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Asymmetrical genetic attributions for the presence and absence of health problems

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ABSTRACT

Objective: Recent research has suggested that people more readily make genetic attributions for positively valenced or desirable traits than for negatively valenced or undesirable traits—an asymmetry that may be mediated by perceptions that positive characteristics are more 'natural' than negative ones. This research sought to examine whether a similar asymmetry in genetic attributions would emerge between positive and negative health outcomes.

Design: Across seven experiments, participants were randomly assigned to read a short vignette describing an individual experiencing a health problem (e.g. hypertension) or a corresponding healthy state (e.g. normal blood pressure).

Main Outcome Measures: All participants provided ratings of naturalness and genetic attributions for the outcome described in their assigned vignette.

Results: For diagnoses other than addictive disorders, participants rated the presence of a diagnosis as less genetically caused than its absence; for addictive disorders, the presence of a diagnosis was rated as more genetically caused than its absence. Participants consistently rated the presence of a health problem as less natural than its absence.

Conclusion: Even within a single domain of health, people ascribe differing degrees of 'naturalness' and genetic causation to positive versus negative health outcomes, which could impact their preferences for treatment and prevention strategies.

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Introduction

Recent years have seen major advances in genomic science, leading to an increased understanding of the role of genes in a wide variety of physical and mental disorders and promising to revolutionize healthcare in the near future through the application of genomics in clinical practice (Auffray et al., 2019). Indeed, genetic advances are expected to play an important role in informing the development of precision approaches to healthcare across a wide range of disease contexts (Zeggini et al., 2019).

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These advances in human genetics and the accompanying embrace of precision medicine are likely to entail increased exposure of members of the public to information about the genetic bases of health outcomes. This could have important real-world implications, as causal explanations for health problems can affect important variables such as individuals' preferences for treatment and risk-reduction strategies, prognostic expectations, and stigmatizing attitudes toward affected individuals, as well as clinicians' treatment recommendations and interpersonal reactions to patients (Ahn et al., 2009; Kvaale et al., 2013; Lebowitz & Ahn, 2014; Lebowitz & Appelbaum, 2017; Senior & Marteau, 2007; Wright et al., 2003).

Notably, there may be reason to expect that people's beliefs about the role of genes in causing different health outcomes may depend on more than just the latest scientific findings. Genetic explanations for human characteristics are often viewed through an essentialist lens, with genes seen as containing the fundamental and immutable essence of a person or category of persons (Dar-Nimrod & Heine, 2011; Prentice & Miller, 2007). As such, genetic explanations for health outcomes may be associated with deterministic or fatalistic perspectives that view genetic liability to a particular health outcome as indicating that the outcome in question is inevitable or immutable (Heine et al., 2017; Lebowitz & Appelbaum, 2019, but c.f. Collins et al., 2011). By the same token, attributes that are particularly likely to be essentialized—that is, seen as stemming from a fundamental essence that is shared by all who exhibit them—may be especially readily attributed to genetic causes. Prior work has suggested that positive or attractive characteristics, such as being a good person or having desirable personality traits, are more essentialized than negative or unattractive ones, such as being a morally bad person or having undesirable personality traits (Haslam et al., 2004; Heiphetz, 2019). While these studies used measures that included biological etiology as one of multiple components of essentialism, our own work suggests that such an asymmetry can emerge regarding perceptions of genetic causality specifically. In particular, we have previously shown that positively valenced phenotypes, such as prosocial behavior and physical attractiveness, are more readily attributed to genetic causes than are their negatively valenced counterparts (Lebowitz et al., 2019, 2021). It appears that this asymmetry may be driven, at least in part, by a perception that positively valenced phenotypes are more 'natural' than negatively valenced phenotypes (Lebowitz et al., 2021).

What does it mean for people to rate positively valenced phenotypes as more 'natural'? To clarify this, in previous research (Lebowitz et al., 2021, Experiment 3) we asked participants to rate the naturalness of a variety of positive and negative phenotypes (using the same measure as in the present research; see below) and then prompted them to write a free-text response describing what meaning of 'natural' they had in mind when making their rating. A thematic analysis of these responses found that the most common meaning of 'natural' invoked by participants reflected the notion that a trait was not attributable to any special effort, thought, work, or intention, but instead was manifested automatically, effortlessly, or instinctually. Importantly, no participants defined natural as akin to 'good' or 'praiseworthy', indicating that naturalness ratings were not simply capturing positive valence itself. Rather, naturalness may function as a mediator of asymmetries in genetic attributions at least in part because people view negatively valenced human phenotypes as

deviations from a default 'natural' state, and are thus reluctant to ascribe them to genetic causes.

At present, it is unclear whether asymmetries like those observed in non-health domains, in which positively valenced phenotypes are more readily attributed to genetic causes than negatively valenced phenotypes, would be observed in the context of health outcomes—that is, whether good health would be more readily attributed to genes than ill health. Moreover, it is not known whether such an asymmetry, if it occurs, would also be mediated by asymmetric ascriptions of naturalness. If there is an asymmetry in genetic attributions for good versus bad health, reluctance to ascribe negative health outcomes to genetic causes could affect important real-world considerations, such as what treatments and risk-reduction strategies are seen as appropriate. This could therefore have clinical importance, especially if the asymmetries are particularly pronounced in some areas of health compared to others (e.g. mental versus physical disorders, or health outcomes that are perceived as less versus more determined by volitional behavior).

In the present research, we measured genetic attributions for a variety of health outcomes, including both mental disorders (e.g. depression) and physical disorders (e.g. hypertension), as well as disorders whose origins are often perceived as attributable to volitional factors (e.g. obesity, alcohol use disorder) and those that are generally not perceived this way (e.g. osteoporosis). For each disorder, we compared genetic attributions for the presence of the disorder to genetic attributions for the absence of the disorder (i.e. the corresponding positive health outcome), as we sought to examine whether there would be an asymmetry in genetic attributions for negative health outcomes versus positive ones. We also examined ascriptions of naturalness for each outcome, as a potential mediator of any differences in genetic attributions.

Experiments 1a-1e

Methods

Participants and recruitment

We calculated that a two-group comparison (using independent-samples *t*-tests) would require 394 participants per condition to reach 80% power to detect a small effect (d=.2). Rounding up this number to 400 participants per condition, we sought to recruit approximately 800 participants per experiment. Participants were U.S. adults recruited using the Prolific online platform (Palan & Schitter, 2018), which allows researchers to quickly recruit and compensate participants for online studies. Prolific maintains a relatively diverse participant pool of tens of thousands of individuals, has been shown to yield data quality that is comparable to or higher than what is attained through other popular online participant-recruitment services, and has been used to recruit participants for hundreds of published studies (Peer et al., 2017).

The sample for Experiment 1a (which focused on depression) consisted of 798 adults and was 45.4% male, 53.0% female, and 1.6% other or unknown gender, with a mean age of 35.15 years (SD = 12.77). The sample for Experiment 1 b (obesity) consisted of 802 adults and was 49.9% male, 48.8% female, and 1.3% other or unknown gender, with a mean age of 34.46 years (SD = 12.62). The sample for Experiment 1c

(hypertension) consisted of 800 adults and was 45.25% male, 53.75% female, and 1.0% other or unknown gender, with a mean age of 34.12 years ($SD = 12.22$). The sample for Experiment 1d (osteoporosis) consisted of 801 adults and was 48.4% male, 50.3% female, and 1.2% other or unknown gender, with a mean age of 33.66 years ($SD = 12.29$). The sample for Experiment 1e (alcohol use disorder) consisted of 801 adults and was 42.2% male, 56.7% female, and 1.1% other or unknown gender, with a mean age of 35.89 years ($SD = 12.34$).

Procedures

Procedures were approved by the Institutional Review Board at the New York State Psychiatric Institute (protocol 7369). Experiments 1a-1e followed identical procedures, except that they each targeted a different mental or physical disorder. After providing informed consent, participants were randomly assigned to a Diagnosis Present condition or a Diagnosis Absent condition. Then, in each experiment, participants read a short vignette describing an individual named Jane. The vignette always began with the following stem: 'Jane is an adult woman living in the United States. According to her doctor, she meets medical standards for…'; the final word(s) of the vignette constituted the 'descriptor' (e.g. 'depression' or 'a normal mood') that was experimentally varied according to the condition to which a participant was randomly assigned in each experiment. In the Diagnosis Present condition, the descriptor referred to the disorder in question (e.g. 'obesity'), whereas in the Diagnosis Absent condition, the descriptor referred to a 'normal' instantiation of the trait that was implicated in the Diagnosis Present condition (e.g. 'a normal weight'). The word 'normal' was always used in the Diagnosis Absent descriptor to maximize consistency across the different disorders. The descriptors for all experiments are listed in Table 1.

After reading the vignette, participants were asked to provide two ratings. As a measure of perceived naturalness, they were asked, 'How natural do you think it is for Jane to have [descriptor]?'; responses were provided on a scale from '1 (Not at all natural)' to '7 (Very natural)'. As a measure of genetic attributions, they were asked, 'How much of a role do you think genetics play in causing Jane to have [descriptor]?'; responses were provided on a scale from '1 (No role or a very minor role)' to '7 (A very major role) '. At the end of the study procedures, participants were asked basic demographic questions and were debriefed as to the fictitious nature of the vignettes.

Data analysis

For each experiment, we used independent-samples *t*-tests to compare the mean naturalness ratings and mean genetic attribution ratings of participants in the Diagnosis Absent condition to those of participants in the Diagnosis Present condition. To

Table 1. Vignette contents for Experiments 1a-1e. AUD = alcohol use disorder.

Experiment	Target Condition	Diagnosis Present descriptor	Diagnosis Absent descriptor
1a	depression	depression	a normal mood
1b	obesity	obesity	a normal weight
1c	hypertension	hypertension (high blood pressure)	normal blood pressure
1d	osteoporosis	osteoporosis (loss of bone density)	normal bone density
1e	AUD	alcohol use disorder (alcoholism)	normal alcohol use

examine whether the pattern of results was consistent with the possibility that any observed differences in genetic attributions were mediated by differences in naturalness ratings, we used the PROCESS procedure (version 3.4) for SPSS to examine the indirect effect of condition, through naturalness ratings, on genetic attributions (Hayes, 2018). This produces estimates of the 'direct effect' of condition on genetic attributions and of the 'indirect effect' of condition on genetic attributions through naturalness ratings. The direct effect represents a regression-based estimate of the extent to which two participants would be expected to differ in genetic attributions if one were assigned to the Diagnosis Absent and the other were assigned to the Diagnosis Present condition but their naturalness ratings were equivalent. The indirect effect estimate is the multiplicative product of two regression coefficients: one estimating the extent to which two participants would be expected to differ in genetic attributions by virtue of being assigned to different conditions, and one estimating the extent to which two participants with equivalent naturalness ratings would be expected to differ in genetic attributions if assigned to the same condition. This represents the extent to which two participants in different conditions would be expected to differ in genetic attributions as a result of the association between condition and naturalness ratings. Put another way, the indirect effect corresponds to the magnitude of the association between condition and genetic attributions without controlling for naturalness ratings, minus the magnitude of the association between condition and genetic attributions when controlling for naturalness ratings. That is, the indirect effect is an indicator of how much of the relationship between condition and genetic attributions may actually be a result of the relationship between condition and naturalness ratings.

To infer whether each observed indirect effect in the present research was significant, we generated 95% bootstrap confidence intervals around each indirect effect estimate and concluded that the indirect effect was significant if these intervals did not include zero. To calculate these confidence intervals, the software randomly generates a large number (in this case 5,000) of 'bootstrap samples' composed of subsets of the full sample and estimates the magnitude of the indirect effect within each bootstrap sample, sorting the estimates derived from smallest to largest in magnitude. In the resulting distribution, the value corresponding to the 2.5^{th} percentile is taken as the lower bound of a 95% confidence interval for the indirect effect observed in the full sample, and the value corresponding to the 97.5^{th} percentile is taken as the upper bound, as 95% of bootstrap samples yielded indirect effect estimates within these boundaries.

Results

Specific analyses for each experiment (1a-1e) are reported below. To summarize, in Experiments 1a-1d, genetic attribution ratings were significantly higher in the Diagnosis Absent condition compared to the Diagnosis Present condition, but in Experiment 1e (alcohol use disorder) this pattern was reversed (see Table 2). Across Experiments 1a-1e, naturalness ratings were significantly higher in the Diagnosis Absent condition compared to the Diagnosis Present condition (Table 2). Bootstrap mediation analyses using the PROCESS procedure (version 3.4) for SPSS with 5,000 bootstrap samples (Hayes, 2018),

Table 2. Mean naturalness and genetic attribution ratings, with standard deviations, for the presence and absence of a diagnosis in experiments 1a-2b.

	Condition	Mean (SD) Naturalness Rating	Mean (SD) Genetic Attribution Rating
Experiment 1a (depression)	Diagnosis Absent	5.94 (1.19)	4.85 (1.31)
	Diagnosis Present	4.94 (1.51)	4.39 (1.31)
Experiment 1 b (obesity)	Diagnosis Absent	5.67 (1.36)	4.73 (1.36)
	Diagnosis Present	3.60 (1.50)	3.99 (1.37)
Experiment 1c (hypertension)	Diagnosis Absent	6.04 (1.09)	4.98 (1.19)
	Diagnosis Present	4.24 (1.37)	4.81 (1.17)
Experiment 1d (osteoporosis)	Diagnosis Absent	6.23 (0.99)	5.33 (1.11)
	Diagnosis Present	4.68 (1.34)	5.07 (1.13)
Experiment 1e (alcohol use disorder)	Diagnosis Absent	5.61 (1.37)	4.12 (1.54)
	Diagnosis Present	3.59 (1.59)	4.37 (1.45)
Experiment 2a (nicotine use disorder)	Diagnosis Absent	6.28 (1.31)	2.82 (1.51)
	Diagnosis Present	3.86 (1.87)	3.27 (1.55)
Experiment 2 b (gambling disorder)	Diagnosis Absent	5.52 (1.62)	3.28 (1.56)
	Diagnosis Present	3.74 (1.47)	3.79 (1.64)

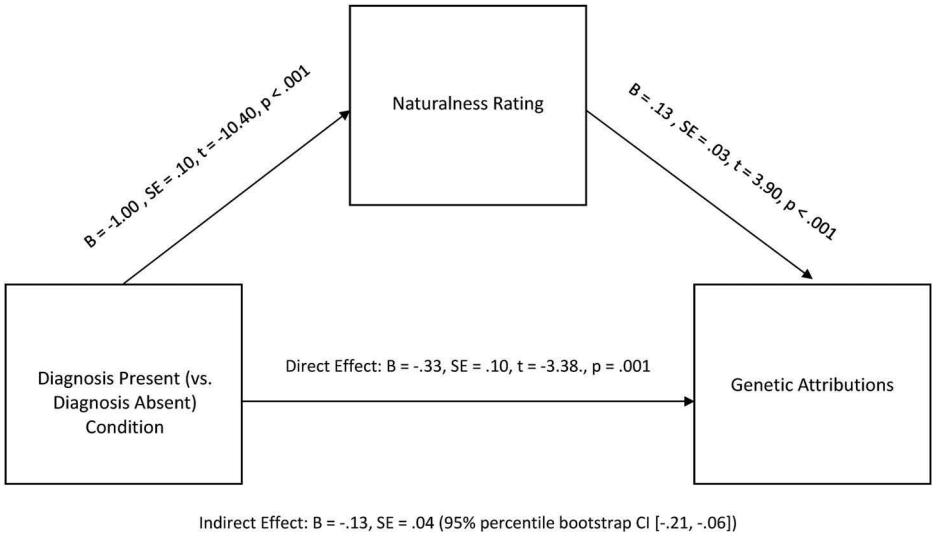

Figure 1. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 1a (in which the diagnosis in question was depression).

in which the Diagnosis Present condition was coded as 1 and the Diagnosis Absent condition was coded as 0, showed that across Experiments 1a-1d, there was a significant indirect effect consistent with the interpretation that asymmetries in perceived naturalness helped to account for the observed asymmetries in genetic attributions (see Figures 1–4). A similar indirect effect was observed in Experiment 1e despite the direct effect and the total effect operating in opposite direction (Figure 5).

Experiment 1a

Participants who read about Jane having a 'normal mood' endorsed genetic attributions significantly more strongly ($M = 4.85$, $SD = 1.31$) than those who read about her

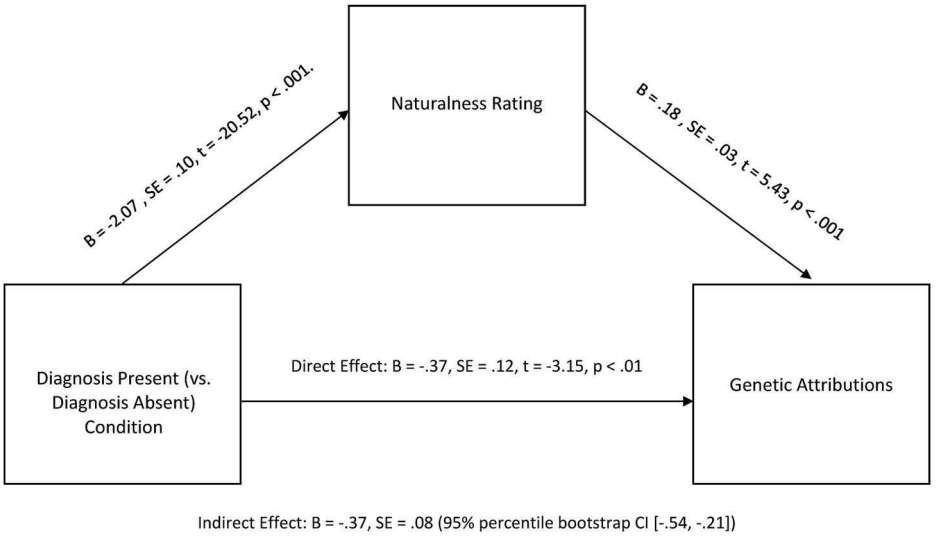

Figure 2. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 1b (in which the diagnosis in question was obesity).

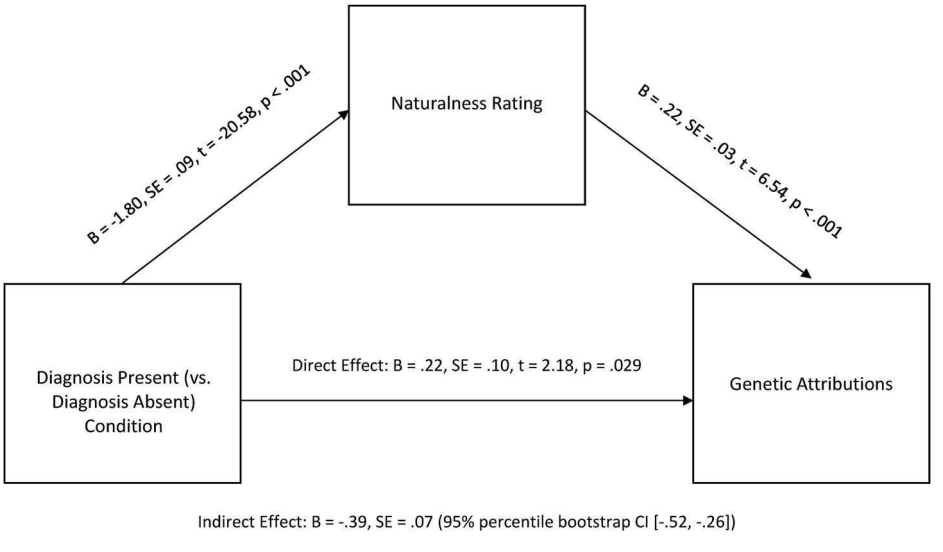

Figure 3. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 1c (in which the diagnosis in question was hypertension).

having depression ($M = 4.39$, $SD = 1.31$), $t(796) = 4.99$, $p < .001$, $d = .35$. Jane's 'normal mood' also evoked significantly higher naturalness ratings ($M = 5.94$, $SD = 1.19$) than her depression ($M = 4.94$, $SD = 1.51$), $t(796) = 10.40$, $p < .001$, $d = .74$. The indirect effect (see Figure 1) of condition on genetic attributions through naturalness ratings was significant ($B = -.13$, SE=.04, 95% percentile bootstrap CI [-.21, −.06]).

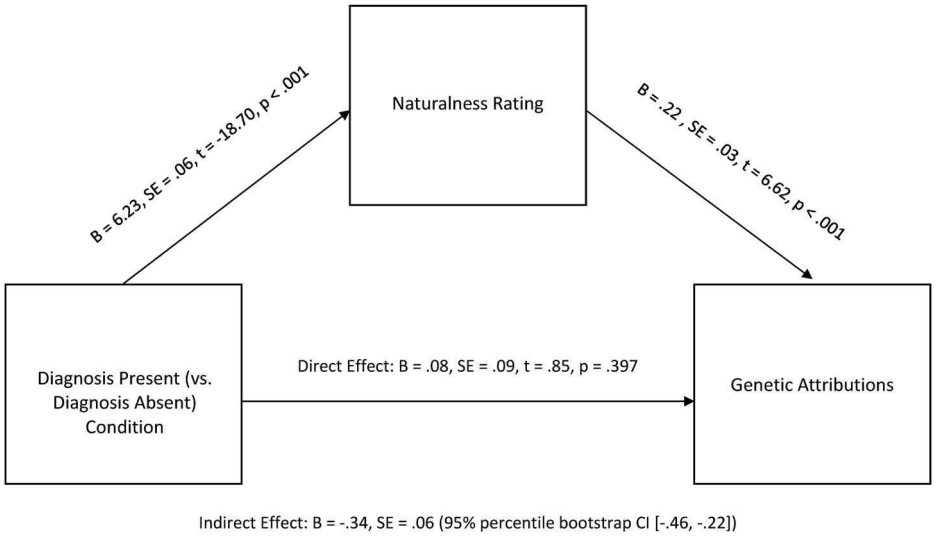

Figure 4. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 1d (in which the diagnosis in question was osteoporosis).

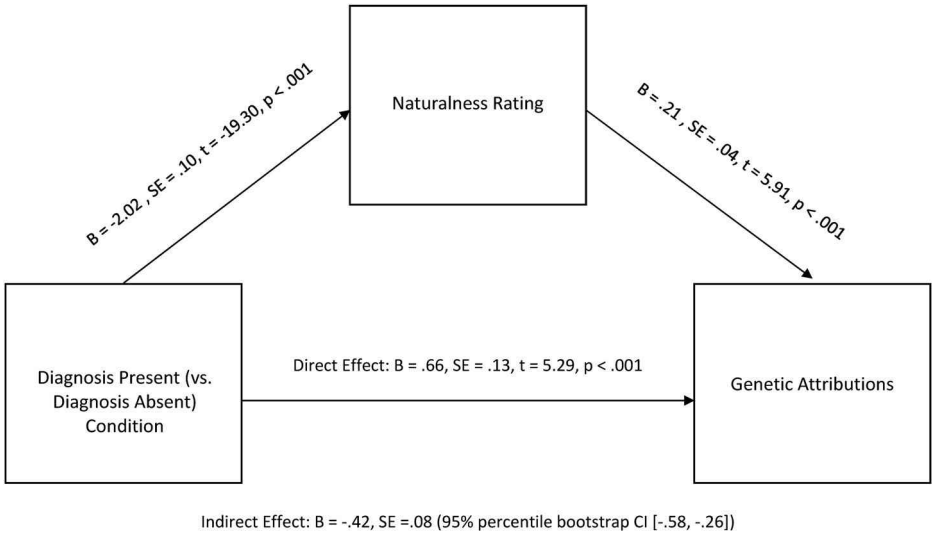

Figure 5. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 1e (in which the diagnosis in question was alcohol use disorder).

Experiment 1b

Participants who read about Jane having a 'normal weight' had significantly higher genetic attribution ratings ($M = 4.73$, $SD = 1.36$) than those who read about her having obesity ($M = 3.99$, $SD = 1.37$), $t(799) = 7.69$, $p < .001$, $d = .54$. Those who read about Jane having a 'normal weight' also gave stronger naturalness ratings ($M = 5.67$, $SD = 1.36$)

than those who read about Jane having obesity ($M = 3.60$, $SD = 1.50$), $t(799)=20.52$, $p<.001$, $d=.1.45$. The indirect effect (see Figure 2) of condition on genetic attributions through naturalness ratings was significant ($B=-.37$, $SE=.08$, 95% percentile bootstrap CI [-0.54, −.21]).

Experiment 1c

Jane's 'normal blood pressure' was attributed to genetic causes significantly more strongly ($M = 4.98$, $SD = 1.19$) than her hypertension ($M = 4.81$, $SD = 1.17$), $t(798)=2.01$, $p=.044$, $d=.14$. Participants who read about Jane having 'normal blood pressure' also had significantly higher naturalness ratings ($M = 6.04$, $SD = 1.09$) than those who read about Jane having hypertension ($M = 4.24$, $SD = 1.37$), $t(798)=20.59$, $p<.001$, $d=.1.46$. The indirect effect (see Figure 3) of condition on genetic attributions through naturalness ratings was significant ($B=-.39$, $SE=.07$, 95% percentile bootstrap CI [-.52, −.26]).

Experiment 1d

Jane's 'normal bone density' was attributed to genetic causes significantly more strongly ($M = 5.33$, $SD = 1.11$) than her osteoporosis ($M = 5.07$, $SD = 1.13$), $t(799)=3.27$, $p=.001$, $d=.23$. Participants who read about Jane having 'normal bone density' also had significantly higher naturalness ratings ($M = 6.23$, $SD=.99$) than those who read about Jane having osteoporosis ($M = 4.68$, $SD = 1.34$), $t(799)=18.70$, $p<.001$, $d = 1.32$. The indirect effect (see Figure 4) of condition on genetic attributions through naturalness ratings was significant ($B=-.34$, $SE=.06$, 95% percentile bootstrap CI [-.46, −.22]).

Experiment 1e

Participants who read about Jane having 'normal alcohol use' had significantly higher naturalness ratings ($M = 5.61$, $SD = 1.37$) than those who read about Jane having alcohol use disorder ($M = 3.59$, $SD = 1.59$), $t(799)=19.30$, $p<.001$, $d = 1.37$. However, Jane's 'normal alcohol use' was attributed to genetic causes significantly less strongly ($M = 4.12$, $SD = 1.54$) than her alcohol use disorder ($M = 4.37$, $SD = 1.45$), $t(799)=-2.32$, $p=.021$, $d=.16$. The indirect effect (see Figure 5) of condition on genetic attributions through naturalness ratings was nonetheless significant and negative ($B=-.42$, $SE=.08$, 95% percentile bootstrap CI [-.58, −.26]), indicating that although the overall effect of Jane having (vs. not having) alcohol use disorder was to yield higher genetic attribution ratings, its indirect effect through naturalness perceptions operated in the opposite direction.

Experiments 2a-2b

Across experiments 1a-1e, encompassing five different health domains, positive health states characterized as normal were rated as significantly more 'natural' than were negative health states characterized by the presence of a diagnosis. For depression, obesity, hypertension, and osteoporosis—a diverse set of diagnoses including a mental disorder, an addictive disorder, as well as physical disease states varying in the extent to which their perceived etiologies typically involve volitional behavior—genetic

attributions were significantly higher when the diagnosis was absent than when it was present. However, for alcohol use disorder—the only addiction examined—the opposite pattern was observed: genetic attributions were higher when the diagnosis was present than when it was absent, even though the presence (versus absence) of the diagnosis yielded lower naturalness ratings, which were positively associated with genetic attributions. This finding raises the possibility that the role of genetics in health outcomes may be perceived differently for addictive behaviors than for other mental and physical disorders. We therefore conducted two further experiments (2a and 2b) examining whether the same pattern of effects would be observed when we systematically varied the presence (versus absence) of other addictive disorders.

Methods

Participants and recruitment

As in experiments 1a-1e, we sought to recruit approximately 800 participants per experiment. Participants were again U.S. adults recruited using the Prolific online platform. The disorders chosen were nicotine use disorder and gambling disorder, to enable exploration of whether the pattern of results seen with alcohol use disorder would also emerge for another substance use disorder and for a behavioral addiction.

The sample for Experiment 2a (which focused on nicotine use disorder) consisted of 801 adults and was 43.1% male, 54.7% female, and 2.2% other or unknown gender, with a mean age of 34.17 years ($SD = 12.86$). The sample for Experiment 2b (gambling disorder) consisted of 800 adults and was 47.3% male, 51.9% female, and 0.9% other or unknown gender, with a mean age of 32.65 years ($SD = 11.94$).

Procedures

As in experiments 1a-1e, participants were randomly assigned to a Diagnosis Present condition or a Diagnosis Absent condition and read a short vignette describing an individual named Jane that was experimentally varied according to the condition to which the participant had been assigned. We reasoned that laypeople might be somewhat unfamiliar with the terms 'nicotine use disorder' and 'gambling disorder', so we included slightly more detailed definitions in experiments 2a and 2b. In experiment 2a, the vignette began, 'Jane is an adult woman living in the United States. When she had her annual medical checkup, her doctor noted that she…'; the possible endings were '…meets medical standards for nicotine use disorder (that is, she is addicted to smoking cigarettes)' (Diagnosis Present) or '…does not smoke cigarettes' (Diagnosis Absent). In experiment 2b, the vignette began, 'Jane is an adult woman living in the United States. As part of a recent medical assessment, her doctor asked her about a variety of her behaviors, including gambling, and determined that she …'; the possible endings were '…meets medical standards for gambling disorder (that is, an addiction to gambling)' (Diagnosis Present) or '…does not gamble' (Diagnosis Absent).

After reading the vignette, participants were asked to provide a naturalness rating ('How natural do you think it is for Jane…') and a genetic attribution rating ('How much of a role do you think genetics play in causing Jane…') using the same response

scales as in experiments 1a-1e. In experiment 2a, the item prompts ended with either '…to have nicotine use disorder (that is, to be addicted to smoking cigarettes)' (Diagnosis Present) or '…to not smoke cigarettes' (Diagnosis Absent). In experiment 2b, the item prompts ended with either '…to have gambling disorder (that is, an addiction to gambling)' (Diagnosis Present) or '…to not gamble' (Diagnosis Absent).

At the end of the study procedures, participants were asked basic demographic questions and were debriefed as to the fictitious nature of the vignettes.

Results

Specific analyses for each experiment (2a and 2b) are reported below. To summarize, as in Experiment 1e, genetic attribution ratings were significantly higher in the Diagnosis Present condition compared to the Diagnosis Absent condition across both experiments, and naturalness ratings were significantly higher in the Diagnosis Absent condition compared to the Diagnosis Present condition (see Table 2). Bootstrap mediation analyses revealed similar patterns of results to the one observed in experiment 1e (see Figures 6 and 7).

Experiment 2a

When participants read that Jane was a nonsmoker, this was attributed to genetic causes significantly less strongly ($M = 2.82$, $SD = 1.51$) than when she was described as having nicotine use disorder ($M = 3.27$, $SD = 1.55$), $t(799) = -4.12$, $p < .001$, $d = .29$. Participants who read that Jane 'does not smoke cigarettes' had significantly higher naturalness ratings ($M = 6.28$, $SD = 1.31$) than those who read about Jane having nicotine use disorder ($M = 3.86$, $SD = 1.87$), $t(799) = 21.24$, $p < .001$, $d = 1.50$. The indirect

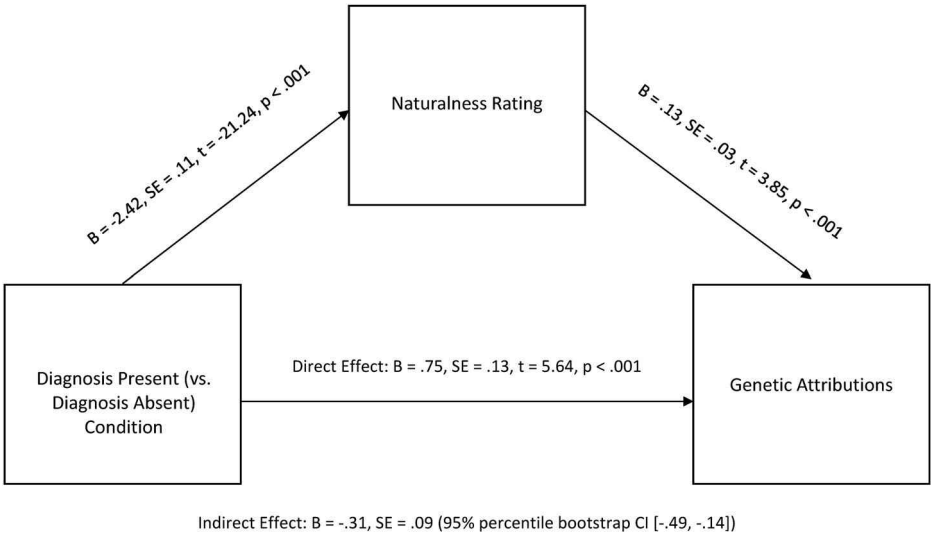

Figure 6. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 2a (in which the diagnosis in question was nicotine use disorder).

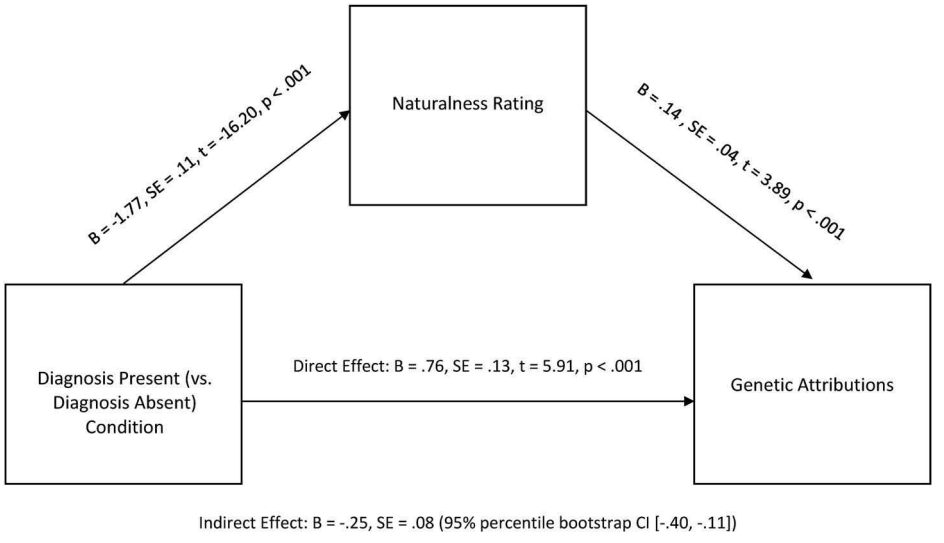

Figure 7. Analysis of naturalness ratings as a mediator of the effect of the presence (versus absence) of a diagnosis on genetic attribution ratings in Experiment 2b (in which the diagnosis in question was gambling disorder).

effect (see Figure 6) of condition on genetic attributions through naturalness ratings was significant and negative (B=-.31, SE=.09, 95% percentile bootstrap CI [-.49, −.14]), as in experiment 1e, indicating that although the overall effect of Jane having (vs. not having) nicotine use disorder was to yield higher genetic attribution ratings, its indirect effect through naturalness perceptions operated in the opposite direction.

Experiment 2b

Participants for whom Jane was described as not gambling gave significantly weaker genetic attribution ratings (M = 3.28, SD = 1.56) than those for whom she was described as having gambling disorder (M = 3.79, SD = 1.64), t(798)=-4.54, p<.001, d=.32. Participants who read that Jane 'does not gamble' had significantly higher naturalness ratings (M = 5.52, SD = 1.62) than those who read about Jane having gambling disorder (M = 3.74, SD = 1.47), t(798)=16.20, p<.001, d = 1.15. The indirect effect (see Figure 7) of condition on genetic attributions through naturalness ratings was significant and negative (B=-.25, SE=.08, 95% percentile bootstrap CI [-.40, −.11]), as in experiments 1e and 2a, again indicating that although the overall effect of Jane having (vs. not having) gambling disorder was to yield higher genetic attribution ratings, its indirect effect through naturalness perceptions operated in the opposite direction.

Discussion

The present research examined genetic causal attributions for the presence (versus absence) of a variety of physical and mental diagnoses. Previous research (Lebowitz et al., 2019, 2021) found that across a variety of physical and behavioral traits, people tend to make stronger genetic attributions for positively valenced phenotypes (e.g.

prosocial behavior, physical attractiveness, being highly organized) than for negatively valenced phenotypes (antisocial behavior, physical unattractiveness, being highly disorganized). According to recent research (Lebowitz et al., 2021), this asymmetry appears to be strongly mediated by the perception that positively valenced phenotypes are more 'natural' outcomes, often defined as typical or normal characteristics that are inborn and/or do not arise from special effort or external influence, than their negatively valenced counterparts. The present research sought to expand these findings to determine whether the same kinds of asymmetries would be present in the health context—that is, whether the presence of a disorder or diagnosis would be rated as less natural and less genetically caused than its absence. An existing body of research has examined the psychological and behavioral effects of genetic information and genetic explanations for health outcomes (e.g. Collins et al., 2011; Hollands et al., 2016; Roberts, 2019). However, research examining factors that affect willingness to attribute health outcomes to genetic causes in the first place is lacking, and the present work constitutes a contribution toward addressing this gap.

The current findings revealed that an asymmetry between ratings of positive and negative health outcomes was consistently observed for naturalness: across the seven diagnoses examined, naturalness ratings were significantly lower when the diagnosis was present than when it was absent. However, the pattern for genetic attributions was not similarly universal. Instead, while the presence of a diagnosis evoked lower genetic attributions than the absence of one in the context of depression, obesity, hypertension, and osteoporosis, the opposite pattern was observed for three addictive disorders (alcohol use disorder, nicotine use disorder, and gambling disorder). For those disorders, genetic attributions were higher when the disorder was present than when it was absent. Mediation analyses revealed that for all disorders, the presence (versus absence) of a disorder had an indirect effect, through naturalness ratings, of reducing genetic attributions, even in cases (i.e. for the addictive disorders) where the overall effect was for genetic attributions to be higher when a diagnosis was present. This suggests that for addictive disorders, there may be other mediators that counter the effect of asymmetric naturalness perceptions by affecting genetic attributions more strongly in the opposite direction. While the present data do not shed light on what such mediators might be, an inspection of the numerical means from the present experiments (i.e. those included in Table 2) suggests that in the Diagnosis Absent condition, genetic attributions appeared to be lowest in experiments 1e, 2a, and 2b. That is, for as-yet-unknown reasons, genetic attributions may be especially weak for *not* having an addictive disorder (in comparison to the absence of other disorders), and this may help to explain why the absence of an addictive disorder consistently yielded lower genetic attributions than its presence, despite naturalness ratings being higher when such a diagnosis was absent. Future research could explore whether genetic attributions for having normal drinking behavior, being a nonsmoker, and being a non-gambler might indeed be lower than genetic attributions for other positively perceived health outcomes (e.g. having a normal mood, weight, blood pressure, and bone density), and if so, why this might be.

One possibility is that because the absence of an addiction is characterized by the absence of a *behavior* (e.g. not gambling, smoking, or drinking excessively), participants viewed these outcomes as less 'biological' than other positive health states

(e.g. having a normal mood or normal blood pressure). However, it is notable that the 'Diagnosis Absent' condition in the experiments examining addictive disorders did not universally contain merely descriptions of an absence of behavior. While the 'Diagnosis Absent' conditions in experiments 2a and 2b described, respectively, an individual who 'does not smoke cigarettes' and one who 'does not gamble', the 'Diagnosis Absent' condition in experiment 1e described an individual whose behavior 'meets medical standards for normal alcohol use'. That is, two of the three addiction-related experiments referred, in the Diagnosis Absent condition, to an individual who does not engage in a particular behavior, and one of the three instead referred to an individual who engages in the 'normal' version of the pathological behavior described in the 'Diagnosis Present' condition. This suggests that reluctance to attribute an absence of behavior to genetic causes does not likely account fully for the lower genetic attribution ratings in the 'Diagnosis Absent' conditions for the experiments dealing with addictive disorders. We speculate that the relevant distinction may instead be that all phenotypes across the spectrum from normal to pathological alcohol use, smoking, and gambling are defined by behavior, rather than by a physiological state like blood pressure or body mass or by a mental state like mood. This may have led to a general reluctance to make strong genetic attributions among participants in experiments 1e, 2a, and 2b, and perhaps this reluctance was partially counteracted by the presence of a diagnostic label in the Diagnosis Present conditions of these experiments. This would be consistent with prior research showing that applying a diagnostic label to behaviors can increase the extent to which they are attributed to biomedical causes (Scior et al., 2013), potentially helping to explain the higher genetic attribution ratings in the Diagnosis Present conditions for experiments 1e, 2a, and 2b. This speculative account—that diagnostic labels may increase genetic attributions more for addictions and other behaviors than for physiological and emotional states—awaits future empirical testing.

Another possibility is that people may be aware that people with addictive disorders, even more than people with other mental disorders, tend to be highly stigmatized and blamed for their addictions (Schomerus et al., 2011) and that genetic attributions, by ascribing causality to genes rather than factors under individual control, can serve to deflect blame and stigma (Lebowitz & Appelbaum, 2017; Schomerus et al., 2014). Thus, they may be especially inclined to endorse genetic explanations for addictive disorders as a socially desirable means of appearing to disavow blame-laden, stigmatizing views of addiction. However, people with obesity are also commonly blamed for their own weight status (Lusk & Ellison, 2013; Oliver & Lee, 2005), and we did not observe higher genetic attributions for the presence (vs. absence) of obesity. Thus, if the higher genetic attributions in the 'Diagnosis Present' conditions for addictive disorders were a result of participants wishing to disavow blame-based conceptions of addiction, it would appear that they had no corresponding motivation to deflect blame from individuals with obesity. Future studies could further investigate the role of blame (or a motivation to deflect blame) as a potential mediator of genetic attributions for health outcomes, including potentially by examining how differences in genetic attributions for different health-related phenotypes (e.g. obesity vs. addiction) might be related to differences in ascription of blame or other stigmatizing attitudes.

Regardless of the reason, the fact that genetic attributions and naturalness ratings are pushed in opposite directions by descriptions of the presence (vs. absence) of an addictive disorder provides evidence that naturalness perceptions alone do not account for the observed differences in genetic attributions. The notion that other mediators besides perceptions of naturalness might play a role in determining differential genetic attributions is also supported by the effect sizes observed in the present research. Across all seven experiments reported here, the observed naturalness asymmetry, in which the absence of a disorder was consistently rated as more natural than its presence, was characterized by large effect sizes, with all but one equivalent to a difference greater than one standard deviation. By contrast, the observed differences in genetic attributions, while statistically significant given the relatively large sample sizes used in the present research, were generally small to medium in magnitude. That is, whether the differences in genetic attributions were in the same or opposite direction as the asymmetries in naturalness ratings, they were generally much smaller, buttressing the suggestion that other factors besides perceptions of naturalness must be influencing genetic attributions for health outcomes.

The present research is not without limitations. The stimuli used to manipulate the presence/absence of mental and physical diagnoses in the present research were hypothetical vignettes that contained minimal information. While this approach was useful for standardizing our experimental manipulations to isolate the effects of our independent variable, it is unclear to what extent the same patterns of naturalness perceptions and genetic attributions would emerge in response to real people with and without various health problems. Future research could examine these variables in more realistic contexts, such as by examining healthcare providers' reactions to real patients.

Additionally, most of the experiments included the word 'normal' in the stimuli for the Diagnosis Absent condition, and there may be overlap in laypeople's concepts of 'normal' and 'natural'. However, it is notable that in past work, when participants were asked to explain what meaning of 'natural' they had in mind when completing the same naturalness rating used in the present research, less than a quarter 'referred to something being probable, likely, typical, usual, ordinary, normal, common, average, or the like' (Lebowitz et al., 2021); when excluding those who also invoked another definition, the proportion was less than one in five. Furthermore, experiments 2a and 2b, which did not include the word 'normal' in any of the stimuli, yielded the same asymmetry in naturalness ratings that was observed in the other experiments, suggesting that this asymmetry was not merely due to the inclusion of the word 'normal'. In addition, the stimuli in experiment 1e did include the word 'normal' in the Diagnosis Absent condition ('normal alcohol use'), so the presence vs. absence of the word 'normal' was not confounded with the addictive vs. nonaddictive disorder distinction, and is thus not a plausible explanation for the differences in the patterns of results between addictive and nonaddictive disorders.

Another concern that could be raised by the potential overlap between the meaning of 'natural' and that of 'normal' is that if participants understood 'natural' to mean 'normal' or 'common', they could have rated the absence of a disorder as more natural than its presence simply because the positive health states invoked in the stimuli are more prevalent than the corresponding disorders. However, as mentioned above, most

people do not seem to be invoking the concept of 'typical' or 'common' when they complete the naturalness rating used here (Lebowitz et al., 2021). Moreover, all of the disorders used in our stimuli were common rather than rare, with some (i.e., obesity and hypertension) affecting more than four in 10—that is, nearly half of—American adults (National Center for Health Statistics, 2020). Thus, the differences in prevalence rates between the presence and absence of the disorders are unlikely to have accounted for the asymmetries in naturalness ratings, given the small magnitude of such differences.

Another limitation of the present research was that although it included a range of physical, mental, and addictive disorders, the finite permutations of stimuli mean that it was not possible to capture reactions to the full range of possible health outcomes. Future research could examine whether the pattern of results observed here generalizes to an even broader range of diagnoses and their corresponding positive health states. Additionally, the present research only compared genetic attributions for positive and negative health outcomes in each domain, but did not examine endorsement of other causal attributions for each outcome. Future research could examine whether the patterns observed in the present research are unique to genetic attributions by investigating whether similar patterns of findings might emerge for other causal explanations.

These experiments indicate that when people consider the causes of a person's health status, they ascribe differing degrees of genetic causation and of 'naturalness', even within the same domain of health (e.g. bone density, body weight), depending on whether the target individual is experiencing a positive health state (e.g. normal bone density) or a negative one (e.g. osteoporosis). To the extent that the observed pattern of genetic attribution reflects a reluctance to attribute some health problems to genetic causes, this could have real-world consequences for healthcare delivery, as belief in genetic and other biomedical etiologies may affect health behaviors, treatment preferences, prognostic expectancies, and even patient-provider relationships (Ahn et al., 2009; Kvaale et al., 2013; Lebowitz & Ahn, 2014; Lebowitz & Appelbaum, 2017; Senior & Marteau, 2007; Wright et al., 2003). In particular, the finding that people may be relatively resistant to genetic explanations for many health problems could help to explain results observed in the existing literature. Years of research have examined the effects of providing individuals with information about their genetic risk for various disorders (e.g. the presence of risk-conferring genes) and found that such information often does not prompt positive health behaviors (Hollands et al., 2016; McBride et al., 2010). If, as our findings suggest, people are somewhat resistant to the notion that genes can predispose people to many poor health outcomes, this could help to explain why personalized genetic risk information often does not lead to behavior change: perhaps a bias toward assuming that genetically influenced health outcomes will be positive prevents people from internalizing information about their own genetic risk for disease to a sufficient extent to produce behavior change. This possibility is speculative, however, and is a potential focus for future studies.

While more research is needed to fully understand why people may, in many cases, disfavor genetic explanations for negative, versus positive, health outcomes, our findings suggest that this preference may be related to a desire to view good health as a default

or 'natural' state. This would be largely consistent with, and build upon, existing research suggesting that people often view genes as representing the fundamental 'essence' of a person (Dar-Nimrod & Heine, 2011; Heine et al., 2019) and prefer to view one's essence, or 'true self', in a positive light (De Freitas et al., 2018; Newman et al., 2014; Strohminger et al., 2017). It may also be an example of a tendency described in prior research for people to 'deploy' genetic explanations in 'strategic' or self-interested ways, such as to support maintaining the belief that their own health risks are relatively low or that they have control over their health and to avoid the fatalism that might arise if they viewed their health problems as deterministically resulting from genetic causes (Condit, 2011, 2019).

Additionally, the notion that negative health outcomes—and particularly those in whose etiology genetics are seen as playing a limited role—are less 'natural' than positive ones may be an as yet under-studied driver of the stigma surrounding a number of mental and physical health problems, which in turn is often a barrier to healthcare access and positive health outcomes (Hatzenbuehler et al., 2013; Hinshaw & Stier, 2008; Puhl & Heuer, 2010; Volkow, 2020). Educating members of the public about the fact that genes play a role in *all* positive and negative health outcomes could ultimately help to dispel problematic beliefs and attitudes that might otherwise prove detrimental to public health.

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Data availability statement

The data that support the findings of this research, as well as the vignettes used in the experimental procedures, are available at https://osf.io/b4wec/?view_only=3e315ec7306a49a7bb5bef1696de187e (hosted via the Open Science Framework).

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