Published in final edited form as:

Nat Hum Behav. 2019 September ; 3(9): 940–949. doi:10.1038/s41562-019-0651-1.

Asymmetrical genetic attributions for prosocial versus antisocial behavior

Matthew S. Lebowitz[*,1], **Kathryn Tabb**[1,2], **Paul S. Appelbaum**[1]

[1]Center for Research on Ethical, Legal and Social Implications of Psychiatric, Neurologic and Behavioral Genetics, Department of Psychiatry, Columbia University Irving Medical Center, 1051 Riverside Drive, Unit 122, New York, NY 10032, USA

[2]Department of Philosophy, Columbia University, 1150 Amsterdam Avenue, New York, NY 10027, USA

Abstract

Genetic explanations of human behavior are increasingly common. While genetic attributions for behavior are often considered relevant for assessing blameworthiness, it has not yet been established whether judgments about blameworthiness can themselves impact genetic attributions. Across six studies, participants read about individuals engaging in prosocial or antisocial behavior and rated the extent to which they believed that genetics played a role in causing the behavior. Antisocial behavior was consistently rated as less genetically influenced than prosocial behavior. This was true regardless of whether genetic explanations were explicitly provided or refuted. Mediation analyses suggested that this asymmetry may stem from people's motivating desire to hold wrongdoers responsible for their actions. These findings suggest that those who seek to study or make use of genetic explanations' influence on evaluations of (e.g., antisocial) behavior should consider whether such explanations are accepted in the first place, given the possibility of motivated causal reasoning.

Attributing human behaviors and characteristics to genetic causes can influence how people perceive and evaluate those behaviors and characteristics.[1] However, the literature suggests that the effects of genetic explanations may depend, in part, on what sort of behaviors or characteristics are being explained.[2] For stigmatized health conditions, genetic attributions are consistently linked to decreased perceptions of affected individuals as blameworthy for their disease or disability.[3–5] That is, conceptualizing conditions like obesity and mental disorders as stemming from genetic and other biological causes can reduce the extent to which individuals are held responsible for them.[3–5] This may be because attributing a

[*]Corresponding author: Matthew S. Lebowitz, Department of Psychiatry, Columbia University, NY State Psychiatric Institute, 1051 Riverside Drive, Unit 122, New York, NY 10032, USA, **Phone**: 914-450-7335, **Fax**: 646-774-8633, msl2207@cumc.columbia.edu.

Author Contributions: M.S.L, K.T and P.S.A contributed to the conception and design of the experiments. M.S.L. collected and analyzed the data. M.S.L. wrote an initial draft of the paper. All authors participated in editing the paper to create the final version.

Competing interests statement: The authors declare no competing interests.

Data Availability

The data that support the findings of this study are available from the corresponding author upon request.

person's behavior to genetic causes can cast him or her as having diminished self-control, insofar as genetic explanations can be interpreted as implying that someone's actions are determined rather than free.[6,7] Substantial evidence suggests that people view agency and the freedom to do otherwise as a prerequisite to holding others responsible for their behavior, on the presumption that people can only be blamed for intentional actions.[8,9]

However, the results have not been as straightforward in studies assessing the application of this logic to judgments about antisocial behaviors, such as crime and other types of wrongdoing. At first glance, this is surprising. In a growing number of countries, purported evidence for the presence of biological predispositions toward antisocial behavior has been introduced on behalf of defendants in pursuit of reduced sentences.[10–12] But while some evidence has suggested that biomedical (including genetic) explanations for criminal behavior can lead defendants to be seen as less responsible for their misdeeds, and therefore treated more leniently,[13] the findings have been quite mixed.[14] Indeed, in most cases genetic explanations for criminal and other antisocial behavior have been found to have no effect on decisions about how to punish criminals and wrongdoers.[15–18]

Why might genetic explanations fail to consistently reduce blame for antisocial behavior? One answer to this question that has been posited in the literature rests on the notion that morality is dyadic in nature, with people perceived as either moral agents (capable of doing good or evil) or as moral patients (recipients of good or evil).[19] It has been suggested that an inverse relationship exists between the two perceptions—a phenomenon known as moral typecasting.[20] According to this formulation, blame can be reduced by portraying a perpetrator as a victim of harm, that is, as a moral patient rather than a moral agent.[21] Unlike some environmental explanations for misconduct, which can compellingly portray the wrongdoer as a victim of harm (e.g., childhood trauma or abuse), recent research has suggested that genetic explanations are not perceived as casting a perpetrator as a victim, or moral patient.[19] These findings may explain why genetic explanations do not always reduce perceptions of blame.

Here we explore another possible reason why genetic explanations might be relatively ineffective at reducing perceptions of blame for antisocial behavior. We do so by investigating whether people are less open to adopting genetic explanations for some types of behavior than for others. In particular, we explore the question of whether people are differentially receptive to genetic explanations for bad acts (operationalized here as antisocial behaviors) versus good acts (prosocial behaviors). If people are more resistant to believing genetic explanations for wrongdoing in the first place, such an asymmetry could illuminate why genetic explanations for misdeeds generally fail to secure more lenient treatment for wrongdoers—such explanations are simply rejected out of hand before decisions about desert are made.

Wide-ranging evidence has shown that the evaluative valence of an event—i.e., whether it is seen as good or bad—affects how and to what extent people will reason about its causes: negative events tend to elicit more causal reasoning than positive ones.[22,23] Additionally, there is ample evidence that people's willingness to accept genetic and other biological explanations for the behaviors and characteristics of others often depends on factors beyond

the intrinsic quality of the explanations themselves, including aspects of the perceivers' own social identities and whether they view the genetic explanations as consistent with their pre-existing social and political commitments.[24–28] Although to our knowledge no research has examined how the moral valence of a behavior affects its likelihood of being attributed to genetic causes, a variety of studies have uncovered other kinds of intuitions and judgments about behavior that can be influenced significantly by moral considerations.[29–31]

One well established example is that people attribute more agency, intentionality, and control to actions that have negative consequences than to those with positive consequences.[32–36] Relatedly, people are more inclined to attribute negative events (rather than positive events) to the agency of others.[37] Moreover, learning about or experiencing the immoral behavior of others causes people to endorse greater belief in free will, apparently in an effort to justify holding wrongdoers responsible for their actions.[38] This may be an example of "blame validation processing," which refers to a "proclivity to favor blame versus nonblame explanations for harmful events and to de-emphasize mitigating circumstances."[39] In light of lay assumptions that genetic explanations for a behavior render it less under the actor's control,[6,7] genetic attributions may be an example of a "nonblame explanation" that, according to the *blame validation* view, would tend to be disfavored in the case of antisocial acts.[39]

Another explanation that has been offered for asymmetries in intuitions about morally good versus morally bad behaviors is the notion that people generally assume that "deep inside every individual there is a 'true self' calling him or her to behave in ways that are morally virtuous."[40] In other words, people generally assume that the essence of every person is inherently good.[41] Because DNA is often seen as representing the essence of a person,[1] this *true self* view might predict that people would be resistant to the idea of genes causing antisocial behavior, because this would imply that a person could have a "bad essence," whereas the true self is assumed by default to be morally good.

Based on the above-described motivations predicted by both the *true self* and *blame validation* views, we hypothesized that people would more readily attribute prosocial (good) behavior to genetic causes than they would antisocial (bad) behavior. We reasoned that these motivations could help explain the often-reported but counterintuitive lack of effect of genetic attributions for wrongdoing. The present research used six vignette experiments to test this hypothesis. Study 1 asked people to estimate the causal role of genes in two instances of either prosocial or antisocial behaviors. Study 2 tested whether differences in genetic attributions for prosocial and antisocial behaviors could be eliminated by giving participants explicit information about the role (or lack thereof) of genes in causing the behaviors described. Study 3 expanded on the findings of Studies 1 and 2 by using a broader range of prosocial and antisocial behaviors, as well as by providing different sorts of explanations regarding the role of genes in causing them. Study 4 tested possible mediators of the asymmetry in genetic attributions for anti- and prosocial behavior. Study 5 examined whether the asymmetry in genetic attributions between prosocial and antisocial actions would occur in response to descriptions of general patterns of behavior, rather than only in response to potentially idiosyncratic accounts of specific behaviors. Finally, Study 6 examined whether such an asymmetry would emerge when the antisocial behaviors

described in the experimental stimuli were violent crimes, such as those judged in the courtrooms where genetic evidence is increasingly brought to bear.

Results

Study 1

We began by examining whether people are differentially likely to perceive genetic causes for prosocial versus antisocial behaviors. In Study 1, participants (see Methods, Studies 1 and 2, Participants) were randomly assigned to either a prosocial condition (n=126) or an antisocial condition (n=125). They were presented with two vignettes (featuring protagonists named “Jane” and “Tom”) in a randomized order. A sentence describing how each protagonist responded to the situation was systematically varied to depict either prosocial behavior in both vignettes (prosocial condition) or antisocial behavior in both vignettes (antisocial condition). Participants rated their genetic attributions for each protagonist’s behavior. See Methods, Studies 1 and 2, Stimuli and Procedures, for more detail. We analyzed these genetic attribution ratings using a univariate ANOVA with condition (prosocial vs. antisocial) as a between-subjects factor and vignette as a within-subjects factor (see Figure 1, panel *a*). The key finding was a significant main effect of condition, $F(1, 249)=19.47$, $p<.001$, $d=.56$, 95% Confidence Interval (CI) = [.37, .75]: participants gave significantly higher genetic attribution ratings for prosocial behavior ($M=3.15$, 95% CI = [2.88, 3.43], $SD=1.58$) than for antisocial behavior ($M=2.29$, 95% CI = [2.02, 2.56], $SD=1.52$). There was also a main effect of vignette, $F(1, 249)=10.11$, $p=.002$, $d_{rm}=.21$, 95% CI = [.03, .38], which emerged because genetic attributions were stronger on average for Tom’s behavior (M=2.83, 95% CI = [2.62, 3.04], SD=1.72) than for Jane’s behavior (M=2.62, 95% CI = [2.42, 2.83], SD=1.65). However, there was no significant vignette × condition interaction, $F(1, 249)=.13$, $p=.723$, meaning that the effect of condition did not significantly differ across the two vignettes. The data from Study 1 are presented as a box-and-whisker plot in Supplementary Figure 1.

Study 2

Having demonstrated a difference in genetic attributions for prosocial and antisocial behaviors in Study 1, we were interested in Study 2 in exploring whether this difference could be eliminated by supplying participants with information about the role (or lack thereof) of genes in causing the behaviors described. Thus, we used the same vignettes as in Study 1, but varied whether the protagonist had been found to be genetically predisposed to the type of behavior exhibited (genetic explanation, n=126) or to lack such a genetic predisposition (no genetic explanation, n=124) (see Methods, Studies 1 and 2). Participants’ genetic attribution ratings (see Figure 1, panel *b*) were again analyzed using a univariate ANOVA, this time with two between-subjects factors—condition (prosocial vs. antisocial) and explanation (genetic explanation vs. no genetic explanation)—and one within-subjects factor (vignette: Jane vs. Tom). As in Study 1, findings were consistent with our hypothesis: participants in the prosocial condition provided higher genetic attribution ratings ($M=2.55$, 95% CI = [2.29, 2.82], $SD=1.48$) than did participants in the antisocial condition ($M=2.18$, 95% CI = [1.93, 2.44], $SD=1.45$), $F(1, 246)=4.48$, $p=.035$, $d=.25$, 95% CI = [.07, .43]. There was also a significant main effect of explanation, $F(1, 246)=50.20$, $p<.001$, $d=.90$, 95% CI =

[.73, 1.06]: participants exposed to the genetic explanation provided stronger genetic attribution ratings (M=2.96, 95% CI = [2.69, 3.24], SD=1.57) than those exposed to no genetic explanation (M=1.76, 95% CI = [1.57, 1,95], SD=1.08). However, there was no significant explanation × condition interaction, $F(1, 246)=.21$, $p=.651$, meaning that the type of explanatory information provided did not significantly moderate the asymmetry in genetic attributions between prosocial and antisocial behavior. There was also no significant main effect of vignette, $F(1, 246)=1.59$, $p=.209$, and no significant vignette × condition [$F(1, 246)=2.71$, $p=.101$], vignette × explanation [$F(1, 246)=.04$, $p=.846$], or vignette × condition × explanation [$F(1, 246)=1.27$, $p=.261$] interactions. The data from Study 2 are presented as a box-and-whisker plot in Supplementary Figure 2.

Study 3

To examine the boundary conditions of the effects documented in Studies 1 and 2, Study 3 used vignettes describing a wider range of behaviors and provided participants with more potent information about the role (or lack thereof) of genes in the behaviors described. Participants (see Methods, Study 3, Participants) read about a woman named Jane encountering one of six situations and behaving in either an antisocial or prosocial manner, depending on the condition to which they had been assigned (see Methods, Study 3, Stimuli and Procedures). Participants were also randomly assigned either to receive a genetic explanation (designed to be more forceful and compelling than the one used in Study 2) or no genetic explanation of Jane's behavior (see Methods, Study 3, Stimuli and Procedures). We analyzed genetic attributions for Jane's behavior using a 6 (situation) × 2 (condition: antisocial vs. prosocial) × 2 (explanation: genetic explanation vs. no genetic explanation) ANOVA. This revealed a significant main effect of condition, with prosocial behavior (n=305, M=3.31, 95% CI = [3.09, 3.53], SD=1.96) attributed significantly more strongly to genetic causes than was antisocial behavior (n=304, M=3.04, 95% CI = [2.82, 3.25], SD=1.92), $F(1, 585)=5.71$, $p=.017$, $d=.14$, 95% CI = [−.01, .29] (see Figure 2, panel *a*). There was also a significant main effect of explanation, $F(1, 585)=573.27$, $p<.001$, $d=1.94$, 95% CI = [1.83, 2.05]; again, the genetic explanation yielded significantly greater genetic attributions (n=305, M=4.52, 95% CI = [4.35, 4.69], SD=1.54) than the lack of a genetic explanation (n=304, M=1.82, 95% CI = [1.68, 1.96], SD=1.23). However, there was no main effect of situation, $F(5, 585)=.84$, $p=.519$, and there were no significant explanation × condition [$F(1, 585)=.69$, $p=.408$], condition × situation [$F(5, 585)=1.16$, $p=.330$], explanation × situation [$F(5, 585)=.1.41$, $p=.218$], or situation × condition × explanation [$F(5, 585)=.42$, $p=.832$] interactions, meaning that the effect of condition (prosocial or antisocial) was not significantly moderated by the situation or explanation to which a participant was assigned. The data from Study 3 are presented as a box-and-whisker plot in Supplementary Figure 3.

Study 4

In order to evaluate the *blame validation* and *true self* accounts described earlier as potential explanations for the findings of Studies 1–3, Study 4 (N=608; see Methods, Study 4, Participants) examined two psychological phenomena that could possibly be mediating people's tendency to endorse genetic explanations less readily for antisocial behavior than for prosocial behavior. In particular, we tested whether the difference in genetic attributions

for antisocial and prosocial behavior was consistent with mediation by differences in ascriptions of responsibility (as would be predicted by the *blame validation* view) and/or by differences in judgments of the extent to which the behavior reflected who the actor truly was (as would be predicted by the *true self* view). Study 4 was identical to Study 3, except that there was no manipulation of the presence or absence of a genetic explanation, and participants rated Jane's level of responsibility for behavior and the extent to which her behavior reflected "who she truly is," in addition to rating their genetic attributions (see Methods, Study 4, Stimuli and Procedures). We initially analyzed the data from Study 4 using a 6 (situation) × 2 (condition: antisocial vs. prosocial) ANOVA. This revealed a main effect of condition on genetic attribution ratings, replicating the results of Studies 1–3: genetic attributions were significantly weaker for antisocial behavior (M=3.00, 95% CI = [2.82, 3.18], SD=1.56) than for prosocial behavior (M=3.72, 95% CI = [3.54, 3.90], SD=1.63), F(1, 596)=31.02, p<.001, d=.45, 95% CI = [.33, .58] (see Figure 2, panel *b*). There was no significant effect of situation, F(5, 596)=1.04, p=.394, and no significant situation × condition interaction, F(5, 596)=.38, p=.862, meaning that the asymmetry in genetic attributions did not differ significantly between vignette versions.

There was also a significant effect of condition on responsibility ratings; participants rated Jane as more responsible for antisocial behavior (n=303, M=6.44, 95% CI = [6.33, 6.54], SD=.96) than for prosocial behavior (n=305, M=6.20, 95% CI = [6.09, 6.31], SD=.98), F(1, 596)=8.85, p=.003, d=.25, 95% CI = [.17, .33]. Like genetic attributions, responsibility ratings showed no significant effect of situation, F(5, 596)=.77, p=.570, and no significant situation × condition interaction, F(5, 596)=.53, p=.752.

Unlike for genetic attribution and responsibility ratings, there was no significant difference in "true self" ratings between the antisocial (M=6.28, 95% CI = [6.17, 6.39], SD=.99) and prosocial (M=6.35, 95% CI = [6.26, 6.45], SD=.85) conditions, F(1, 596)=1.03, p=.31. Once again, there was no significant effect of situation, F(5, 596)=1.60, p=.158, and no significant situation × condition interaction, F(5, 596)=1.12, p=.349.

We evaluated the two mediation hypotheses using the PROCESS procedure (version 3.2) for SPSS with 5,000 bootstrap samples.[42] Figure 3 illustrates the results of this analysis. In particular, there was a significant indirect effect of condition on genetic attributions through responsibility ratings (unstandardized B=.06, 95% percentile bootstrap CI [0.01, .11]), consistent with the notion that participants' motivation to hold Jane responsible for harmful behavior mediated their tendency to rate her behavior as less genetically influenced when she acted antisocially than when she acted prosocially. However, the indirect effect through "true self" ratings was not significant (unstandardized B<.0001, 95% percentile bootstrap CI [−.02, .02]), suggesting that the extent to which participants considered Jane's pattern of behavior to reflect who she truly was did not mediate their differential willingness to make genetic attributions for her prosocial and antisocial behavior. The data from Study 4 are presented as a box-and-whisker plot in Supplementary Figure 4.

Study 5

Study 5 investigated whether the same asymmetry found in the earlier studies for prosocial and antisocial behaviors would occur in response to broad descriptions of a person's

behavior, rather than just potentially idiosyncratic vignettes describing specific actions. This approach also allowed us to attain a more careful degree of experimental control, by writing the prosocial and antisocial versions of the stimuli to more closely parallel each other. Participants (see Methods, Study 5, Participants) were randomly assigned to be told that Jane was generally either “kind, generous, and caring” (prosocial condition) or “mean, selfish, and uncaring” (antisocial condition) (see Methods, Study 5, Stimuli and Procedures).

As in Studies 1–4, participants in the antisocial condition provided significantly lower genetic attribution ratings for Jane’s tendencies (M=2.90, 95% CI = [2.64, 3.16], SD=1.49) than did those in the prosocial condition (M=3.61, 95% CI = [3.36, 3.85], SD=1.41), $t(258)=3.93$, $p<.001$, $d=.49$, 95% CI = [.32, .67]. Notably, the mean of 3.61 in the prosocial condition neared the scale midpoint of 4, which may suggest that, in general, people do not consider genetic attributions to be implausible as explanations for prosocial behavior. By contrast, the mean in the antisocial condition was 2.90, well below the midpoint and significantly lower than for the prosocial condition.

Study 6

The results of Studies 1–5 consistently found an asymmetry in which people were less willing to endorse genetic causes for antisocial (as opposed to prosocial) behavior. Study 6 aimed to investigate whether this asymmetry would emerge when the antisocial behaviors that participants were judging involved serious criminal acts. This is an important question because much of the previous research that has observed no significant effects of genetic explanations on the punishment deemed appropriate for wrongdoing focused on criminal behavior.[15–18] The courtroom is also the most prominent real-life circumstance in which people are asked to evaluate the relevance of genetic causes to responsibility and desert. If people are relatively unwilling to ascribe criminal behavior to genetic causes, this could provide a plausible account of why genetic explanations often do not seem to have significant effects on punishment in such contexts. However, while Studies 1–5 suggest that people may be relatively unwilling to attribute antisocial behavior in general to genetic causes, most of the actions used as examples of antisocial behavior in these studies did not rise to the level of serious criminality, leaving the real-world applicability of the findings (e.g., to criminal cases) somewhat in doubt. Thus, Study 6 used violent crimes for its examples of antisocial behavior, instead of the milder, less extreme examples of antisocial behavior used in Studies 1–5.

Participants (see Methods, Study 6, Participants) were randomly assigned to read about one of two characters, Michael or Nick, either rescuing another person from being the victim of a violent crime (prosocial condition), or committing a violent crime (criminal condition) (see Methods, Study 6, Stimuli and Procedures). Replicating the asymmetry observed in Studies 1–5, participants in the criminal condition provided significantly lower genetic attribution ratings for the behavior they read about (M=3.00, 95% CI = [2.80, 3.20], SD=1.91) than did those in the prosocial condition (M=3.42, 95% CI = [3.20, 3.63], SD=1.97), $F(1, 682)=7.74$, $p=.006$, $d=.21$, 95% CI = [.06, .36].

There was also a significant main effect of character (Michael vs. Nick), $F(1, 682)=4.13$, $p=.043$, $d=.15$, 95% CI = [0.003, 0.30]. This effect emerged because overall, across the different

types of behavior (criminal and prosocial), participants provided greater genetic attributions for Michael's behavior (n=343, M=3.35, 95% CI = [3.15, 3.55], SD=1.88) than for Nick's (n=343, M=3.05, 95% CI = [2.84, 3.27], SD=2.01). However, there was no significant character × behavior interaction [$F(1, 682)$=1.73, p=.189], meaning that the effect of behavior (criminal vs. prosocial) did not differ significantly depending on the character to which a participant was assigned.

Discussion

The primary finding of the present research is that people make weaker genetic attributions for antisocial behavior than for prosocial behavior. Although the magnitude of this effect generally fell in the small-to-medium range (by conventional effect-size definitions), it was replicated across six studies using a range of stimuli that described a variety of prosocial and antisocial behaviors. The asymmetry was present when participants were only given descriptions of behavior and asked to rate how much of a role genetics played in causing it, without being told anything about the actor's genetic predisposition; and it persisted even when participants were told explicitly whether the individual in question was genetically predisposed to the type of behavior exhibited, suggesting that people may remain relatively reluctant to accept even explicit ascriptions of antisocial behavior to genetics. The asymmetry was present across a variety of prosocial and antisocial behaviors, including when the antisocial behaviors in question were violent and criminal, suggesting a degree of generalizability. The relative resistance to genetic explanations for antisocial behaviors demonstrated across these studies might help to explain findings from prior studies, which indicate that genetic evidence often fails to influence the punishments deemed appropriate for criminal wrongdoing.[15–18]

In the present studies, the asymmetry was not moderated statistically by which vignette a participant viewed, despite our use of a wide range of stimuli across the different studies. Moreover, we observed this asymmetry when participants were only provided with general characterizations of a person's broad tendencies that used careful experimental control to maximize the parallels between the prosocial and antisocial stimuli, and were not told about any specific behaviors.

The present research also considered two possible explanations for the observed asymmetry. The results of our mediation analyses in Study 4 were consistent with the *blame validation* account, and not with the *true self* account. The blame validation view would suggest that people see genetic explanations as deflecting moral responsibility for behavior, and therefore disfavor them in the case of antisocial behavior, out of a desire to maintain the ability to assign blame. Our data suggest that this sort of motivation for rejecting genetic explanations for antisocial behavior is more likely than one predicated on resistance to the notion that people's genetic "essences" or true selves could ever predispose them toward morally bad behavior. However, the indirect effect through which responsibility ratings mediated the difference (between prosocial and antisocial behavior) in genetic attributions was small, suggesting that other mediators are also in play. Moreover, the variable we used to assess participants' motivation to hold people responsible for their behavior simply asked participants to rate how responsible they judged people to be, rather than assessing blame or

desire to punish *per se.* We chose to use the item we did because it allowed us to use the same measure for both prosocial and antisocial behavior, but other measures could clarify the question further (see Supplementary Note 1 for further discussion). Additionally, our mediation analyses were correlational, and we did not explicitly manipulate blame, so our data's support for the blame validation account is only preliminary. Elucidating a specific mechanistic account of *why* people reject genetic explanations in some situations more than others was not our primary goal, but future research could examine the mechanisms behind the asymmetry with different methods to further explore this intriguing question.

Our analyses suggest that participants' "true self" ratings in Study 4 did not significantly mediate their lesser willingness to attribute antisocial behavior to genetic causes (as compared to prosocial behavior), but it is notable that "true self" ratings tended to be quite high for both types of behavior (with means above 6 on a 7-point scale). This may be seen as somewhat surprising in light of prior research suggesting that people tend to assume that the true self is morally virtuous.[41] Perhaps participants interpreted the rating scale differently depending on the type of behavior they read about. That is, participants in the prosocial condition may have provided high ratings because prosocial behavior seemed consistent with the notion of a morally virtuous true self. At the same time, those in the antisocial condition may have considered their high ratings as providing further justification or validation of their responsibility judgments (i.e., their ascriptions of blame) by conceptualizing the antisocial behavior as stemming from the perpetrator's deep-seated nature.

Our findings add to the substantial body of existing evidence suggesting that factors beyond the inherent quality of biological explanations for behavior can influence people's likelihood of endorsing them.[24–28] The findings have specific real-world implications, particularly for situations in which genetic explanations for antisocial behavior are deployed strategically, such as when genetic evidence about a defendant is introduced in court as a means of seeking more lenient sentencing. If people are generally resistant to genetic explanations for antisocial behavior—including crime, as we observed in Study 6—judges and jurors may be unlikely to be swayed by such evidence. Indeed, this resistance might help to explain why providing genetic explanations for misdeeds often fails to affect judgments about criminal culpability and punishment in the ways we might expect,[14] as well as the finding that Americans tend to disfavor genetic explanations for violent behavior, as compared to environmental and choice-based explanations.[43]

The present findings may also have implications for other experimental research seeking to measure the impact of genetic explanations for morally significant behaviors on downstream variables, such as ascriptions of blame and praise or judgments about punishment and reward. In particular, any time genetic explanations for undesirable behavior are found not to have an effect on such downstream variables, particularly in the case of antisocial behavior, [17,18] researchers would be wise to consider the possibility that people simply did not accept the genetic explanations in the first place. Studies that attempt to experimentally manipulate people's causal attributions for behavior through the use of genetic explanations should consider employing "manipulation checks" to measure whether such explanations are

effectively impacting participants' beliefs about the influence of genes, especially in cases of antisocial behavior.

Our findings raise a number of important questions for future research. First, it is unclear how unique genetic explanations are in being endorsed more strongly in cases of prosocial behavior than cases of antisocial behavior. Future research could examine whether a similar pattern would emerge for other kinds of explanations, by assessing what other potential "nonblame explanations"[39] might be afforded less weight in attempts to explain antisocial (as opposed to prosocial) behavior. These could include various explanations for behavior that appear to deflect responsibility away from the actor—such as attempts to link criminality to early childhood trauma or socioeconomic deprivation, or attributions of antisocial behavior to neurobiological factors or acquired medical problems.[44]

Insofar as genes are viewed as constitutive of human character and behavior,[1] it may be that they are seen as uniquely or unusually exculpatory, and that causes not associated with people's inner essences would not yield the same asymmetry, or would yield it less strongly. A recent meta-analysis suggests that neurobiological explanations (at least in the context of mental disorders) may not be seen as nonblame explanations to the same extent that genetic explanations are,[45] which would suggest that people would be less resistant to neurobiological explanations of antisocial behavior. As for environmental explanations, some research suggests that they may reduce blame for wrongdoing more powerfully than genetic explanations—which might suggest that people are less resistant to environmental explanations than to genetic explanations in such cases.[19] However, more research is needed to establish this conclusively. It remains to be definitively determined whether other kinds of explanations for behavior besides genetic ones, be they environmental or biological, would yield the same kind of asymmetries observed in the present research.

Future research could also examine whether the asymmetry we report here could be observed using other measures of genetic attribution, such as by gauging genetic attributions on a comparative scale that considers genetic explanations as "one end" of the scale and environmental or psychosocial explanations as the other. All of the present studies used a single measure of genetic attributions that assessed them non-comparatively, which allowed us to assess genetic attributions alone without introducing the possibility of other competing explanations or suggesting to participants that rejecting genetic attributions was akin to embracing another type of attribution. Researchers could also consider using different methods of gauging responsibility/blame and "true self" beliefs rather than the scales used in the present research, which have the potential limitation of being single-item measures.

Additionally, future studies could investigate whether certain individual-difference variables might moderate or interact with the asymmetry documented here. For example, individuals with greater dispositional tendencies toward punitiveness (or those who work in the criminal justice system) might be especially resistant to genetic attributions for antisocial behavior, while perhaps those with expertise in behavioral genetics would show less motivation toward blame validation. Finally, future research could examine whether the asymmetry we observed in the endorsement of genetic explanations is unique to comparisons of prosocial and antisocial behavior, or whether there is a more general willingness to make genetic

attributions for positively valenced phenotypes (e.g., physical attractiveness) than for negatively valenced ones (e.g., ugliness).

When taken together, our results suggest that people's interpretations and evaluations of findings in behavioral genetics may depend not only on the scientific merit of the evidence, but also on the moral valence of the behaviors in question. This kind of motivated reasoning about empirical information can pose obstacles to scientific literacy,[46] underscoring the importance of identifying exactly what motivations are affecting intuitions about behavioral genetics and precisely what impact biological explanations are having on people's thinking.

Methods

All procedures for all studies were administered using Qualtrics.com data-collection software and were approved by the Institutional Review Board of the New York State Psychiatric Institute, indicating compliance with ethical guidelines. In all studies, participants indicated their informed consent to participate via an online form. Qualtrics settings were used to build random assignment of participants to experimental conditions into the study procedures, and because this process was computerized and participants completed the studies online, performing the data collection blind to condition was not a concern.

Studies 1 and 2

Studies 1 and 2 used similar methods.

Participants—In Study 1, participants were U.S. adults (N=251, 45.8% male, 54.2% female, age range 19–73 years, M=34.70 years, SD=10.31) recruited online using Amazon.com's Mechanical Turk (MTurk) platform,[47] which allows participants to complete short tasks in exchange for payment. Participants who completed the study were provided with a "completion code," which they entered into the MTurk system to receive a $1 payment. The sample size was chosen based on a pilot study using the same methods (N=100) that yielded a difference in genetic attributions between prosocial and antisocial behavior with an effect size of d=.36, which requires a minimum sample size of 246 to be detected with 80% power. For Study 2, we recruited a sample of 250 U.S. adults (42.8% male, 57.2% female) using MTurk, based on the same considerations used to determine the sample size for Study 1 (participants in Studies 2–6 were not asked to report their ages; those in Studies 2–5 were recruited and compensated in the same manner as participants in Study 1).

Stimuli and Procedures—The vignettes used for Studies 1 and 2 were largely identical. One described Tom, a 13-year-old student who happened upon a bullying incident in the hallway of his school; the other described Jane, a 30-year-old woman who came across an unconscious homeless man lying in a parking lot in her neighborhood. So that all participants would be exposed to mentions of all of the same behaviors, these sentences took the format "Instead of [prosocial behavior], Jane/Tom [antisocial behavior]" in the antisocial condition, and vice-versa in the prosocial condition (see Table 1 for text of experimental manipulation). In Study 1, other than this experimental manipulation, participants in both

conditions read identical vignettes. In Study 2, several sentences were added to the end of each vignette, explaining that the target individual had recently undergone genetic testing, and the final sentence of each vignette (see Table 2) was randomly assigned to state either that the individual was genetically predisposed to exhibit the type of behavior described in the vignette (genetic explanation, n=126) or lacked such a genetic predisposition (no genetic explanation, n=124).

In both Studies 1 and 2, after reading each vignette, participants were asked, “How much of a role do you think genetics played in [Tom/Jane]’s behavior in the story you just read?” and provided their responses on a scale from “1 (No role or a very minor role)” to “7 (A very major role).” At the end of each study, participants were asked basic demographic questions and were debriefed as to the fictitious nature of the vignettes.

Full stimuli for Studies 1 and 2 are reproduced in Supplementary Table 1.

Study 3

Participants—Study 3 used a 6×2×2 between-subjects design, yielding a total of 24 groups. As such, we calculated that a total sample of at least N=505 would be required for 80% power to detect an effect with the magnitude of the difference in genetic attributions between prosocial and antisocial behavior observed in Study 2. Given that Study 3 used novel stimuli, we reasoned that an even larger sample size might be necessary. Thus, participants in Study 3 were 609 U.S. adults (66.2% female, 33.3% male, .5% other gender or no response) recruited via MTurk.

Stimuli and Procedures—Participants were randomly assigned to read one of six vignettes, each of which described a woman named Jane finding herself in one of six different situations. Depending on the condition (i.e., prosocial or antisocial) to which they had been assigned, participants read about Jane acting either prosocially or antisocially in their assigned situation. Here, participants were not told about the behavior that Jane did *not* perform (as they were in Studies 1–2), because we wished to rule out the possibility that the asymmetry would only emerge when both behaviors were mentioned and the individual explicitly was said to have engaged in one but not the other. All participants were told that the behavior they had read about was “consistent with how Jane usually behaves.” This information was included because we reasoned that participants might have difficulty attributing one specific behavior to Jane’s genetic makeup, but might find a genetic explanation for Jane’s actions more plausible if they were said to be part of a consistent pattern of behavior. Participants were further randomly assigned either to receive a genetic explanation or no genetic explanation of Jane’s behavior.

The genetic explanation used in Study 3 was intended to be more forceful and compelling than the one used in Study 2. To this end, the genetic explanation included an image (see Figure 4), used in previous research,[18] purporting to illustrate the location in the genome of genes that had been shown to cause the type of behavior exhibited by Jane. It also stated, “According to recent testing, Jane has these genes. In other words, Jane’s genetic makeup—the DNA that she inherited from her parents—leads her to behave the way she does in situations like these.” Participants who were assigned to receive no genetic explanation, by

contrast, were not shown this image and were told, “According to recent testing, Jane does not have any genes that are known to lead people to behave this way. In other words, there is no evidence that Jane’s genetic makeup—the DNA that she inherited from her parents—leads her to behave the way she does in situations like these.”

After reading their assigned vignette and explanation, participants were asked, “How much of a role do you think genetics have played in Jane’s patterns of behavior that you just read about?” and provided their responses on the same scale used in Studies 1 and 2. See Supplementary Tables 2 and 3 for the full stimuli used in Study 3.

Study 4

For Study 4, which explored mediation models, although it had fewer conditions than Study 3 (because it did not involve randomly assigning participants to a genetic or no genetic explanation), we wished to be conservative with our sample size because we did not have a clear *a priori* estimate of effect size for our measures of the potential mediator variables. Therefore, we again recruited participants via MTurk, aiming to obtain a sample similar in size to the one used for Study 3.

Participants—Participants were 608 U.S. adults (39.1% male, 60.5% female, .4% other gender or preferring not to answer).

Stimuli and Procedures—The stimuli used in Study 4 were the same as those used in Study 3, except that participants were not randomized to receive a genetic explanation or no genetic explanation for Jane’s behavior. Thus, the study employed a 6 (situation) × 2 (condition: antisocial vs. prosocial) fully randomized, between-subjects design. After participants read their assigned vignette, they were posed two new questions before being asked to rate their genetic attributions for Jane’s behavior using the same scale employed in Study 3. One was a responsibility rating, for which they were asked “To what extent to do you believe Jane is responsible for her patterns of behavior that you just read about?” and provided their responses on a scale from “1 (Not at all)” to “7 (Very Much).” The other, a “true self” rating, asked “To what extent do you think Jane’s patterns of behavior that you just read about reflect who she truly is?” with participants again providing their responses on a scale from “1 (Not at all)” to “7 (Very Much).” The genetic attribution rating appeared below the two mediator ratings in the onscreen procedures, as measuring mediators before measuring outcome variables has been advocated as the preferred temporal sequence for mediation analyses.[48] See Supplementary Table 2 for the full stimuli used in Study 4.

Study 5

Study 5’s methods attempted to address some potential limitations of the methods used in Studies 1–4. In particular, in Studies 1–4, the prosocial and antisocial behaviors about which participants read took the form of vignettes that we created for the purpose of this research. Because the stimuli were not pretested and might be seen as somewhat idiosyncratic, it is possible that they did not manipulate the prosocial/antisocial distinction as intended, that precise experimental control was not achieved or additional variables were manipulated in unforeseen ways. While this appears unlikely given the consistent results of Studies 1–4,

across which a variety of stimuli were used, the possibility cannot be ruled out. Furthermore, because the vignettes in Studies 1–4 described specific behaviors, it is possible that the asymmetry observed so far occurs for the specific behaviors described in these vignettes but not more widely. By contrast, Study 5 used broad descriptions of "Jane" to depict her as either prosocial or antisocial.

Participants—Because Study 5 had only two between-subjects conditions, like Study 1, we aimed to recruit a sample similar in size to the one used for Study 1. Participants were 260 adults recruited via MTurk (36.5% male, 62.7% female, .8% other gender or preferring not to answer).

Stimuli and Procedures—Participants in the prosocial condition (n=130) were told, "Jane has a strong tendency to be kind, generous, and caring toward others. She often goes out of her way to treat people well and help them." By contrast, those in the antisocial condition (n=130) were told, "Jane has a strong tendency to be mean, selfish, and uncaring toward others. She often goes out of her way to mistreat people and take advantage of them."

Participants were given no other information about Jane and were asked, "How much of a role do you think genetics play in Jane's behavior?"; they provided their responses on the same scale used in Studies 1–4. See Supplementary Table 4 for the full stimuli used in Study 5.

Study 6

Study 6, testing whether the asymmetry would occur when serious criminal behavior was compared with strongly prosocial actions, used completely novel vignettes that had not been employed as stimuli in the previous studies, so we aimed to be more conservative in specifying our sample size, choosing to recruit a larger number of participants per condition than for Studies 1–5. We also chose to use a different recruitment method than the one used for Studies 1–5, to enhance the external validity of the findings by examining whether the same pattern of results would emerge in a non-MTurk sample.

Participants—Participants in Study 6 were recruited by Qualtrics, which offers participant recruitment services for a fee, in addition to providing the online data-collection software used for all of the present research. Qualtrics assembled a panel of 686 adult members of the U.S. population to complete the study procedures in the form of an online survey. These participants were 43.9% male, 55.7% female, and 0.4% unknown or unspecified gender. They received compensation equivalent to approximately $6.40 (consistent with standard pay rates used by Qualtrics Panels) via Qualtrics Panels' e-reward system, in which individuals can choose whether to receive their compensation in the form of a gift card, a voucher for free goods/services (e.g., free movie tickets), or points that can be pooled across surveys and then exchanged for a larger reward (e.g., a more valuable gift card).

Stimuli and Procedures—Study 6 used a 2 (character: Michael vs. Nick) × 2 (behavior: criminal vs. prosocial) fully between-subjects design. Participants assigned to the prosocial condition read a vignette describing an incident in which their assigned character rescued another person from falling victim to a violent crime, whereas those in the criminal

condition read a vignette describing an incident in which their assigned character was the perpetrator of a violent crime. The specifics of the crime in question and the events described in the vignette varied depending on the character to which the participant had been assigned.

Similar to Studies 3 and 4, in which participants were told that the behavior they had read about was "consistent with how Jane usually behaves," participants in Study 6 were told that Michael/Nick had "a long history of consistently behaving the way he did during this incident." As in Studies 3 and 4, this information was included because we reasoned that a genetic explanation might befit a pattern of behavior more readily than one specific action. After reading their assigned vignette, participants were asked, "How much of a role do you think genetics have played in [Michael/Nick]'s behavior?" and rated their answers on the same scale used in the earlier studies. See Supplementary Table 5 for the full stimuli used in Study 6.

Data Analysis

Data analysis for all studies was performed using SPSS Statistics Version 25. Two-tailed tests were used for analyses. Data distribution was assumed to be normal, but this was not formally tested. No stand-alone custom code was produced to perform the data analyses. Data analysis was not performed blind to the conditions of the experiments. No participants were excluded from analyses.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This work was funded by a grant from the Program on Genetics and Human Agency of the John D. Templeton Foundation. The first author also received support from National Institutes of Health grant K99HG010084. Additional support for Dr. Appelbaum came from NIH grant RM1HG007257. The funders had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

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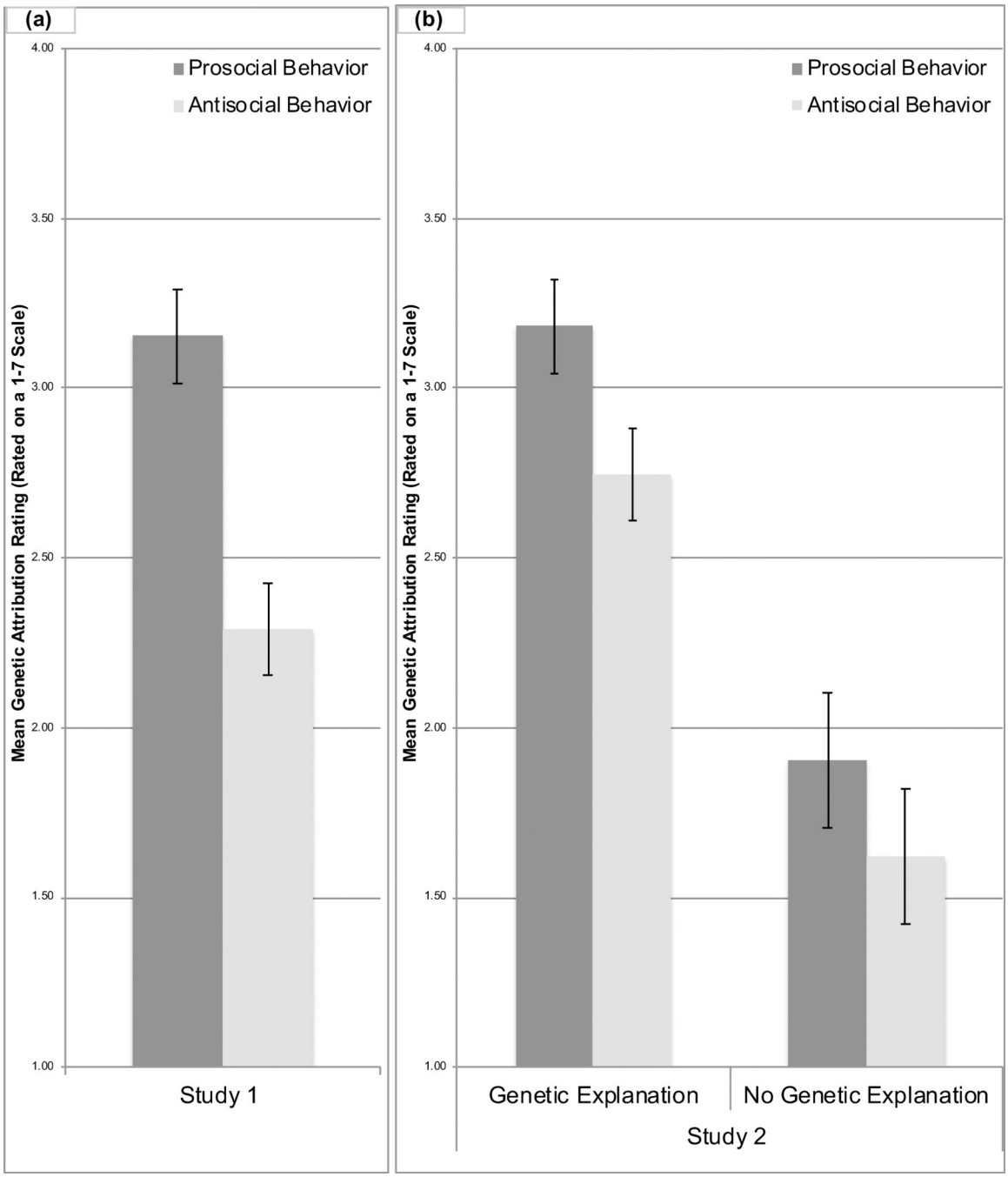

Fig. 1.

Mean genetic attribution ratings in Study 1 (panel *a*) and Study 2 (panel *b*), collapsed across vignettes. After reading two vignettes about either prosocial or antisocial behavior, participants rated how much of a role they believed genetics had played in causing it, on a scale from "1 (No role or a very minor role)" to "7 (A very major role)." Genetic attribution ratings were higher for prosocial behavior than for antisocial behavior in both Study 1, N=251, F(1, 249)=19.47, p<.001, d=.56, 95% CI = [.37, .75], and Study 2, N=250, F(1, 246)=4.48, p=.035, d=.25, 95% CI = [.07, .43]. In Study 2, this was true regardless of

whether or not participants were given a genetic explanation for the behavior they read about. Error bars represent +/− 1 SE.

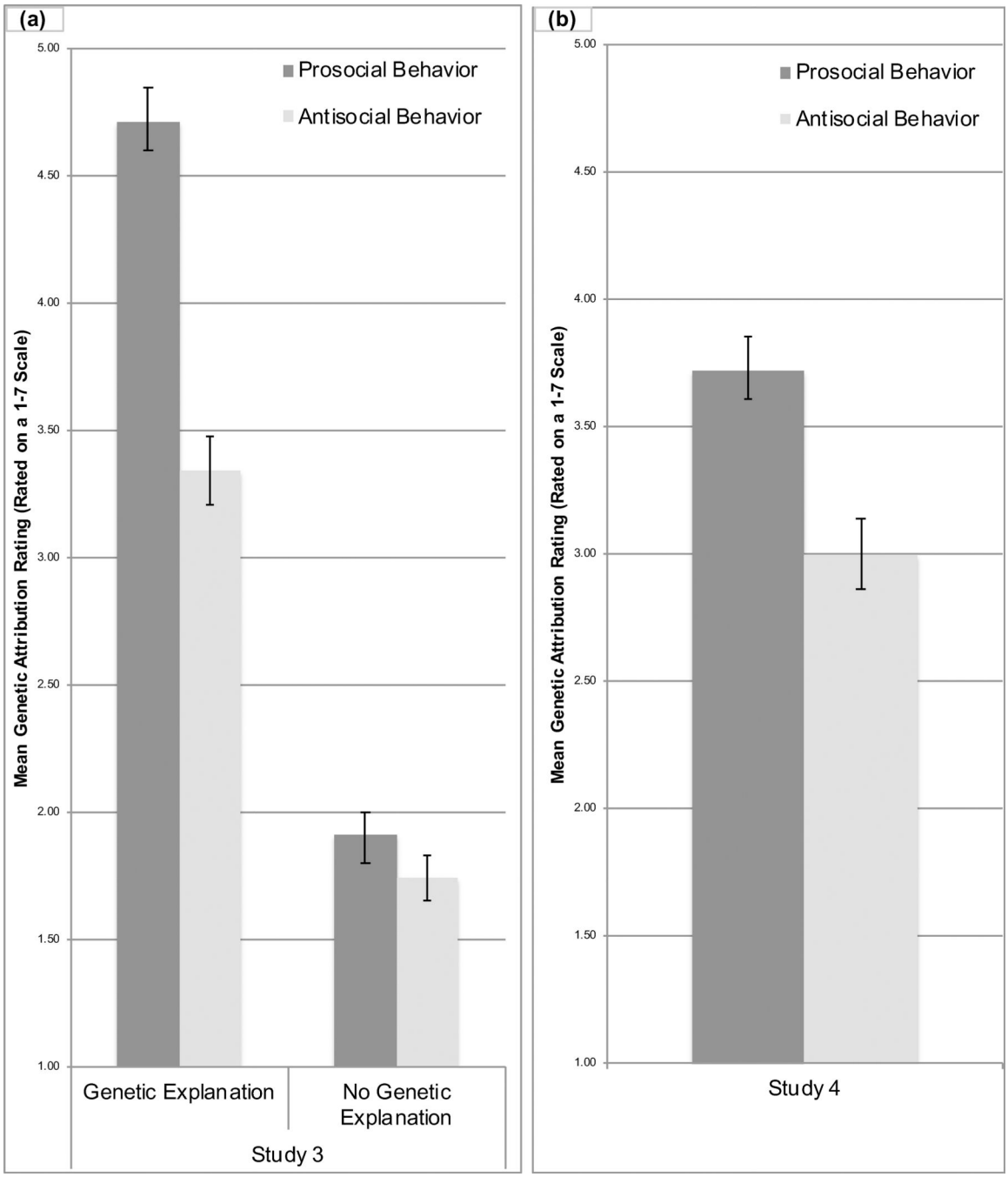

Fig. 2.

Mean genetic attribution ratings in Study 3 (panel *a*; divided by explanation) and Study 4 (panel *b*), collapsed across vignettes. After reading one of six vignettes about either prosocial or antisocial behavior, participants rated how much of a role they believed genetics had played in causing the behavior, on a scale from "1 (No role or a very minor role)" to "7 (A very major role)." Genetic attribution ratings were higher for prosocial behavior than for antisocial behavior in both Study 3, N=609, $F(1, 585)=5.71$, p=.017, d=.14, 95% CI = [−.01, .29], and Study 4, N=608, $F(1, 596)=31.02$, $p<.001$, d=.45, 95% CI = [.33, .58]. In Study

3, this was true regardless of whether or not participants were given a genetic explanation for the behavior they read about. Error bars represent +/− 1 SE.

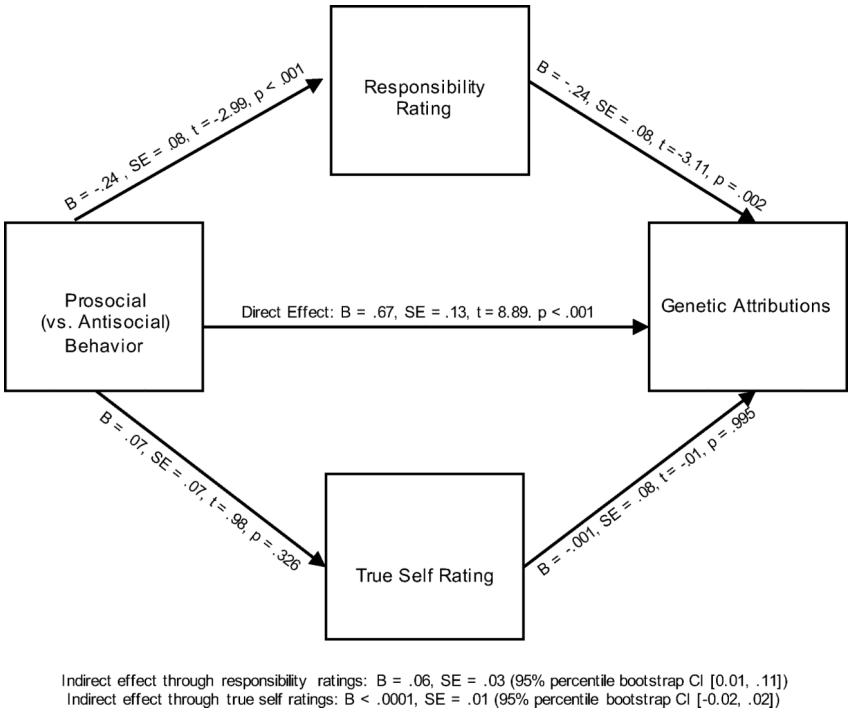

Fig. 3.

Bootstrap mediation analyses in Study 4. After reading one of six vignettes describing a woman named Jane engaging in either prosocial or antisocial behavior, participants rated how much of a role they believed genetics had played in causing Jane's behavior, on a scale from "1 (No role or a very minor role)" to "7 (A very major role)." They also rated how responsible they considered Jane to be for her behavior and provide a "true self" rating of the extent to which Jane's behavior reflected "who she truly is" — both rated on a scale from "1 (Not at all)" to "7 (Very Much)." Mediation models were tested using the PROCESS procedure (version 3.2) for SPSS with 5,000 bootstrap samples.[42] There was a significant indirect effect of condition on genetic attributions through responsibility ratings, but the indirect effect through "true self" ratings was not significant. Regression coefficients are unstandardized.

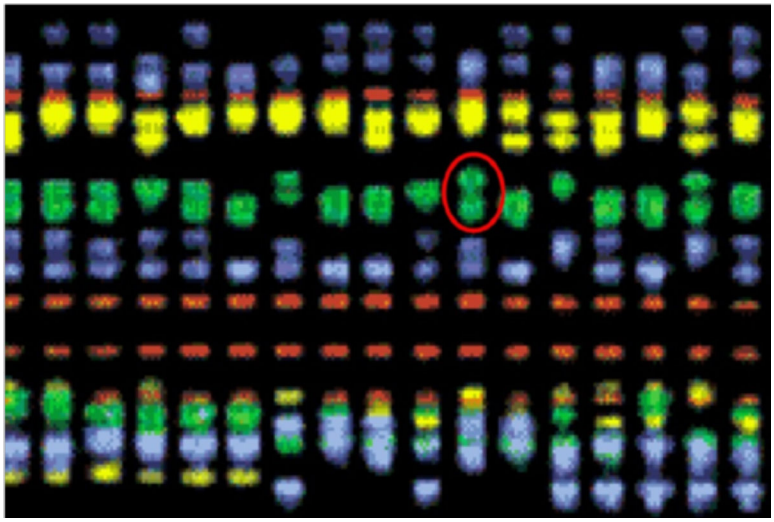

Fig. 4.

Image used in the genetic explanation in Study 3. When Study 3 participants read about either prosocial or antisocial behavior, some were randomly assigned to be given a genetic explanation for the behavior. This explanation, designed to be more forceful and compelling than the text-only one used in Study 2, stated, “Scientists have found that people can have genes that lead them to behave this way. Here is a graphic that illustrates the area of the genome where these genes are found.” This image, which actually shows polymorphic simple sequence repeat markers from several individuals (with alleles for one marker from one individual circled in red), was then displayed. Reproduced with permission from Oxford University Press [Ref 18].

Table 1.

Experimental manipulation used in Study 1.

Vignette Version	"Jane"	"Tom"
Antisocial Condition	Rather than approaching the man and shaking him by the shoulder to wake him up and make sure that he was OK, Jane took the man's cup of money and left him lying in the parking lot.	Instead of coming to the defense of the younger student, Tom joined in with some taunts of his own.
Prosocial Condition	Rather than taking the man's cup of money and leaving him lying in the parking lot, Jane approached the man and shook him by the shoulder to wake him up and make sure that he was OK.	Instead of joining in with some taunts of his own, Tom came to the defense of the younger student.

Table 2.

Experimental manipulation of genetic vs. no genetic explanation, used in Study 2.

Vignette Version	"Jane"	"Tom"
Genetic Explanation	It turns out Jane carries a combination of genes that can make behavior like her response to the homeless man more likely.	It turns out Tom carries a combination of genes that can make behavior like his response during the bullying incident more likely.
No Genetic Explanation	It turns out Jane does not carry a combination of genes that can make behavior like her response to the homeless man more likely.	It turns out Tom does not carry a combination of genes that can make behavior like his response during the bullying incident more likely.