genomic information has become increasingly affordable in recent years. Thus, despite challenges to the validity of the risk assessments and clinical utility of reports provided by DTC companies, the opportunity to obtain information on one's own "book of life" continues to drive this burgeoning industry. Only recently have the effects of such information attracted researchers' attention.

The most comprehensive (but nonexperimental) study to evaluate the affective and behavioral impact of DTC genomic profiling found little evidence that such reports have significant effects on changes in anxiety, fat intake, exercise, or medical screening intentions in the first 3 months after receiving the results. The ability to draw strong inferences from this study, an important first step in exploring the effects on DTC genomic reports in its own right, was limited by the nature of the quasi-experimental design, the nature of the narrow population that was sampled (paying DTC customers), and the rate of attrition (more than 50%). In addition, the investigation did not assess the immediate impact of the results. Finally, the study addressed the cumulative effects of DTC reports regardless of individuals’ specific susceptibility information; i.e., the effects of specific genetic susceptibility risk estimates were not examined.

To address some of these limitations, we used a randomized experimental design that eliminates most of the quasi-experimental shortcomings. This study was designed to assess affective, attitudinal, motivational, and behavioral effects of simultaneously learning about one's own and others' genetic susceptibility to alcoholism. We adopted an experimental approach using deception, whereby participants were randomly assigned to receive different forms of bogus information about their genetic susceptibility to alcohol disorders. Genetic susceptibility to alcoholism was chosen for four reasons. First, genes have received much attention from the alcohol research community in the past couple of decades. During this time, the media has highlighted much of the research that purportedly found genes associated with alcoholism, thus raising public awareness of this issue. Second, genetic susceptibility to alcoholism is already featured in DTC reports available to the public. Third, the population we sampled (college students) is especially susceptible to problem drinking behaviors. Fourth, unlike many other diseases, alcohol consumption is voluntary and, as such, can be mitigated using behavioral modifications.

### METHOD

#### Participants

A total of 160 undergraduates (101 women; mean age = 20.51, SD = 3.82, predominantly white [96] and Asian [37]) participated in return for course credit.

#### Procedure

Participants arrived at an agreed upon meeting place at a hospital to take part in a study titled "The genetics of sleep disorders and alcoholism." An experimenter wearing a white lab coat escorted them to a lab room. On the way to the lab, the experimenter stirred the conversation toward the exciting advancements in the field of genetics, indicating that genetic tests are now widely available and can be conducted quickly. Upon arrival, individuals were asked to sign an informed consent form, which indirectly facilitated the deception. Specifically, the participant read that, they would be paired with "another participant" (who did not exist) to design an advertisement for a responsible drinking workshop for students. This was mentioned in part to prepare the participant to receive bogus genetic information about the faux other participant (for reasons discussed in the following). After they signed the form, participants provided a saliva sample to be allegedly tested for a gene associated with alcoholism and a gene associated with sleep disorders. They were told that the test takes ~15 min.

While their saliva was purportedly being genotyped, participants were asked to complete a questionnaire package that assessed, among other elements, their present emotional state. Fifteen minutes later, the experimenter returned to the room and told the participants that the genotyping test is taking longer than expected because a new technician had just been hired. This procedure was designed to increase participants’ belief that an actual test was being conducted and set the stage for an upcoming "error." About 5 min later, the experimenter returned with the alleged test results in a sealed envelope and left the participant alone to read the genotyping results while allegedly delivering test results to the other participant.

A couple of minutes later, the experimenter returned looking alarmed. The experimenter apologized and indicated that the new technician inadvertently shuffled the sealed envelopes,
so that the participant had received someone else’s results. The experimenter indicated that this other participant drew their attention to the switch, noticing the wrong patient’s name on the test results form.

The experimenter handed the participant their alleged “real” results (with their name on the form). They took the “other participant’s” test results and left to deliver these results. All the forms indicated that the participants (real and faux) did not have a gene associated with sleep disorders. Approximately half (52%) of the forms indicated that the “other participant” (who was matched for gender with the real participant) had a specific gene associated with alcoholism and the other half indicated no such susceptibility. Similarly, approximately half (48%) of the participants received a bogus indication of their own alcoholism susceptibility and the other half learned they do not have the susceptibility allele. This 2 (”other participant’s” genetic susceptibility, dichotomized) × 2 (real participant’s genetic susceptibility, dichotomized) randomized experimental design enabled us to examine the effects of simultaneously learning about one’s own and someone else’s genetic susceptibility to alcoholism. We stratified the random assignment of participants to these conditions by sex as previous research indicated different alcoholism rates among men and women.32

The experimenter returned about 2 min after delivering the “real results” and asked the participant to complete an additional set of questionnaires, measuring affect, beliefs, and future intentions. After leaving the participant alone for about 10 min to complete the questionnaires, they returned to escort the participant to a different lab room where the “other participant” was allegedly waiting for a debriefing. When they reached the room where the “other participant” was allegedly waiting, the real participant saw a line of six chairs with the one at the end supporting a bag indicating the “other participant’s” seat. “Noticing” the “other participant” absence, the experimenter asked the real participant to take a seat anywhere. This task (adapted from Heckel and Hiers, 1977)33 was designed to examine social distance. Although we had hypothesized that participants’ seating choice would depend on the faux participant’s perceived genetic susceptibility to alcoholism, the distance measure was not affected by any of the experimental variables, $F$s < 1, and will not be discussed further.

Once the participant sat, the experimenter left to search for the “other participant.” A minute later, the experimenter returned to assess the participant’s state of mind and thoroughly debrief him or her regarding the bogus test results and the need for deception. In addition, all participants were asked to provide their consent for the use of their data once they were informed of the true purpose of the study. A visual representation of the different stages of the experiment is presented in Figure 1.

**Figure 1 Summary of experimental design.**

Ten items assess positive emotions ($\alpha = 0.88$) and 10 items assess negative emotions ($\alpha = 0.60$).

**Drinking frequency.** Participants used a 5-point scale (1 = never; 5 = four or more times a week) to indicate frequency of drinking in response to the question “How often do you have a drink containing alcohol?” This frequency did not vary across conditions, $P > 0.1$, and using it as a covariate had little effect on the results. Therefore, it will not be discussed further.

**Postmanipulation**

**Emotional state.** The Positive Affect and Negative Affect Schedule was readministered (positive emotions $\alpha = 0.90$; negative emotions $\alpha = 0.83$).

**Control over drinking problems.** A six-item measure was constructed assessing participant’s beliefs about control over drinking problems with suggested genetic etiology (three items, e.g., “a heavy drinker with a genetic mutation associated with...”)

**Relevant measures**

**Premanipulation**

*Emotional state.* Participants’ current mood was measured using the Positive Affect and Negative Affect Schedule,34 a 20-item self-report measure to which participants respond on 5-point scales, indicating their feelings at the present moment.
drinking problems $\alpha = 0.77$) or environmental etiology (three items, e.g., “a heavy drinker whose lifestyle is associated with drinking problems” $\alpha = 0.84$). A 5-point scale was used for all items (1 = no control over drinking problems; 3 = a reasonable amount of control over drinking problems; 5 = complete control over drinking problems). Similar questions addressed insomnia to obscure the focus on alcoholism.

**Perceived personal control.** Participants indicated how much control they believe they have over their own alcohol consumption (“to what extent can you avoid drinking alcohol?”), using a 9-point scale (1 = cannot avoid it at all; 9 = can avoid it completely).

Future drinking intentions: using 3-point scales (1 = less than last month; 2 = similar to last month; 3 = more than last month), participants responded to two alcohol consumption inquiries. Specifically, they were asked to indicate how many times they intend to drink alcohol in the following month; and the highest number of drinks they intend to consume in one sitting in the following month.

**Behavioral intentions and behavior.** Participants were asked to indicate their willingness (motivation) to participate in a “responsible drinking” workshop for students (yes or no). In addition, they had the opportunity to enroll in such a workshop (actual behavior).

**Demographics.** Participants indicated their sex and ethnicity. These variables are discussed only insofar as they had a significant impact on the results.

**Analyses**
Mixed-design analyses of variance were used to examine changes in emotional states after reception of purported personal genetic susceptibility to alcoholism information, stratified by the experimental manipulation (negative or positive genetic susceptibility of self). Similar analysis explored the difference in evaluation of the perceived control over drinking problems between two target persons, one with perceived genetic etiology and the other with perceived environmental etiology for such problems. To examine whether the perception of control of these target persons was more or less than the scale’s midpoint indicating “reasonable amount,” $t$-tests were used. A between-subject analysis of variance compared perception of personal control over alcohol consumption between individuals who were led to believe that they have an allele associated with alcoholism and individuals who were led to believe that they do not. Differences in future drinking intentions between these two groups of individuals were assessed using $\chi^2$ tests. Logistic regression analyses examined the differences between these groups on willingness to participate in a responsible drinking workshop and actual enrollment. Participants’ sex is included in the reported analyses only when it played a significant role. All analyses were carried out using SPSS (Version 18; IBM, Somers, NY).

**RESULTS**

**Positive emotions**
The analysis showed a significant drop in positive affect between the pre- and the postmeasures, $F (1, 158) = 23.42, P < 0.001$. As expected, this effect was moderated by the test results manipulation, $F (1, 158) = 9.15, P = 0.003$. Follow-up analyses indicated that there was a significant drop in positive affect among individuals who learned that they have a gene associated with alcoholism (premanipulation $M$ (SD) = 2.84 (0.69); postmanipulation $M = 2.55$ (0.73)), $F (1, 76) = 29.56, P < 0.001, \eta^2_p = 0.28$; in contrast, no such effect was evident among individuals who learned they do not have this allele, $F (1, 82) = 1.73, P = 0.19$ (Figure 2).

**Negative emotions**
A similar analysis of variance indicated a significant moderation effect of test results on reported negative affect, $F (1, 158) = 13.91, P < 0.001$. Follow-up analyses showed a significant increase in negative affect among individuals who learned that they have the susceptibility allele (premanipulation $M$ (SD) = 1.27 (0.31); postmanipulation $M = 1.37$ (0.38)), $F (1, 76) = 5.62, P = 0.02, \eta^2_p = 0.07$ (Figure 3). Individuals who learned they have no such allele showed a reduction in negative affect (premanipulation $M = 1.34$ (0.51); postmanipulation $M = 1.19$ (0.32)), $F (1, 82) = 8.62, P = 0.004, \eta^2_p = 0.10$.

**Control over drinking and etiology**
The analysis showed that target persons with genetic etiology were perceived as having less control over drinking problems ($M = 2.68, SD = 0.69$) than target persons with environmental etiology ($M$ (SD) = 3.15 (0.93)), $F (1,157) = 32.9, P < 0.001, \eta^2_p = 0.17$. This effect was not moderated by the test results manipulation, $F (1, 157) < 1$, not significant. Furthermore, analyses indicated that a target person with genetic susceptibility was rated as having less than a reasonable amount of control $t (158) = −5.81, P < 0.001$, whereas a target person with environmental
antecedents was rated as having more than a reasonable amount of control over drinking problems, \( t (158) = 1.99, P < 0.05 \).

**Perceived personal control**

An analysis of variance indicated that individuals who learned that they have an allele associated with alcoholism evaluated their own ability to avoid drinking alcohol as weaker (\( M \) (SD) = 7.52 (2.14)) as compared with individuals who learned that they do not have that susceptibility allele (\( M = 8.30 \) (1.21)), \( F (1,158) = 8.26, P = 0.005, \eta_p^2 = 0.05 \). Unlike the affect measure, this result reflects a between-subject finding rather than intra-individual changes.

**Future alcohol consumption**

The majority of the participants indicated that in the following month, they planned to drink the same amount of times and similar number of drinks in one sitting as they did in the previous month (\( n = 120 \) and \( n = 121 \)). The experimental manipulation had no significant effect on the percentage of participants who indicated that they planned to consume less (\( \chi^2(1) < 1.00 \), not significant) or more (\( \chi^2(1) < 1.00 \), not significant) alcohol in the following month.

**Seeking drinking intervention**

A logistic regression indicated that participants' sex (\( B \) (se) = 2.11 (0.84), Wald = 6.31, \( P = 0.01 \)) and the experimental manipulation (\( B = 1.67 \) (0.82), Wald = 4.18, \( P = 0.04 \)) affected the willingness to participate in a responsible drinking workshop, as did their interaction (\( B = -2.36 \) (1.07), Wald = 4.86, \( P = 0.03 \)). Follow-up analyses indicated that among men, the manipulation did not significantly affect willingness to participate in such a workshop (Wald < 1.00, not significant). Among women, learning that one has an allele associated with alcoholism increased willingness to participate as compared with women who learned they do not have the allele (\( B = 1.67 \) (0.82), Wald = 4.18, \( P = 0.04 \)).

With respect to the effect on enrollment in the workshop, a logistic regression analysis indicated a significant effect for the experimental manipulation (\( B \) (se) = 1.67 (0.82), Wald = 4.18, \( P = 0.04 \)); individuals who were led to believe they have a genetic susceptibility to alcoholism were more likely to enroll as compared with those who learned they do not.

**DISCUSSION**

Markets for personalized genomic analysis are increasing continuously, yet little is known about the psychological effects of receiving personalized genetic information. Previous studies, which focused on the effects of revealing genetic susceptibility information to individuals with high family risk profiles for diseases have shown a mix of negative, positive, and null findings.\(^{18-26}\) Even among DTC customers, previous research suggested that the population effects of such reports are largely insignificant but individuals may experience adverse emotional effects after learning about certain specific diseases' susceptibilities.\(^{29}\) In an effort to draw causal inferences, this study supplemented previous research such as the REVEAL study,\(^{20,24}\) which used a randomized block experimental design, to explore effect of learning about one's own genetic susceptibility to diseases. The use of deception afforded utilization of a complete randomized experimental design in which participants learned about their own (as well as a stranger's) purported genetic susceptibility to alcoholism. The results indicate that such information affected people's emotions, perceptions of behavioral control, enrollment in a responsible drinking intervention, and (only) women's motivation for such participation. Learning about one's genetic susceptibility to alcoholism did not significantly affect intentions to drink in the near future, nor did learning...
about someone else's susceptibility significantly affect how close to that person participants were willing to sit.

It was found that learning about one's genetic susceptibility to alcoholism has immediate emotional effects. These results may point to an appropriate timing for intervention recruitment as negative emotional states increase motivation to engage in change-related behaviors. In light of these findings, it may not be surprising that some of the DTC companies offer "genetically tailored" nutrition supplements to potentially harness the emotional state of their consumers for marketing purposes.

These studies' results also indicate that individuals who learn that they have genetic susceptibility to alcoholism perceive lower personal ability to control alcohol consumption as compared with individuals who learn of no such susceptibility. This finding is important because of evidence showing that perceived behavioral control influences both behavioral intentions and actual behavior. In fact, perception of personal control was demonstrated to play a role in alcohol-related behaviors. The modification of perceived behavioral control is therefore a concern that should be addressed when genetic susceptibility information is disseminated to individual consumers, emphasizing the nondeterministic, interactive relationship between genes and environment, for example.

As compared with individuals who learned that they do not have an allele associated with alcoholism, people who learned that they purportedly have such an allele were more likely to actually enroll in a drinking intervention. A less stringent test (i.e., no measure of actual behaviors) indicated that women (but not men) with the purported allele were also more willing to participate in such a workshop as compared with women purportedly without the allele. This gender difference was not predicted. A post hoc interpretation might be that a lower social approval of drinking among women (relative to men) might have motivated our women participants who learned they had alcohol susceptibility to do something about it. Stated differently, relative to males, women might have a more negative attitude toward drinking problems; thus, learning that they have a genetic susceptibility to alcoholism is more likely to push them to action.

Previous research has shown mixed findings with regard to behavioral intentions and modifications after learning of increased genetic susceptibility to diseases. Some researchers suggest that increased disease risk, which may be rooted in genetic susceptibility information, should increase vigilant behaviors. Others have pointed out that the increased fatalism that often accompanies genetic attributions may reduce vigilant behaviors as the genetic influence is interpreted as destiny. Research aimed at reconciling these inconsistencies is needed, as is further theoretical development to examine the moderators involved in behavioral changes following indications of genetic susceptibility.

The nature of the experimental design implies that these studies' findings provide a strong indication that learning about personalized genetic susceptibility to diseases and tendencies can have immediate effects. The affective, cognitive, and behavioral changes that followed the revelation of genetic susceptibility to alcoholism are arguably derived from people's cognitive biases associated with perceived genetic etiology as evident by the clear etiology-based differentiation of the amount of control associated with drinking problems. This study extends previous research on these biases by demonstrating their applicability for personalized genetic information as well.

Despite the strengths of the experimental design, this study does have certain limitations. First, the genetic information was confined to alcoholism and sleep disorders. Most DTC reports, however, provide much broader susceptibility information, often covering dozens of tendencies and diseases. The focus on alcoholism susceptibility allows more specific conclusions but at a cost of ecological validity. Second, there may be differences between our sample of college students and the individuals who obtain DTC genetic analysis reports. The individuals who pay for DTC genomic profiling may be more likely to be affected by the information they obtain than the less invested student sample. This limitation indicates that our findings may underestimate the effect one might expect from DTC clientele. However, our findings may reflect the effects of such information once personalized genomics becomes part of regular medical practice. Third, our sample was composed primarily of individuals of European and Asian ethnicities. Future research is needed to address the generalizability of these findings to other ethnicities. Finally, this study was limited to immediate impact without assessing longitudinal effects. This limitation is an outcome of obvious ethical considerations that limit the length of deception used to the time the participant is under supervision.

Conclusion
This study unequivocally reveals that learning about one's own genetic susceptibility to specific diseases has psychological consequences. It expands the growing area of the ethical, legal, and social implications of the genomics revolution to include emotional, motivational, and behavioral changes following receipt of genetic information. It also points toward the need for theoretical development to explain past inconsistent findings.

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DISCLOSURE
The authors declare no conflict of interest.

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