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Testing Different Communication Formats on Responses to Imagined Risk of Having vs. Missing the GSTM1 Gene

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Abstract

Genetic markers of lung cancer susceptibility, such as the common variant of the glutathione S-transferase Mu 1 gene (*GSTM1*-null), confer small probabilities of disease risk. We explored the influence of different approaches to communicating the small variations in risk associated with this biomarker. College smokers ($N = 128$) imagined that they had the *GSTM1* wild-type vs. the *GSTM1* null-type variant. We presented lung cancer risk in six ways that varied the risk format (absolute risk vs. incremental risk) and the presentation style of the information (no graphics vs. graphic display of foreground only vs. graphic display of foreground + background). Presentation style had minor effects. However, absolute risk information increased negative emotions more than did incremental risk information. Perceptions of risk and negative emotions were most profoundly affected by the difference between having the *GSTM1* wild-type vs. the *GSTM1* null-type variant. We discuss implications for conveying small probabilities related to genetic risk.

Keywords

Negative emotions; risk perceptions; GSTM1; lung cancer; genes; risk communication; fuzzy trace theory

A central and well supported component of many health models (e.g., the health belief model, the theory of planned behavior, protection motivation theory, social learning theory) is that increasing perceived health risk can motivate positive health behavior changes such as smoking cessation (Bowen, Helmes, Powers, Andersen, Burke, Mctiernan et al., 2003; Cummings, Becker, & Maile, 1980; Fishbein, 1980; Rippetoe & Rogers, 1987; Rosenstock, Stretcher, & Becker, 1988). To this end, health campaigns increasingly inform smokers of their health risks via biologically-based feedback and biomarkers. These include exposure

levels to chemical constituents of harm in tobacco smoke (e.g., carbon monoxide (CO), nitrosamines), indicators of physiological damage (e.g., pulmonary functioning), and genetic markers of susceptibility to tobacco-related diseases (Bize, Burnand, Mueller, Rège, & Cornuz, 2009; McClure, 2002, 2004). The benefit of genetic biomarkers of susceptibility is that they can indicate chance for harm before harm occurs. Nonetheless, a recent Cochrane review of randomized controlled trials revealed that providing smokers with DNA-based genetic susceptibility feedback do not have higher cessation rates relative to comparison groups (Marteau, French, Griffin, Prevost, Sutton, Watkinson, Attwood, & Hollands, 2010). Although many factors likely contribute to these null findings (e.g., small sample sizes resulting in low statistical power, nature of the comparison group, etc.), one area that requires more investigation is how smokers interpret small increases in genetic risk conferred by many of these markers, such as the glutathione S-transferase Mu 1 gene (*GSTM1*).

GSTM1, which encodes the GSTM1 enzyme, is one of several genes involved in the detoxification of the carcinogens that increases risk for lung cancer, albeit by a small amount (Sobti, Kaur, Kaur, Janmeja, Jindal et al., 2008; Vineis, Anttila, Benhamou, Spinola, Hirvonen et al., 2007). Meta-analytic research links this common gene variant (called *GSTM1*-null or *GSTM1*-missing) with increased lung cancer risk (odds ratio = 1.17, 95% CI, 1.07–1.27) (Benhamou, Lee, Alexandrie, Boffetta, Bouchardy et al., 2002). Assuming that about 10 out of 100 smokers develop lung cancer, we expect on average one to two more to develop lung cancer among smokers who have the *GSTM1* null-type variant, and one to two fewer to develop lung cancer among smokers who have the *GSTM1* wild-type variant. At the population level, the small increase in risk (i.e., 1% to 2%) is quite important. In the United States where approximately 21% of adults smoke (Pleis, Lucas, & Ward, 2009), a 1% increase means 33,000 additional smokers who have the *GSTM1* null-type variant will get lung cancer over their lifetime, and that 33,000 fewer smokers who have the *GSTM1* wild-type variant will get lung cancer over their lifetime. However, when viewed at an individual level, the small increase in risk from having the *GSTM1* null-type variant likely seems negligible to most people and insufficient to motivate cessation. However, even small probability events can have meaning; indeed, research based on prospect theory (Kahneman & Tversky, 1979) shows that people often overweigh small probabilities events they have not experienced (Hertwig, Barron, Weber, & Erev, 2004; Hertwig & Erev, 2009). Thus, from a communications perspective, the challenge is how to make small probability events, such as the slightly elevated risk from having the *GSTM1* null-type variant, persuasive to motivate quitting.

One approach to addressing this challenge is to capitalize on the context of the information, which provides a reference point that influences interpretation. For small probability events, risk can seem relatively large or relatively trivial depending on the context, and changing the context can change risk aversion and the perceived riskiness of a behavior (Stone, Sieck, Bull, Yates, Parks & Rush, 2003). This context effect was illustrated in a study by Stone and colleagues that provided participants information about gum disease risk in response to using a standard versus improved toothpaste (Stone et al., 2003, Study 1). Participant in the *numeric* condition received frequency information describing the number of users affected and unaffected by gum disease as a result of using the standard versus improved toothpaste (30 out of 5,000 versus 15 out of 5,000). Participants in the *foreground + background* condition received the same information as participants in the numeric condition plus a graphical display that depicted both the number of people affected and unaffected. Finally, participants in the *foreground only* condition received the numeric information plus a graphical display depicting only the number of gum disease cases (30 versus 15) resulting from using the standard versus improved toothpaste. Compared with numeric information

and foreground + background information, the foreground only information increased perceived risk aversion, as assessed by willingness to pay more for the improved product.

This study illustrates that using numeric and graphical displays to highlight the number of people harmed makes salient a different reference point which in turn produces a change in risk aversion. In the foreground condition, the graphical display highlighted the number of people affected, inducing people to compare 15 cases of gum disease with the referent point 30 cases. In the foreground + background condition, the graphical display highlighted the number of people affected relative to the number unaffected, thereby inducing people to compare 15 cases of gum disease with the referent point of 5000 people total. A reduction of fifteen cases of gum disease seems relatively large when compared with 30 (relative risk reduction of 50%), but relatively small when compared with 5000.

In sum, for small probability events, highlighting both foreground (i.e., number affected) and background (i.e., total at risk) information makes a risk seem trivial compared with highlighting only foreground information, especially when presenting risk graphically. Indeed, graphical displays are better suited than are numbers to capture attention and make discrimination among data elements (Chua, Yates, & Shah, 2006; Jarvenpaa, 1990). We anticipate that graphic displays that shift attention to different reference points will have similar effects on perceived risk of lung cancer in response to having different variants of *GSTM1*. Specifically, graphic displays that highlight the number of people expected to get lung cancer due to different variants of *GSTM1* (foreground only) should evoke greater effects than graphic displays that highlight the number of people expected to get lung cancer relatively to the number unaffected.

A second way to contextualize information is by varying whether information presents the absolute number of people harmed or the incremental increase and decrease in the number of people harmed (Zikmund-fisher, Ubel, Smith, Derry, McClure et al., 2008). For example, for the *GSTM1* null-type variant, presentations can highlight the 11 smokers expected to get lung cancer with the *GSTM1* null-type variant versus the 9 expected to get lung cancer with the *GSTM1* wild-type variant. Assuming about 10 out of 100 smokers develop lung cancer, we can highlight the additional one or two smokers expected to develop lung cancer with the *GSTM1* null-type variant as well as the one to two fewer smokers expected to develop lung cancer with the *GSTM1* wild-type variant. Once again, although the basic information is the same, the different displays subtly shift the reference point from focusing on 9% versus 11% to focusing on 1%. In general, for small probability events, highlighting the absolute number of people harmed or not should prompt greater perceptions of risk than should highlighting incremental changes in risk (Zikmund-fisher et al., 2008).

It is important to note that people do not merely form judgments based on concrete risk information (e.g., the number of smokers at greater or less risk due to *GSTM1*). According to Fuzzy Trace Theory (Reyna, 2008), people also form judgments based on the general meaning or *gist* they derive from the information. When evaluating *GSTM1* risk information, the gist likely represents two conclusions: 1) that the risk overall is small; and 2) the *GSTM1* null-type variant conveys more risk than does *GSTM1* wild-type variant, and is therefore, bad. Often the gist weighs more heavily than concrete information in perceptions of risk.

Fuzzy Trace Theory offers important predictions. First, we should see only minor differences in perceptions of risk as a function of display format. If the displays do justice in conveying that the risk of lung cancer is small across gene variants, and if people respond to the bottom line gist, then study participants should discriminate little between the display conditions – unless, they are explicitly instructed to make fine distinctions. Second, people

should respond strongly – reporting greater perceived risk and greater negative emotions – to the gist of the information that lung cancer risk is higher with the *GSTM1* null-type variant than with the *GSTM1* wild-type variant. Third, the gist that the *GSTM1* null-type variant is more harmful than the *GSTM1* wild-type variant should be accompanied by more negative emotional responses (e.g., worry, fear, anxiety) and covary with greater perceptions of risk. Indeed, findings from the psychometric paradigm (Slovic, 1992), the Risk as Feeling model (Loewenstein, Weber, Hsee & Welch, 2001), and the affect heuristic (Slovic, Peters, Finucane, & MacGregor, 2005) all document that perceived riskiness of activities are influenced by emotional responses (e.g., dread). Of import, emotional reactions are in turn related to a greater desire to quit (Dijkstra & Brosschot, 2003) Lipkus & Prokhorov, 2007). In sum, based on Fuzzy Trace Theory, the effects of different presentation forms should be relatively minor compared with the possibility that one has the *GSTM1* null-type versus the *GSTM1* wild-type variant. These weak effects should apply especially towards information that conveys incremental changes in risk magnitude because incremental changes would make risks seem even more negligible.

Overview and Hypotheses

Similar to Stone et al., we varied the context of risk information by presenting it either numerically (using frequencies) or graphically (using a matrix of 100 stickmen). Within the graphic conditions, we presented either the number of people expected to get lung cancer based on variants of *GSTM1* (i.e., foreground information only) or the number of people expected and not expected to get lung cancer based on variants of *GSTM1* (foreground + background). Finally, we varied whether participants viewed information about the absolute number of people harmed (9 or 11 out of 100) or the incremental increases or decreases in the number harmed (one more or less harmed out of 100). The six versions (V1 – V6) we tested appear in Figure 1.

We tested three hypotheses. First, we hypothesized that participants would report greater perceived risk and negative affect in the foreground only condition (V2 and V5) than in the numeric (V1 and V4) and in the foreground + background conditions (V3 and V6). Second, we hypothesized that participants would report greater perceived risk and negative affect in the absolute condition (V1, V2 and V3) than in the incremental conditions condition (V4, V5 and V6). Both hypotheses are consistent with prior research (by Stone et al., 2003) and with our reasoning that the referent point of people unaffected makes personal harm seem relatively trivial. Third, consistent with Fuzzy Trace Theory (Reyna, 2008), we hypothesized that our biggest effects would emerge in response to imagining the *GSTM1* null-type and wild-type variant. Specifically, participants would report greater perceived risk and greater negative emotions in response to imagining the *GSTM1* null-type than the *GSTM1* wild-type variant.

We tested the effects of our formats on perceptions of risk and emotional reactions to the feedback in college smokers. We chose this population for three reasons. First, college smokers represent an important target for smoking intervention; the stress associated with the transition to college, as well as the lack of parental supervision, may increase the likelihood that the habit of smoking becomes entrenched. Second, younger smokers do not typically experience severe smoking-related symptoms. While they acknowledge risks of smoking for others, they often are optimistic about their own risk of personally experiencing these harms (Hansen & Malotte, 1986; Slovic 2001, 2003). Third, the response of college-age smokers to genetic susceptibility testing for lung cancer remains unknown. If one or two formats show positive effects of increasing risk and worry while reducing optimistic biases, researchers could evaluate the findings in larger randomized controlled trials to motivate cessation in this population.

Methods

Study eligibility and participants

Participants were college smokers at the University of Florida, ages of 18–21, who had smoked at least one cigarette during the last week and at least 50 cigarettes in their lifetime. We chose a minimum of 50 cigarettes to include people who were experimenting with smoking and thus for whom the issue of genetics and smoking might be at least somewhat personally relevant. We recruited participants either by approaching smokers on campus where they congregated, through Craig's List (an internet site), or through campus advertisements (i.e., flyers, newspapers). Smokers who expressed interest completed a brief screener. Smokers who were eligible and consented, completed a baseline survey, and then came to a psychology lab to evaluate a brochure describing genetic testing for lung cancer susceptibility (*GSTM1*). This report describes findings from the 128 smokers who evaluated the brochure. Their baseline characteristics appear in Table 1.

Procedure and measures

Participants were randomly assigned to conditions in a 2 (Type of Risk Information) x 3 (Presentation Style) between subjects factorial design. On arrival at the lab, the experimenter seated participants at a computer displaying a web-based version of a paper brochure. Because people are increasingly using the web to gain health information, we opted to present the brochure on the web. The experimenter instructed participants to read the brochure at their own pace and then complete a web-based questionnaire. The brochure first provided an overview of genes and genetic tests and described the *GSTM1* gene. Next it described how the *GSTM1* enzyme produced by the *GSTM1* gene is involved in the removal of toxins in the body from tobacco smoke. The brochure stated that between 30% and 50% of people are missing the gene and hence the enzyme, and that missing the gene raises a smoker's risk of lung cancer. Because they were likely unfamiliar with alleles, participants received simplified (albeit technically inaccurate) descriptors of having vs. lacking the gene.

Following this information were the manipulations of *Type of Risk Information* and *Presentation Style*. The Risk Information manipulation varied whether participants learned the absolute vs. incremental risk of lung cancer associated with having the *GSTM1* wild-type variant vs. the *GSTM1* null-type variant. In the *absolute* condition, the brochure explained that, out of 100 smokers with the *GSTM1* null-type variant, 11 are expected to get lung cancer in their lifetime, and that out of 100 smokers with the *GSTM1* wild-type variant, 9 are expected to get lung cancer in their lifetime. In the *incremental* condition, the brochure explained that, out of 100 smokers, 10 are expected to get cancer in their lifetime, but that among smokers with the *GSTM1* null-type variant, one more will get lung cancer, and among smokers who have *GSTM1* wild-type variant, one fewer would get lung cancer. The purpose of the incremental manipulation was to focus attention of the absolute value of the incremental decrease or increase in lung cancer risk.

The Presentation Style manipulation varied how the brochure visually depicted the risk information to participants (See Figure 1). In the *no display* condition, participations received no visual depiction to supplement the numerical risk information. In the *foreground only* condition, the brochure presented only the number of people affected by having the *GSTM1* wild-type variant vs. the *GSTM1* null-type variant. For example, the absolute risk condition displayed a set of 9 stickmen and a set of 11 stickmen depicting the number of smokers out of 100 expected to get lung cancer as a result of having the *GSTM1* wild-type variant vs. the *GSTM1* null-type variant. The incremental condition displayed a set of 10 and a set of 11 stickmen. With *GSTM1* wild-type variant, one of the 10 stickmen appeared

in a different pattern illustrating that one less smoker was expected to get lung cancer. With *GSTM1*/null-type variant, one of the 11 stickman appeared in a different pattern to illustrate that one more smoker was expected to get lung cancer. The *foreground + background* condition was identical to the foreground condition except that the brochure displayed two sets of 100 stickmen associated with having the *GSTM1* wild-type variant vs. the *GSTM1* null-type variant. The additional stickmen in each set were black, indicating the number of smokers who would not get lung cancer.

After reading the brochure, participants completed the following measures relevant to this report:

Risk estimates—Participants estimated their risk of lung cancer using two items that are similar to measures typically used in risk research (e.g., Weinstein & Klein, 1995). These items asked, “Overall, if you have the *GSTM1* enzyme, what is your risk of getting lung cancer?”, and “Overall, if you are missing the *GSTM1* enzyme, what is your risk of getting lung cancer?” Participants responded to both items using a 5-step scale (1 = *much lower than average*; 5 = *much higher than average*).

Negative Emotions—We asked participants to imagine that they had the *GSTM1* null-type variant. They then reported on a 7-step scale the extent to which they would feel nervous, fearful and regretful (1 = *not at all*; 7 = *a great deal*). These type of adjectives are commonly used in measures of negative emotions such as the PANAS and the STAI (Mackinnon, Jorm, Christensen, Korten, Jacomb, & Rogers, 1999; Spielberger, Gorsuch & Lushene, 1970). The items were averaged together to form a measure of negative emotions about having the *GSTM1* null-type variant ($\alpha = .89$). We then asked participants to imagine that they had *GSTM1* wild-type variant and responded again to the same three items. These items were averaged together to form a measure of negative emotions about having *GSTM1* wild-type variant ($\alpha = .88$).

Analyses

We analyzed responses to our measures of risk and our measures of negative emotions using a mixed model simultaneous regression analysis in which Years of Smoking (after centering to control for multicollinearity) was entered as a covariate, Type of Risk Information and Presentation Style were treated as between-subjects variables and the questions corresponding to having the *GSTM1* wild-type variant vs. the *GSTM1* null-type variant were treated as the within-subject variables.

Results

Risk Estimates

Analysis of participants' risk estimates yielded a significant within-subjects main effect of Imagined Gene Status, $F(1, 120) = 983.65$, $p < .0001$, $d = 2.77$. Consistent with Hypothesis 3, participants reported that they faced a higher risk of getting lung cancer if they had the *GSTM1* null-type variant ($M = 4.0$, $SD = .47$) than if they had the *GSTM1* wild-type variant ($M = 2.2$, $SD = .52$). Analysis also revealed a marginally significant main effect of Presentation Style, $F(1, 120) = 2.66$, $p < .08$, $d = .27$. This main effect, however, was qualified by a marginally significant Presentation Style x Imagined Gene Status Interaction, $F(1, 120) = 2.76$, $p = .06$, $d = .23$ (see Table 2). Contrary to Hypothesis 1, when asked to imagine that they had the *GSTM1* wild-type variant, participants reported lower risk of lung cancer in the foreground only condition than in the foreground + background condition, $d = .76$. However, when asked to imagine that they had the *GSTM1* null-type variant, participants reported no difference in lung cancer risk as a function of condition, $d = .19$.

Analysis revealed no support for Hypothesis 2; although the means were in the predicted direction, participants in the absolute ($M = 3.1$) and incremental conditions ($M = 3.0$) did not differ in their lung cancer risk estimates.

Finally, we compared participants' responses to the two items to the scale midpoint (3 = *same average risk as others*). Dependent t-tests revealed that, when asked to imagine that they had the *GSTM1* wild-type variant, participants reported a mean risk estimate that was significantly lower than average, $t(127) = 17.56$, $p < .0001$, $d = 1.54$. Conversely, when asked to imagine that they had the *GSTM1* null-type variant, participants reported a mean risk estimate that was significantly higher than average, $t(127) = 23.63$, $p < .0001$, $d = 2.09$. Both findings are consistent with Hypothesis 3.

Negative Emotions

Analysis of the negative emotions measure yielded only two significant effects (see Table 3). First, consistent with Hypothesis 2, participants reported greater negative emotions in the absolute condition ($M = 3.4$, $SD = 1.25$) than in the incremental condition ($M = 3.0$, $SD = 1.07$), $F(1, 120) = 4.80$, $p < .05$, $d = .34$. This finding suggests that directing attention to the absolute number of smokers with the *GSTM1* wild-type vs. the *GSTM1* null-type variant who get lung cancer fosters greater negative emotions than directing attention to the incremental increase and decrease in lung cancer cases associated with having the *GSTM1* wild-type vs. the *GSTM1* null-type variant. Second and consistent with Hypothesis 3, participants reported that they would feel negative emotions more strongly if they had the *GSTM1* null-type variant ($M = 4.5$, $SD = 1.73$) than if they had the *GSTM1* wild-type variant ($M = 2.0$, $SD = 1.07$), $F(1, 120) = 274.86$, $p < .0001$, $d = 1.6$. Although the finding itself is perhaps not surprising, the magnitude of the effect was greater than the objective risk seems to warrant.

Finally, it is noteworthy risk estimates and negative emotions were uncorrelated both when participants imagined having the *GSTM1* wild-type variant ($r = .00$) and when participants imagined having *GSTM1* null-type variant ($r = -.11$). These null correlations account for the general lack of correspondence in findings for the risk estimates and negative emotions.

Discussion

We explored whether the context of risk information influences negative emotions and risk perceptions. To our knowledge, this study is the first to examine the psychological consequences of genetic risk feedback regarding lung cancer risk among smokers, and whether the consequences depend on the method of communication of the risk information. Our data yield several findings. First, presentation style influenced risk perceptions, but not negative emotions and in a direction counter to predictions. Contrary to Hypothesis 1 and to other research testing the foreground-background hypothesis (Stone et al., 2003), when imagining that they had the *GSTM1* null-type variant, participants reported lower risk of lung cancer in the foreground only condition than in the foreground + background condition. Importantly, the effect was only marginally significant and thus should be interpreted cautiously.

Second, consistent with Hypothesis 2 and with prior research (Zikmund-fisher et al., 2008), type of risk information influenced negative emotions. Participants reported greater negative emotions when we presented lung cancer risk in absolute terms compared with incremental terms. As noted in the introduction, we suspect that attending to incremental changes trivializes the influence of genetic variations in lung cancer risk. Of note, while this effect emerged for negative emotions, it did not emerge for risk perceptions, possibly because of lack of associations between these constructs.

Finally, consistent with Hypothesis 3, participants reported that they would face higher lung cancer risk if they had the *GSTM1* null-type variant than if they had the *GSTM1* wild-type variant. They likewise reported that they would experience stronger negative emotions if they had the *GSTM1* null-type variant than if they had the *GSTM1* wild-type variant. These two findings suggest that our participants were sensitive to the difference in gene status. Moreover, participants' attention to the difference between having the *GSTM1* wild-type vs. the *GSTM1* null-type variant may explain why type of risk information did not affect risk perceptions, and why our other effects were small. Simply put, unlike the Stone et al. study, our discussion of the *GSTM1* variants may have so focused participants on the difference between having the *GSTM1* wild-type versus the *GSTM1* null-type variant that they paid less attention to our numeric format and presentation style manipulations.

These findings provide partial support for fuzzy trace theory while also providing boundary conditions for the foreground-background hypotheses. Consistent with fuzzy trace theory, participants reacted more powerfully to the underlying gist conveyed by different *GSTM1* variants than they did to the small absolute differences in risk conveyed across formats. This more powerful reaction also manifested in participants' greater negative emotional response. The strong emotional response perhaps is ultimately a more important take-home public health message than whether participants attend to details of the differences in risk. With respect to the foreground-background hypotheses, prior work tested very small risks (e.g., 15 cases in 5000) but large risk *differences* between conditions (e.g., 15 vs. 30 cases). In our study, the absolute risks were substantially larger than prior work (e.g., 9 cases out of 100), but the differences between conditions (9 vs. 11 cases) were small. It is possible that our findings would more closely replicate prior findings were the absolute risks and the risk differences more in line with prior research (e.g., larger denominator that would coincide with larger numerators). In addition, our findings may have more closely replicated prior findings had we used a within subject design where the differences in formats would appear more pronounced.

Limitations

Our study has several limitations. First, our undergraduate sample differs from other smokers in ways (e.g., education, affluence, age and health) that may have influenced their comprehension of the information as well as their response to the manipulation and their expressions of negative emotions and perceived risk. However, the fact that our study provided some replication of prior research using a different sample and methods suggests that our findings are robust. Second, participants viewed the information on a website and it remains unknown what effects a different presentation medium might have on responses. Third, we focused on negative emotions and perceived risk. It is unknown how our manipulations might affect future quit attempts. However, our focus was on the psychological consequences of receiving genetic risk feedback, not on cessation. Moreover, we suspect the risk and negative emotions may influence cessation indirectly rather than directly by prompting related cognitions such as attitudes about smoking and willingness to quit, and by instigating more subtle actions such as information seeking and discussing the results with others. Fourth, we caution against over interpreting the large effects we found for risk perceptions and worry because presenting the gene variants in a within-subjects design may have magnified differences. Finally, all participants answered the questions in the order described and it is possible that responses to the risk estimates influenced responses to the negative emotion items. It is important to note that such effects are likely bi-directional. For instance, evidence suggests affective states can strongly influence risk estimates (Loewenstein, Weber, Hsee, & Welch, 2001). This latter finding suggests that our effects may have been even stronger had we assessed negative emotions before risk estimates. Also relevant to the order of items, it is possible that response to the "have" risk

item constrained response to the “missing” risk item. That is, regardless of how participants responded to the “have” item, they may have felt compelled to respond to the “missing” item in the direction of greater risk. Yet an examination of the frequency of responses revealed that 78.1% of participants selected option of 2 (*slightly lower than average*) in response to the “have” item and that 89.1% of participants selected option of 4 (*slightly higher than average*) in response to the “missing” item. Apparently, participants were not merely responding to the “missing” item in the direction of greater risk, they were purposely selecting the response option that matched the information they received in the brochure. Importantly, all of these limitations suggest fruitful potentially avenues for future research.

Implications

Our findings have three important implications. First, people are responsive to information regarding genetic biomarkers for lung cancer risk. Our participants reported greater negative emotions and greater perceived risk when considering the consequences of having the *GSTM1* wild-type vs. the *GSTM1* null-type variant. To the extent that negative emotions and perceived risks are precursors to behavioral change as suggested by numerous health behavior models, genetic feedback that conveys higher risk should play a role in increased efforts towards healthy behavior. Second, although the presentation style of genetic risk information (e.g., absolute vs. incremental terms; foreground only vs. foreground + background) had some influence on responses, the influence pales in comparison to the substantive meaning participants attached to having the *GSTM1* wild-type vs. the *GSTM1* null-type variant. The implication is that the content of health communications may be more important than the style. Third, small differences in genetic risk magnitudes can have big psychological effects, particularly when participants attend to the bottom line gist over concrete risk information. The implication is that health professionals need not slant or exaggerate genetic effects to persuade people. Indeed, our participants were quite sensitive to the underlying gist (e.g., having one gene variant is good and having another is bad) implied by different genetic feedback outcomes. We see some cause for concern that small differences in risk produced large differences in negative emotions, particularly when the risk is not linked to behavior and the negative emotions interferes with functioning.

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References

- Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. National Center for Health Statistics. Vital Health Statistics. 2009; 10(242):68.
- Benhamou S, Lee WJ, Alexandrie AK, Boffetta P, Bouchardy C, et al. Meta- and pooled analyses of the effects of glutathione S-transferase M1 polymorphisms and smoking on lung cancer risk. *Carcinogenesis*. 2002; 23:1343–1350. [PubMed: 12151353]
- Bize R, Burnand B, Mueller Y, Rège WM, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. *Cochrane Database System Review*. 2009; 15(2):CD004705.
- Bowen DJ, Helmes A, Powers D, Andersen MR, Burke W, Mctiernan A, Durfy S. Predicting breast cancer screening intentions and behavior with emotion and cognition. *Journal of Social and Clinical Psychology*. 2003; 22:213–232.10.1521/jscp.22.2.213.22875
- Chua HF, Yates FJ, Shah P. Risk avoidance: Graphs versus numbers. *Memory & Cognition*. 2006; 34:399–410.
- Cummings KM, Becker MH, Maile MC. Bringing the models together: An empirical approach to combining variables used to explain health actions. *Journal of Behavioral Medicine*. 1980; 3:123–145. [PubMed: 7420418]

- Dijkstra A, Brosschot J. Worry about health in smoking behaviour change. *Behaviour Research & Therapy*. 2003; 41:1081–92. [PubMed: 12914809]
- Fishbein M. A theory of reasoned action: Some applications and implications. *Nebraska Symposium on Motivation*. 1980; 27:65–116. [PubMed: 7242751]
- Hansen WB, Malotte CK. Perceived personal immunity: the development of beliefs about susceptibility to the consequences of smoking. *Preventive Medicine*. 1986; 15:363–372. [PubMed: 3763561]
- Hertwig R, Barron G, Weber EU, Erev I. Decisions from experience and the effect of rare events in risky choice. *Psychological Science*. 2004; 15:534–539. [PubMed: 15270998]
- Hertwig R, Erev I. The description-experience gap in risky choice. *Trends in Cognitive Sciences*. 2009; 13:517–23. [PubMed: 19836292]
- Jarvenpaa SL. Graphic displays in decision making: The visual salience effect. *Journal of Behavioral Decision Making*. 1990; 3:247–262.
- Kahneman D, Tversky A. Prospect theory: An analysis of decision under risk. *Econometrica*. 1979; 47:263–291.
- Lipkus IM, Prokhorov AV. The effects of providing lung age and respiratory symptoms feedback on community college smokers' perceived smoking-related health risks, worries and desire to quit. *Addictive Behaviours*. 2007; 32:516–32.
- Loewenstein GF, Weber EU, Hsee CK, Welch N. Risk as feelings. *Psychological Bulletin*. 2001; 127:267–286.10.1037/0033-2909.127.2.267 [PubMed: 11316014]
- Mackinnon A, Jorm A, Christensen H, Korten A, Jacomb P, Rodgers B. A short form of the Positive and Negative Affect Schedule: Evaluation of factorial validity and invariance across demographic variables in a community sample. *Personality and Individual Differences*. 1999; 27:405–416.10.1016/S0191-8869(98)00251-7
- Marteau TM, French DP, Griffin SJ, Prevost AT, Sutton S, Watkinson C, Attwood S, Hollands GJ. Effects of communicating DNA-based disease risk estimates on risk-reducing behaviours. *The Cochrane Database of Systematic Reviews*. 2010; (10):CD007275.10.1002/14651858.CD007275.pub2 [PubMed: 20927756]
- McBride CM, Bepler G, Lipkus IM, Lyna P, Samsa G, Albright J, Datta S, Rimer BK. Incorporating genetic susceptibility feedback into a smoking cessation program for African-American smokers with low income. *Cancer Epidemiology Biomarkers Prevention*. 2002; 11(6):521–528.
- McClure JB. Are biomarkers useful treatment aids for promoting health behavior change? An empirical review. *American Journal Preventive Medicine*. 2002; 22(3):200–207.
- McClure JB. Motivating prepartum smoking cessation: A consideration of biomarker feedback. *Nicotine Tobacco Research*. 2004; 6(Suppl 2):S153–161.10.1080/14622200410001669222 [PubMed: 15203818]
- Mellers BA, Schwartz A, Ho K, Ritov I. Decision affect theory: Emotional reactions to the outcomes of risky options. *Psychological Science*. 1997; 8:423–429.
- Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. *Vital Health Statistics* 10. 2009; 242:1–157.
- Reyna VF. A theory of medical decision making and health: Fuzzy trace theory. *Medical Decision Making*. 2008; 28:850–65. [PubMed: 19015287]
- Rippetoe PA, Rogers RW. Effects of components of Protection Motivation Theory on adaptive and maladaptive coping with a health threat. *Journal of Personality and Social Psychology*. 1987; 52:596–604.10.1037/0022-3514.52.3.596 [PubMed: 3572727]
- Rosenstock IM, Strecher VJ, Becker MH. Social learning theory and the health belief model. *Health Education Quarterly*. 1988; 15:394–405.
- Sanderson SC, Humphries SE, Hubbard C, Hughes E, Jarvis MJ, Wardle J. Psychological and behavioural impact of genetic testing smokers for lung cancer risk: a phase II exploratory trial. *Journal of Health Psychology*. 2008; 13(4):481–494.10.1177/1359105308088519 [PubMed: 18420756]
- Slovic, P. Perception of risk: Reflections on the psychometric paradigm. In: Krinsky, S.; Golding, D., editors. *Social Theories of Risk*. Praeger; Westport: 1992. p. 7-51.

- Slovic, P. Cigarette smokers: Rational actors or rational fools?. In: Slovic, P., editor. *Smoking: Risk, perception, and policy*. Thousand Oaks, CA: Sage; 2001. p. 97-124.
- Slovic, P. Affect, analysis, adolescence, and risk. In: Romer, D., editor. *Reducing adolescent risk: Toward an integrated approach*. Thousand Oaks, CA: Sage; 2003. p. 44-48.
- Slovic P, Peters E, Finucane ML, MacGregor DG. Affect, risk, and decision making. *Health Psychology*. 2005; 24(Suppl):S35–S40. [PubMed: 16045417]
- Sobti RC, Kaur P, Kaur S, Janmeja AK, Jindal SK, Kishan J, Raimondi S. Combined effect of GSTM1, GSTT1 and GSTP1 polymorphisms on histological subtypes of lung cancer. *Biomarkers*. 2008; 13(3):282–95. [PubMed: 18415801]
- Spielberger, CD.; Gorsuch, R.; Lushene, R. *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologist's Press; 1970.
- Stone ER, Sieck WR, Bull BE, Yates JF, Parks SC, Rush CJ. Foreground: background salience: Explaining the effects of graphical displays on risk avoidance. *Organizational Behavior and Human Decision Processes*. 2003; 90:19–36.10.1016/S0749-5978(03)00003-7
- Vineis P, Anttila S, Benhamou S, Spinola M, Hirvonen A, et al. Evidence of gene interactions in lung carcinogenesis in a large pooled analysis. *Carcinogenesis*. 2007; 28(9):1902–1905. [PubMed: 17307802]
- Weinstein ND, Klein WM. Unrealistic optimism: Present and future. *Journal of Social and Clinical Psychology*. 1996; 15:1–8.
- Zikmund Fisher BJ, Ubel PA, Smith DM, Derry HA, McClure JB, Stark A, Pitsch RK, Fagerlin A. Communicating side effect risks in a tamoxifen prophylaxis decision aid: the debiasing influence of pictographs. *Patient Education & Counseling*. 2008; 73:209–214. [PubMed: 18602242]

Figure 1.
Graphic display of lung cancer risk

Table 1

Sample Characteristics (N=128)

Characteristics	Percent
Age (mean)	19.9 (<i>SD</i> = 1.0)
Sex (male)	50.8
Race	
White	71.9
African	4.7
Hispanic	18.7
Asian	3.9
Native American	.8
Class Rank	
Freshman	14.8
Sophomore	18.0
Junior	35.2
Senior	31.8
Graduate School	.8
Years smoked (mean)	2.6 (<i>SD</i> = 1.6)

Table 2

Mean Lung Cancer Risk Estimates

	No Display	Foreground Only	Foreground + Background
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Risk of Lung Cancer with GSTM1 Wild-type Variant	2.2 ^{ab} (.45)	2.0 ^a (.34)	2.4 ^b (.66)
Risk of Lung Cancer with GSTM1 Null-type Variant	4.0 ^c (.30)	3.9 ^c (.63)	4.0 ^c (.41)

Note: Means with different superscripts differ at $p < .05$.

Table 3

Mean Levels of Worry

	No Display	Foreground Only	Foreground + Background
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Worry if GSTM1 Wild-type Variant			
Absolute Condition	2.0 ^b (1.04)	2.1 ^b (1.08)	2.3 ^b (1.27)
Incremental Condition	2.1 ^b (1.06)	1.9 ^b (1.09)	1.5 ^b (.77)
Worry if GSTM1 Null-type Variant			
Absolute Condition	4.8 ^a (1.55)	4.7 ^a (1.91)	4.8 ^a (1.89)
Incremental Condition	4.3 ^a (1.79)	3.9 ^a (1.57)	4.3 ^a (1.63)