



Scientific supremacy: How do genetic narratives relate to racism?

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Abstract

Recent research suggests that contemporary American society is marked by heightened hostile racial rhetoric, alongside increasing salience of White nationalists who justify an ideology of racial hierarchy with claims of biological superiority. Media coverage of such genetics research has often emphasized a deterministic (or causal) narrative by suggesting that specific genes directly increase negative outcomes and highlighting reported genetic differences between racial groups. Across two experimental studies, we examine the effect of the media's portrayal of scientific findings linking genes with negative health and behavioral outcomes on measures of racism. We find that deterministic genetic attributions for health and behavioral outcomes can lead to more negative racial out-group attitudes. Importantly, we also investigate potential interventions in the presentation of genetic science research. Our research has implications for understanding racial attitudes and racialized ideology in contemporary American politics, as well as for framing scientific communication in intergroup contexts.

Keywords: biopolitics; genetics; media; racial attitudes

Introduction




“An ice cold glass of pure racism”

—White nationalist participating in a milk-chugging party

“Studying human genetic diversity is easier in a society where diversity is clearly valued and celebrated.”

—Dr. John Novembre, University of Chicago evolutionary biologist

In early 2017, shortly after the inauguration of Donald Trump as president of the United States, a group of alt-right White nationalists gathered at the installation of an anti-Trump art piece to hold a “milk party” at which they would chug cartons of cow’s milk together (Harmon, 2018b; Volkov, 2018). The purpose of this display was to highlight and celebrate lactose tolerance, a genetic trait thought to be more common in White people than in others. Unsurprisingly, in addition to consuming lactose together, the White nationalists at this gathering voiced their feelings of racial superiority with racist, antisemitic, sexist, and homophobic rants. Their ideology of racial hierarchy was bolstered, in their view, by science that draws a correlation between White racial identity and the genetic basis for lactose tolerance in adulthood (e.g., Gerbault et al., 2011; Swagerty et al., 2002).¹ These views were widely shared and

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¹Such research theorizes that the genetic basis for lactose tolerance (i.e., the gene that codes for the lactase enzyme, which enables the digestion of lactose) switches off for most people after childhood (e.g., see Gerbault et al., 2011). However, a chance

promulgated among alt-right supporters on social media (e.g., the trending Twitter hashtag #milktwitter) and internet discussion boards (e.g., a 4chan board hosted by the user “Enter the Milk Zone” that featured hate speech telling those who “can’t drink milk” to “go back” to the lands of their ancestors).

Alt-right ideology is characterized by valuing White ethnonationalism and a return to “traditional values” while embracing science and technology (Southern Poverty Law Center, n.d.). Indeed, adherents of alt-right ideology rely heavily on the use of social media and the creation of internet memes to connect with one another and communicate their ideas. Furthermore, they endorse racism that they deem to be scientifically justified, considering themselves “race realists” who believe in “human biodiversity”—in other words, a strong belief in the large genetic differences between different “races” (Panofsky et al., 2021). Even if an individual morally objects to the alt-right’s racial ideology, they may have a difficult time disputing such “scientific justification” because the relationship between genes and race is complex and not particularly well understood among most people. Even those who do understand the relationship better—human geneticists—feel deep discomfort about engaging with and disputing the racist interpretations of their science by White nationalists (Harmon, 2018a).

Part of the challenge of thinking about genes and race is that it is perhaps difficult to separate the ideas that (a) racial categories clearly hold individual, social, and political significance and (b) racial categorizations are commonly based on perceptions of physical features and ancestry. And yet the science broadly suggests that these notions should be separated. Geneticists have demonstrated that “racial classifications are inadequate descriptors of the distribution of genetic variation in our species” (Tishkoff & Kidd, 2004, p. 522). That is, although a minuscule amount of genetic variation across humans may be correlated with socially defined racial categories, such categories are themselves genetically heterogeneous and do not clearly map onto the patterns of human genetic variation in general (Foster & Sharp, 2004; Jorde & Wooding, 2004; Morning, 2011). Yudell et al. (2016) further clarify the distinct notions of heredity and race:

It is important to distinguish ancestry from a taxonomic notion such as race. Ancestry is a process-based concept, a statement about an individual’s relationship to other individuals in their genealogical history; thus, it is a very personal understanding of one’s genomic heritage. Race, on the other hand, is a pattern-based concept that has led scientists and laypersons alike to draw conclusions about hierarchical organization of humans, which connect an individual to a larger preconceived geographically circumscribed or socially constructed group. (p. 565)

It is perhaps unsurprising that for their brand of racial ideology, alt-righters and other White nationalists focus far less on the genetic science that debunks notions of clear racial classifications than on some minor “racial” correlations (like lactose tolerance). But it may be that thinking about genetics through a racial or intergroup lens could have a similar impact on the racial attitudes of the broader population—perhaps especially because of the complexities of the science. Recent research suggests that Americans live in a period of heightened hostile racial rhetoric, and they appear to be increasingly accepting of explicitly negative racial cues (Valentino et al., 2018). Furthermore, racialized cues—subtle or overt—can impact individual attitudes and political decision-making (e.g., Hutchings & Valentino, 2004; Hutchings & Jardina, 2009; Mendelberg, 2001). In this research, we explore how information that is meant to convey scientific findings in an objective, factual, and nonideological manner can impact racial attitudes. Although science itself can often be politicized (e.g., Druckman, 2017), here we suggest that in an intergroup context, the mere communication of scientific information—even without engaging in explicit politicization—can have insidious effects on individuals’ racial attitudes. That is, we argue that subtle shifts in the coverage and explanation of scientific findings can lead to shifts in broader group perceptions that are socially and politically impactful. Our research is thus motivated by

genetic mutation that maintained lactose tolerance into adulthood among the first cattle herders in Europe provided a nutritional advantage that helped proliferate that genetic mutation. A similar evolution occurred among cattle breeders in East Africa, but such evidence may not be of interest to White nationalists.

the notion that “language matters, and the scientific language of race has a considerable influence on how the public (which includes scientists) understands human diversity” (Yudell et al., 2016, p. 565).

Modernizing old-fashioned biological racism

Recent research suggests that old-fashioned racism is back (Huddy & Feldman, 2009; Jardina & Piston, 2019; Jardina & Piston, 2021; Newman et al., 2021; Piston, 2010; Tesler, 2013; Valentino et al., 2018). Explicit, old-fashioned racism that forms the basis for the ideology of White supremacy was particularly accepted and commonplace in the pre-civil rights era, and it is grounded in basic beliefs about the biological inferiority of Black people to White people. The consequences of such beliefs include a social preference for distance between Whites and Blacks, as well as political preferences for formalized, policy-based racial segregation and discrimination (Bobo & Kluegel, 1997; McConahay, 1986; McConahay & Hough, 1976). In contrast, modern racism (also variably called and measured as symbolic racism or racial resentment), which became more commonplace in the post-civil rights era, is justified primarily by a “moral” feeling that Black people violate traditional American values—rather than being biologically inferior per se (e.g., Kinder & Sears, 1981). Whereas for decades in the post-civil rights period, the racial politics literature largely suggested that “old racism” had gone underground in favor of a more symbolic and implicit “modern racism” (e.g., Mendelberg, 2001), contemporary evidence in today’s political landscape suggests the resurgence of the acceptability of explicit racism (e.g., Valentino et al., 2018).² Indeed, in national surveys, over 40% of Americans express the belief that the economic and educational gap between White and Black people can be explained at least “a little” by fundamental genetic differences of race (Huddy & Feldman, 2009). Clearly, at minimum, Americans are open to the idea that biology justifies inequality.

Although the measurement and conceptualization of racial attitudes vary considerably,³ our research focuses on the potentially justifying role of “neutral” biological science findings on negative intergroup attitudes. Therefore, our approach in two experiments is to assess both old-fashioned and modern forms of racism as potential outcomes for thinking about biology through a racialized lens—in part as an initial exploration of how “old” and “new” might meet in the scientific arena.

There is reason to think that the presentation of ostensibly objective scientific information regarding genetics—especially information that reinforces the perception of biological differences between racial groups—leaves an opening for justification of racial disparities. First, genetic science has proliferated in recent decades since the inception of the Human Genome Project in 1990, and this genomic revolution has been accompanied by increased media coverage. A content analysis of coverage by the *New York Times* and the Associated Press between 1985 and 2008 shows not only significant increases in articles about genes, but parallel increases in discussions of race in such articles (Phelan et al., 2013). Accordingly, surveys suggest that among Americans in general—even across ideological, racial, and socioeconomic lines—there are high levels of endorsement of genes as a cause of health and social outcomes (Schneider et al., 2018; Shostak et al., 2009).

Second, genetic science findings, particularly about health, are presented as objective, factual, and nonideological. Phelan et al. (2013) find that news articles that report on genetic differences along racial lines in the context of health (versus other types of outcomes) are less likely to mention issues of racism or ethics, but more likely to affirm genes as causal. These articles therefore suggest that race is not only a

²Some recent theorizing focuses on political context, suggesting that heightened levels of overt racism were fueled in part by White Americans’ reactance to the first Black president, Barack Obama (Piston, 2010; Tesler, 2013), as well as the explicitly racist rhetoric used by Donald Trump during his presidential campaign and subsequent presidency (Newman et al., 2021). However, scholars have also argued that overt racism has been a considerable social and political force even before the presidencies of Obama and Trump (e.g., Huddy & Feldman, 2009).

³A broader discussion of the complexities of measuring and conceptualizing racism and racialized attitudes is outside the scope of this manuscript, but see Huddy et al. (2020) for a recent overview of the literature, including a call for greater consideration of explicit racism measures in ongoing and future research.

valid but a critical way to categorize people, communicated with the authority of science and medicine (Morning, 2008). That is, scientific research and media coverage that endorse any genetic bases of racial difference (health or otherwise) could lead to broader perceptions that there are more general biological differences between racial groups (see Duster, 2003).⁴ Certainly, it appears that such generalizations motivate not only the theatrics of White nationalists' "milk parties," but also provide the veneer of scientific legitimacy to their broader ideology of racial hierarchy.

Finally, when racial group membership is presented as biologically determined, racial inequities are seen as more acceptable, and interest in social interactions with racial out-group members decreases (Williams & Eberhardt, 2008). Critically, such effects are largely independent of individual differences in racial prejudice, which suggests that genetic justifications for racial inequality may not simply be a tool of the alt-right, but rather a cognitive tendency of the broader public.

Modern advances in science and technology may thus play an outsize—if unintended—role in the resurgence of old racism by providing "evidence" for claiming fundamental differences along racial lines, as well as the ideological tools to justify racialized inequities. Our research builds on the foundation in the racial attitudes literature to examine how framing scientific findings in ways that allude to genetically based racial differences in health and behavior might have a more general impact on racial attitudes and political preferences.

The psychological and attitudinal consequences of genetic attributions

Why exactly would genetic attributions be associated with racism? In general, people tend to be genetic essentialists—that is, learning about genetic contributions for human outcomes leads to cognitive biases to perceive those outcomes as immutable and determined, having a specific cause, homogeneous, discrete, and natural (Dar-Nimrod & Heine, 2011). In beliefs about race and inequality, such cognitive biases can be broken down into the constituent components of biological determinism and racial essentialism to form an "ideological double helix" that shapes racial attitudes (Byrd & Hughey, 2015).

Biological determinism is the idea that genes are destiny. When people are told that differences in traits and outcomes are due to genetic differences, they frequently express those outcomes to be inevitable (Jeong, 2007; Phelan et al., 2002). This means that when the concept of race is geneticized, race is perceived as inherited and caused by clear biological markers.⁵ But even among gene-based diseases (which represent only a small subset of gene-based outcomes), only about 2% are monogenic—that is, a particular gene holds a one-to-one relationship with an outcome (e.g., Huntington's disease; Jablonka & Lamb, 2005; Zoghbi & Orr, 2000). However, the vast majority of genotype-phenotype relationships are highly complex. Phenotypes (i.e., observable outcomes) typically emerge from the interaction of many genes, when certain environmental conditions are present, and personal choice factors regarding one's environment that may be both influenced by genes and influence genetic expression. Indeed, many of the ways in which genes relate to human outcomes can be considered "weak genetic explanations" (Turkheimer, 1998), in which an outcome is known to have a genetic basis but the specific mechanisms that translate genotype to phenotype are unknown. Decades of genetics

⁴In addition, analyses of general news media show that representations of racially minoritized group members tend to be negatively stereotyped and portrayed as "law-breaking" compared to representations of white individuals, as well as compared to real-world crime statistics (Dixon & Linz, 2000; Entman, 1994; see also Hutchings & Valentino, 2004). Thus, the existing news environment may well prime consumers of genetic science articles with racially biased perceptions (see Valentino, 1999).

⁵Research on media articles about genetics research also demonstrates the prevalence of simplified genetic explanations that often suggest the strong causal role of genes (Conrad, 1997). Perhaps more problematic, most people receive the majority of their genetic knowledge from the media (as opposed to the scholarly source directly), in part because of the sophisticated level of knowledge required to read and comprehend genetics literature as opposed to other scholarly research (e.g., history, psychology, political science; Conrad, 1997). Dar-Nimrod and Heine (2011) note that such simplified accounts reinforce cognitive error, creating an opportunity for increased stereotyping, in part to deal with the complexity of human behavior and a lack of baseline knowledge regarding genetics research.

research suggest that nearly all human behaviors are, to some extent, heritable, including cigarette smoking (Kendler et al., 2000), divorce (Jocklin et al., 1996), voting behavior (Fowler et al., 2008), and political ideology (Alford et al., 2005). But it is important to keep in mind that the specific genes involved, the precise pathways from genes to outcome, and the particular environmental conditions conducive to genetic expression are complex and not yet well understood. As noted earlier, racial categories do not map onto clear genetic markers (Tishkoff & Kidd, 2004; Yudell et al., 2016), but belief in genetic determinism is positively associated with prejudice, negative racial stereotyping, and nationalism (Keller, 2005).

Racial essentialism follows from the belief that genetic variation is immutable and innate, and so any associated outcomes are also immutable and innate—that is, genes form the “essence” of who a person is. In other words, genetic essentialism “reduces the self to a molecular entity, equating human beings, in all their social, historical, and moral complexity, with their genes” (Nelkin & Lindee, 1995, p. 2). This translates to the group level, such that perceived differences between social groups, insofar as they are attributed to biological causes, are seen as natural differences in group “essence” (Levens et al., 2001). Despite evidence that racial and ethnic “essences” do not exist, the perception of difference along biological essence persists (Gil-White, 2001). Essentialist thinking has been shown to increase perceptions of group homogeneity and stereotype endorsement about social and racial groups (Bastian & Haslam, 2006; Haslam et al., 2006; Haslam et al., 2000, 2002; Hong et al., 2003). For instance, compared to perceptions of race as socially constructed, essentialist views of race lead to greater perceived social distance and less cultural overlap between Asian Americans and American identity and culture (No et al., 2008). Moreover, research by Kimel et al. (2016) demonstrates that emphasizing genetic differences between ethnic in-group and out-group members can increase behavioral aggression and support for hawkish policies.

Clearly, there are psychological and political reasons to be cautious and concerned about widespread and simplified dissemination of genetics research. Indeed, Schmalor et al. (2021) suggest that scientific narratives (i.e., genetics research that describes the geographic clustering of particular allele frequencies) can increase beliefs that group stereotypes are largely due to genetics. Are there ways to mitigate the psychological and attitudinal biases that can stem from reports of genetics research in racialized contexts? Dar-Nimrod and Heine (2011), in an extensive review of genetic essentialism, point out that “although the scientific importance of genetic research is beyond dispute, taking steps to ensure a reduction in the undesirable cognitions and behaviors that have so far dogged the study of genetics will go a long way towards fulfilling the great promise encompassed in such research.” (p. 25) We respond to the call by Dar-Nimrod and Heine, who note the lack of experimental studies on the open question of whether the negative effects of deterministic, essentialist thinking on racial attitudes can be reduced through interventions that frame genetics research in more contextualized ways.⁶ There is reason to think that such interventions could be effective, with at least one study showing that messages portraying mental illness as an outcome of both genes and environment (versus purely genes) reduced perceptions of danger associated with schizophrenics (Walker & Read, 2002). Along these lines, we investigate whether intergroup attitudes can be modulated by varying the type of narrative regarding the relationship between genes and social group outcomes.

The current research

We have two primary goals with the current research, which we address with two experiments. The first goal is to investigate whether it is possible to frame genetic findings in a way that reduces the negative

⁶Yudell et al. (2016) argue that the concept of race should be abandoned altogether in genetics research. Although this could help resolve many of the issues outlined here, given that the science of racialized genetics has persisted, our approach focuses on examining how existing narratives about the relationship between genes and race can be framed to mitigate the negative effects on racial attitudes.

impact on racial attitudes. Specifically, we compare frames of the same racialized genetics research as either deterministic (i.e., simple and causal) or conditional (i.e., complex and contingent on multiple genes and gene by environment interactions). Some previous experimental studies suggest that conditional or contextualized genetic explanations for mental illness and obesity can improve attitudes regarding those outcomes—albeit without the racialized context (Teachman et al., 2003; Walker & Read, 2002). We extend the existing work by investigating whether such conditional narratives can also lead to more tolerant attitudes if genetic attributions are made for *racial* group differences in health and behavioral outcomes. We expect that deterministic genetic narratives will replicate much of the prior work examining genetic essentialism in racial contexts by increasing racist attitudes. We also explore the possibility that conditional genetic narratives will reduce the negative impact on racial attitudes of presenting racialized genetic differences.

A second, more exploratory aim is to conduct a theoretically stringent test of the idea that genetic narratives in racial contexts will lead to higher levels of racism. That is, the literature on intergroup effects of genetic determinism and essentialism largely suggests that genetic explanations for racial differences lead quite consistently to more stereotyping, perceptions of out-group homogeneity, and prejudice (Condit et al., 2004; Dar-Nimrod & Heine, 2011; Heine et al., 2017). However, genetic attributions for some outcomes appear to lead to more tolerant attitudes—that is, “born that way” narratives can elicit greater sympathy for and diminished condemnation of members of typically stigmatized groups. For example, genetic (versus behavioral) explanations for obesity led to lower implicit anti-fat attitudes and greater explicit pro-fat attitudes (Teachman et al., 2003). In addition, genetic explanations for violence or criminality have been associated with lower perceptions of culpability and preferences for less punitive consequences (Cheung & Heine, 2015; Heath et al., 2003; Monterosso et al., 2005). Therefore, we examine genetic attributions for *outcomes* that have been associated with greater tolerance (i.e., obesity and violence) in a racialized context to test the potential strength or persistence of racist attitudes. Our expectation is that the negative attitudinal effects of linking race and genetics may override any positive attitudinal effects of linking traits and behaviors (like obesity and violence) to genetics, consistent with maintenance of the existing racial hierarchy and justification of racialized ideology (Jost & Banaji, 2004; Pratto et al., 1994).

Study 1

Our initial study examines the effects of deterministic versus conditional narratives about racialized genetic associations with obesity on a measure of modern racial prejudice, as well as policy attitudes in the domain of health care. Specifically, we make use of modified news articles that highlight racial variation in the prevalence of genetic variants related to obesity. With this study, we aim to test two primary hypotheses:

H1: Individuals who read news articles emphasizing a *deterministic* genetic narrative about obesity will be *more likely* to report negative attitudes toward African Americans and more negative attitudes toward comprehensive health care policy (than those who receive conditional narratives or unrelated news articles).

H2: Individuals who read news articles emphasizing a *conditional* genetic narrative about obesity will be *less likely* to report negative attitudes toward African Americans and less negative attitudes toward comprehensive health care policy (than those who receive deterministic narratives or unrelated news articles).

Methods

Participants and experimental design

Data for our initial study were collected in May 2018 with an undergraduate student sample ($N = 146$). Participants were recruited to participate in a survey on how “media coverage of academic research

impacts the public's attitudes" for course credit. Several demographic questions that have been shown to be correlated with both racism and deterministic attitudes (Schneider et al., 2018) were asked of the respondents following the post-treatment measures. Survey respondents were asked to best describe their marital status (single, married, widowed, separated, divorced, remarried, living together). A majority of the respondents were single (93.15%), followed by living together (4.79%), married (1.37%), and divorced (0.68%). Respondents were asked to report their age ($M = 21.40$, $SD = 2.51$); the highest level of education they had achieved (61.90% had some college, 19.73% had completed college, 12.93% had completed high school, 2.04 reported other, 1.36% had completed some technical school, 1.36% had completed some high school, 0.68% had completed elementary school); their gender (46.26% reported being female, 51.02% male, 0.68% gender queer/gender nonconforming, 0.68% transgender female, and 1.36% transgender male); and their income (open response, a majority of respondents reported an income less than \$50,000 a year). In addition, respondents were asked to rank their political ideology along the liberalism/conservatism scale (from strongly liberal = 1 to strongly conservative = 7; $M = 3.44$, $SD = 1.53$).⁷ Summary statistics for each item are reported in Appendix A, Table 2.

Participants were randomly assigned to one of three possible conditions, all of which entailed reading a news article (see Appendix A, Table 1 for the distribution of treatments across respondents). The two experimental conditions varied the framing of a genetics research narrative (deterministic versus conditional), and the control condition text was unrelated to genetics research (see Appendix A for full treatment materials).

Deterministic obesity condition. Participants assigned to the deterministic condition read an article titled "If you're obese, a single gene may be to blame," which described a genetics study linking a gene called ankyrin-B to "larger-than-normal" fat cells. The text emphasized a deterministic relationship between the gene and obesity, stating that "a single gene could be at the root of why some people are overweight." The article also described the gene as affecting 8.4% of African Americans compared to 1.3% of Caucasians, and it was accompanied by a picture of a torso of an overweight Black person.

Conditional obesity condition. Participants assigned to the conditional condition read an article titled "If you're obese, genes in addition to diet and exercise may be to blame," which described the same genetics study linking ankyrin-B to "oversized" fat cells. Like the deterministic health treatment article, the conditional treatment article was also accompanied by a picture of a torso of an overweight Black person, and it stated that the proportion of African Americans carrying the ankyrin-B gene was higher than that of Caucasians. However, this article included conditional language regarding the research, stating that "there's a lot more research on the gene that still needs to be done. Researchers will need to look into the family histories, physical characteristics, and metabolism of those with forms of the gene in order to truly figure out how it will affect people in addition to their exercise and diet habits." That is, the conditional treatment highlighted that the outcome is conditioned by other environmental factors in addition to genetic factors, as well as personal choice.

Control condition. Participants assigned to the control condition read an article unrelated to genetics research. The control condition text was instead about summer homework, titled "Summer homework: Seeing vacation homework from the perspectives of educator and parent." The article discussed the purpose and procrastination of summer homework, as well as other kinds of learning that can

⁷We used this scale to create a dichotomous variable *conservatism*. Individuals who scored 5 or greater (above the third quartile) were coded 1 on the variable *conservatism* and 0 otherwise.

Table 1. Deterministic and conditional genetic explanations for obesity and symbolic racism and support for universal health care (Study 1)

Variables	(1)	(2)
	Symbolic racism	Universal health care
Deterministic treatment	0.276* (0.116)	-0.836* (0.339)
Conditional treatment	0.193+ (0.103)	-0.252 (0.340)
Single	-0.007 (0.144)	0.346 (0.386)
Age	-0.009 (0.016)	0.095+ (0.052)
Education	-0.070* (0.034)	-0.123 (0.092)
Conservatism	0.745** (0.114)	-1.924** (0.390)
Black [†]	-0.243 (0.152)	0.792 (0.518)
Asian	0.251* (0.113)	-0.020 (0.378)
Latinx	-0.097 (0.144)	-0.027 (0.437)
Female	-0.156+ (0.093)	0.722* (0.287)
Income	0.003* (0.001)	-0.001 (0.004)
Constant	2.230** (0.475)	3.996** (1.450)
Observations	129	129
R ²	0.414	0.343

Note: Robust standard errors in parentheses.

[†]White is the reference category.

** $p < .01$; * $p < .05$; ⁺ $p < .1$ (two-tailed).

occur over summer vacations without assigned schoolwork. The purpose of this condition was to provide a baseline assessment of participants' racial attitudes and policy preferences without priming them with genetics research regarding health, and without priming perceptions of African Americans.

Table 2. Change in overt racism and genetic explanations for violence (Study 2)

Variables	(1)	(2)
	Overt racism	Overt racism
Deterministic—Race	0.285* (0.113)	0.291* (0.114)
Conditional—Race	0.133 (0.113)	0.133 (0.114)
Deterministic—No race	0.109 (0.112)	0.098 (0.112)
Conditional—No race	0.066 (0.109)	0.060 (0.109)
Age		0.002 (0.003)
Education		0.017 (0.024)
Conservatism		-0.032 (0.020)
Female		-0.005 (0.082)
Black [†]		-0.204 (0.126)
Asian		-0.195 (0.188)
Latinx		0.105 (0.134)
Income		-0.003 (0.028)
Constant	0.004 (0.080)	-0.032 (0.236)
Observations	1,273	1,269
R ²	0.006	0.013

[†]White is the reference category.

***p* < .01; **p* < .05; †*p* < .1 (two-tailed).

Outcome measures

Following the experimental treatment, all participants responded to a multiple-choice manipulation check item to ensure they had accurately read and understood the main themes of the article they were

given.⁸ Then participants responded to attitudinal measures that were presented in random order (see Appendix A for all item wordings).⁹

Symbolic racism. Racial attitudes were measured using the eight-item symbolic racism scale (Henry & Sears, 2002) designed to assess the extent to which the respondent expresses a modern form of racism, predicated upon the belief that African Americans in present-day America do not experience racial discrimination and thus restitution is no longer justified (e.g., “It’s really a matter of some people not trying hard enough; if blacks would only try harder they could be just as well off as whites” on a range from 1 = strongly disagree to 5 = strongly agree).¹⁰ A scale was constructed using the average score of the eight items (see Appendix A for the complete scale items and coding rules); higher values reflect more racist attitudes toward African Americans.

Health care policy attitudes. We expected that genetically deterministic perceptions of obesity could also affect attitudes about health care policy, including access to health care. Thus, we assessed health care policy attitudes by asking respondents their degree of support for universal, guaranteed health care. Specifically, we asked respondents the extent to which they agreed with this statement: “Some people say that health care should be a right for all people and not a privilege for those who are insured by their workplace or participate in some other private plan. Others say that the tax burden in this country is already high and it is unreasonable to expect people who are paying a part of their own private insurance plan to also pay for other people. How do you feel about universal, guaranteed health care?” (1 = strongly oppose universal, guaranteed health care to 7 = strongly support universal, guaranteed health care).

Results

Treatment effects on symbolic racism

In Table 1, we report the results of ordinary least squares regression models (with robust standard errors in parentheses) that assess the effects of the deterministic and conditional treatments on symbolic racism. Consistent with our expectations (*H1*), the results suggest that those who read the deterministic narrative are more likely to express symbolically racist views of African Americans than those who read the control narrative ($p < .05$, two-tailed), controlling for respondent race/ethnicity, age, education, conservatism, gender, and income. However, we also find that individuals who receives the conditional narrative emphasizing genetic factors in addition to environmental factors and personal choice are marginally more likely to report higher levels of symbolic racism than the control group ($p < .10$, two-tailed), while accounting for respondent race/ethnicity and other covariates, which is inconsistent with our expectation (*H2*).

Treatment effects on health care policy attitudes

Next, we examine the association between the treatment conditions and support for universal health care. As reported in Model 2 of Table 1, the results suggest that reading the deterministic narrative about obesity is associated with significantly lower support for universal health care compared to the control condition ($p < .05$, two-tailed), while additionally accounting for differences in respondent race/ethnicity, age, education, conservatism, gender, and income. On the other hand, the conditional

⁸Seventeen participants did not respond to this item. These same 17 participants did not respond to the focal outcome measures; thus, they are not included in the analysis sample.

⁹Respondents who were given the deterministic genetic narrative were significantly more likely to select “There is a strong genetic component to obesity” than any other description of the text following the treatment condition ($p < .001$, two-tailed).

¹⁰More specifically, the term “symbolic racism” comes from the idea that the negative evaluation of African Americans is *racism*—that is, reflective of racial antipathy (Henry & Sears, 2002)—and *symbolic*—that is, representative of a violation of moral values formed by Whites from a young age and not specific to African American individuals but toward African Americans as a collective group.

narrative is not associated with lower support for universal health care compared to the control ($p > .10$, two-tailed).

The results of Study 1 provide partial support for our hypotheses. That is, when respondents are exposed to a deterministic genetic narrative about obesity in a racialized context, they express higher levels of symbolic racism. But the conditional genetic narrative also leads to marginally higher expressions of racism. It may be that simply priming the reader by referencing race in any form increases expressions of racism. Perhaps because both the deterministic and conditional narratives highlight genetic differences between racial groups (relative to an unrelated control condition), such genetic narratives unsurprisingly elicit greater racism. We see a greater effect of the conditional genetic treatment on policy attitudes, however. Whereas the deterministic genetic treatment leads to more restrictive views on universal health care, the conditional genetic treatment does not lead to any more restrictive views on universal health care than the control treatment. These initial results thus suggest the possibility that contextualizing the effects of genes in racialized narratives could affect consequential downstream attitudes.

Study 2

The second study builds on our initial study in several ways, addressing questions that were left open in Study 1. First, because our first study used a between-subjects design, we cannot know whether participants' racial attitudes were changed by the treatments or whether they were simply primed. Indeed, it is possible to interpret the increased expressions of racism in both the deterministic and conditional treatments simply as evidence of racial priming. Repeated measures designs can assess *change* in attitudes, and they have been shown to increase precision without altering treatment effects (Clifford et al., 2021). Therefore, we implement a two-wave study in which we measure racial attitudes before and after treatment to assess whether participants' racial attitudes change as a function of the genetic narrative.

Second, we further increase the precision of our treatment effects by applying a more stringent control group comparison. We do this by (1) disentangling the racial and genetic explanation effects in our treatment conditions and (2) aligning the topic of the control text to be closer to the treatment texts (instead of a completely unrelated topic).

Third, we expand our examination of the effects of genetic explanations on racial attitudes by exploring a measure of overt racism. If, as we suspect, racialized genetic science contributes to heightened racial hostility, then we should also observe greater willingness to express explicitly racist views when given genetic attributions for racial differences. We further seek to understand potential changes in racist attitudes by considering the role of essentialist beliefs—the perception that differences between racial groups are differences in innate and immutable features. That is, we explore essentialist beliefs as a potential mechanism by which genetic narratives may be associated with heightened racism.

Finally, we examine a different outcome domain for which a genetic explanation is given—violence. In addition to increasing the generalizability of genetic narrative effects by assessing a behavioral outcome unrelated to health, the purpose of exploring violence in this study is to examine a behavioral domain in which there are strong racial stereotypes of Black criminality, but which has itself been associated with lower levels of punitiveness given genetic explanations (e.g., Cheung & Heine, 2015).

Drawing on the results from Study 1, we preregistered our hypotheses, which are largely parallel to our expectations in the initial study.¹¹ We expect broadly that exposure to genetic research that is explained in a deterministic way will lead to increased expressions of racism (*H1*). However, we expect that this effect will emerge specifically when the genetic explanation is racialized (i.e., the text reports differences in the prevalence of the genetic variant between White and Black racial groups; *H1a*). That is,

¹¹Our preregistration document can be reviewed at <https://aspredicted.org/5va3p.pdf>. Data and analysis code are available at <https://osf.io/59kqg/>. Although we did not preregister Study 1, our data and analysis code from Study 1 are available at this URL.

although genetic attributions for violence might lead to increased sympathy for perpetrators of violence in the absence of a racialized context (Heine et al., 2017), we anticipate that highlighting racial differences in genetic markers for violence will override such sympathy and instead lead to more hostile racial attitudes.

In contrast, we tentatively hypothesize that conditional genetic narratives—across racial and non-racial contexts—will prevent the heightened racism (*H2*). That is, we explore the possibility that providing contextualized explanations of genetic associations leads to *no* change in racial attitudes, effectively diminishing the attitudinal impact of genetic attributions for racial differences.

Methods

Participants and experimental design

Data were collected on a national sample matched to the U.S. Census ($N \sim 2,500$) via Dynata. Wave 1 of the data was collected near the end of August 2019.¹² These same respondents were then asked to complete Wave 2 of the study approximately two weeks later, September 9, 2019. In total, 2,504 people completed both waves of the survey. The survey comprised two separate experiments; we describe one experiment here, in which participants received one of five treatments ($N = 1,276$).¹³ Participants were surveyed on our items of interest (i.e., expressions of overt racism) and other pre-treatment demographic questions¹⁴ in Wave 1 and then were reassessed on these items following the random assignment of the five conditions (four treatments; one control) in Wave 2.

Experimental conditions

The experimental treatments varied along two primary dimensions: (1) whether the explanation for the outcome is described as caused by a specific genetic variant (*deterministic*) or whether genetic effects are conditioned by other environmental conditions, genetic factors, and personal choice (*conditional*); and (2) whether racial categories are mentioned as part of the discussion of the incidence of a genetic variant associated with violent behavior (i.e., *race* versus *no race*). The control condition was on the subject of violence but without any reference to genetics or race (see [Appendix B](#) for full treatment materials). Post-treatment, subjects were again asked to report their racial attitudes.

Deterministic violence condition—Race. Participants assigned to the deterministic violence condition read a news article titled “‘Warrior gene’ may contribute to violent crime, studies say,” which described real genetics research that found a link between genes and violent behavior. The text emphasized the role of the gene, MAOA, in manifestations of “extremely violent behavior.” In addition, this text referenced the distribution of this gene by race: “Researchers found that more African Americans carry the problematic form of the gene than Caucasians. 8.4% of African Americans and 1.3% of Caucasians carry forms of the gene, which include millions of Americans.”

¹²The Wave 1 sample was balanced to the U.S. Census on age, gender, ethnicity, and census region. $N = 5,000$ were surveyed by Dynata in Wave 1 and Wave 2 with the goal of $N \sim 2,500$ completes. 2,504 people ultimately completed both Wave 1 and Wave 2.

¹³The remaining respondents ($N = 1,228$) received treatments on a separate phenotypic outcome, not the subject of this particular research report. The preceding analyses were conducted on the sample of 1,276 respondents who received only one of the five conditions reported in this study. Subjects were randomly assigned to all conditions and were not preassigned to either study. For a complete breakdown of the distribution of violence treatments across survey respondents, see [Appendix B, Table 1](#).

¹⁴Several demographic questions were asked of the respondents prior to the treatment measures specified to avoid any effect of conditioning on posttreatment variables (Montgomery et al., 2018). Respondents were asked to report their age, their highest level of education, their political identity along the 7-point liberal/conservative scale, their gender, their race and/or ethnic identity, and their income. Summary statistics for each item are reported in [Appendix B, Table 2](#).

Deterministic violence condition—No race. Participants assigned to the deterministic violence condition read a news article titled “‘Warrior gene’ may contribute to violent crime, studies say,” which described genetics research that found a link between genes and violent behavior. The text emphasized the role of the gene, MAOA, in manifestations of “extremely violent behavior” but made no reference to the distribution of this gene by race.

Conditional violence condition—Race

Those assigned to the conditional violence condition read a similar news article titled “‘Warrior gene’ may interact with environment to contribute to violent crime, studies say,” which described similar genetics research linking MAOA to violent behavior, but this article emphasized the “complexity of the issue” given a large environmental component and quoted an expert who asserted that “these genes ... most emphatically do not predetermine you for a life of crime.” In addition, this text referenced the distribution of this gene by race: “Researchers found that more African Americans carry the problematic form of the gene than Caucasians. 8.4% of African Americans and 1.3% of Caucasians carry forms of the gene, which include millions of Americans.”

Conditional violence condition—No race. Those assigned to the conditional violence condition read a similar news article titled “‘Warrior gene’ may interact with environment to contribute to violent crime, studies say,” which described similar genetics research linking MAOA to violent behavior, but this article emphasized the “complexity of the issue” given a large environmental component and quoted an expert who asserted that “these genes ... most emphatically do not predetermine you for a life of crime.” Moreover, this treatment did not reference any distribution of this gene by race.

Control condition. Those assigned to the control condition read an article about violent crime as a function of environmental influences (i.e., trauma), but without reference to genetics or race. See [Appendix B](#) for full treatment materials.

Outcome measures

Overt racism. We employed a measure of overt racial prejudice adapted by Huddy and Feldman (2009) from long-standing questions in the General Social Survey. Of particular interest to us, this measure assesses the extent to which the respondent believes “fundamental genetic differences between the races” serve as explanations for economic disparities among Blacks compared to Whites, more closely assessing the mechanism through which deterministic genetic narratives can lead to overtly racist attitudes (i.e., “On average, African Americans have lower income and worse housing than white people. How much of the economic difference between blacks and whites occurs because of fundamental genetic differences between the races?” measured on a 5-point Likert scale from “none” to “a great deal”). We administered this measure (composed of six items in total) in Wave 1 (pre-treatment) and Wave 2 (post-treatment) and calculated the average response across the items for each subject in Wave 1 and Wave 2; the dependent variable in the analyses below is the within-subject mean difference in reported overt racism post-treatment (Wave 2) – pre-treatment (Wave 1).

Essentialism. We suspected that deterministic genetic narratives were also likely to increase essentialist beliefs that genes make up the core of who a person is. Therefore, we administered a scale of essentialist beliefs in Wave 2 (post-treatment) using the average response reported across the eight-item Bastian and Haslam (2006) essentialism scale (e.g., “There are different types of people and with enough scientific knowledge these different ‘types’ can be traced back to genetic causes”; on a scale from 1 = strongly disagree to 7 = strongly agree). The mean response across all items was approximately 4.27 ($SD = 0.87$).

Results

Treatment effects on overt racism

Table 2 reports the results of a linear regression model assessing the effects of within-subjects treatment on expressions of overt racism. In our models, we regressed our measure of change in overt racism on the experimental treatments in Model 1 of Table 2 and included our demographic covariates in Model 2.

The results indicate that expressions of overt racism increased within subjects following exposure to the deterministic genetic treatment (compared to the control treatment), but only in a racialized context that specifically indicated the prevalence of this gene in the African American population ($p < .05$, two-tailed, in Model 1 and Model 2). Figure 1 displays the marginal effects from Model 2 reported in Table 2. Moreover, in additional robustness checks (reported in Appendix B, Table 3), we collapse the treatments (race versus no race) into deterministic and conditional (versus control). We find that deterministic treatments in general significantly increase expressions of overt racism relative to the control condition ($p < .05$, two-tailed). In contrast, conditional treatments do not have a statistically significant effect on expressions of overt racism relative to the control condition ($p > .10$, two-tailed).

Furthermore, the results suggest that providing contextualized explanations for genetic associations—even in a racialized context—mitigates the heightened racist response. If referencing race alone was the primary driver of increased expressions of racism, then we would expect people receiving the conditional narratives in a racialized context (*Conditional—Race*) to also be significantly more likely to report racist attitudes than those who receive the control news article. This is not the case ($p > .10$, two-tailed), suggesting that although there may be some compounding effect of genes and race in the racialized deterministic narratives, conditional language about the effects of genes can disconnect the link between genetic attributions and racism.

Next, we further probe the mechanisms linking deterministic treatment of genetic effects to expressions of racism. We do so by examining the relationship between deterministic treatment and essentialist attitudes. Table 3 reports the results of a linear regression in which our dependent variable is essentialist attitudes (measured post-treatment) using the average response in the eight-item Bastian and Haslam (2006) essentialism scale. Individuals who received the deterministic treatments with and without explicit reference to race expressed more essentialist attitudes than those who received the control condition ($p < .01$, two-tailed). In addition, individuals who received the conditional treatment

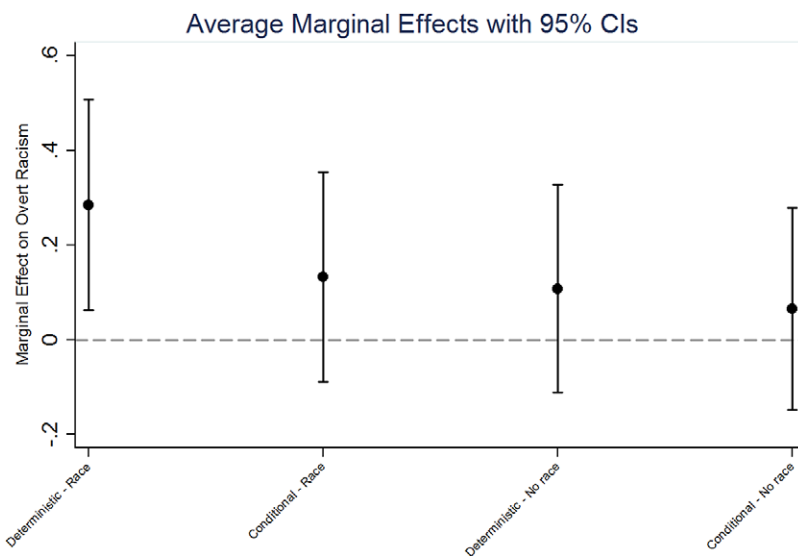


Figure 1. Overt racism: Marginal effects. Error bars represent 95% confidence intervals. (Study 2)

Table 3. Essentialist attitudes and genetic explanations for violence (Study 2)

Variables	(1)	(2)
	Essentialist attitudes	Essentialist attitudes
Deterministic—Race	0.338** (0.079)	0.342** (0.079)
Conditional—Race	0.140+ (0.077)	0.129+ (0.078)
Deterministic—No race	0.284** (0.080)	0.304** (0.081)
Conditional—No race	0.089 (0.075)	0.085 (0.075)
Age		0.005** (0.002)
Education		-0.006 (0.016)
Conservatism		-0.009 (0.015)
Female		0.012 (0.056)
Black [†]		-0.156+ (0.092)
Asian		-0.170+ (0.088)
Latinx		-0.052 (0.077)
Income		0.018 (0.019)
Constant	4.144** (0.054)	3.946** (0.142)
Observations	1,276	1,272
R ²	0.019	0.033

Note: Robust standard errors in parentheses.

[†]White is the reference category.

** $p < .01$; * $p < .05$; + $p < .1$ (two-tailed).

specifying racial allelic differences were somewhat more likely to report essentialist attitudes ($p < .10$, two-tailed), but not when race is not mentioned ($p > .10$, two-tailed). Put another way, it appears that it is only when genetic effects are contextualized in the *absence* of racial cues that essentialist beliefs remain unchanged.

Table 4. Simultaneous regression model—Overt racism and essentialist attitudes (Study 2)

Variables	(1)	(2)
	Overt racism	Essentialist attitudes
Deterministic—Race	0.291** (0.111)	0.332** (0.078)
Conditional—Race	0.133 (0.111)	0.125 (0.078)
Deterministic—No race	0.098 (0.112)	0.302** (0.079)
Conditional—No race	0.060 (0.111)	0.083 (0.078)
Age	0.002 (0.003)	0.005** (0.002)
Education	0.017 (0.022)	−0.007 (0.015)
Conservatism	−0.032 (0.020)	−0.010 (0.014)
Female	−0.005 (0.080)	0.015 (0.056)
Black [†]	−0.204+ (0.118)	−0.154+ (0.083)
Asian	−0.195 (0.166)	−0.166 (0.117)
Latinx	0.105 (0.126)	−0.050 (0.088)
Income	−0.003 (0.027)	0.017 (0.019)
Constant	−0.032 (0.208)	3.955** (0.146)
Observations	1,269	1,269
R^2	0.013	0.033

[†]White is the reference category

** $p < .01$; * $p < .05$; + $p < .1$ (two-tailed).

In Table 4, we model the potential correlation between expressions of overt racism and essentialist beliefs using a simultaneous regression model. By explicitly modeling this potential correlation, we should get more efficient estimators and are less likely to report insignificant associations (Lewis & Linzer, 2005). The simultaneous regression model results in Table 4 show that, indeed, the error terms

in the two models are correlated ($p < .01$, two-tailed, in Model 1 and Model 2), suggesting that there is a correlation between expressions of overt racism and essentialist beliefs. When taking this into account by statistically modeling the joint dependence, we see that there remain statistically significant effects of the deterministic race narrative on expressions of both overt racism and essentialist beliefs. However, there are no longer statistically significant effects of the conditional race treatment on essentialist attitudes ($p > .10$, two-tailed). Moreover, the conditional race treatment is not statistically significantly associated with expressions of overt racism ($p > .10$, two-tailed). Together, these results suggest that deterministic narratives may lead to an increase in the expression of overt racism *through* increased essentialist beliefs.

Discussion

With the current research, we sought to understand how portrayals of genetics research on health and behavioral outcomes can impact racial attitudes. Critically, we investigated the contrasting effects of media narratives that emphasize a deterministic, causal narrative of the role of genes versus a narrative intervention that provides a more conditional and complex explanation of the role of genes in phenotypic outcomes. Specifically, we examined the effects on prejudice and policy attitudes from deterministic and conditional genetic narratives in the domains of obesity and violence in media articles that implicated racialized minorities.

Across two studies, the experimental results suggest that media portrayals (and perhaps even academic, scientific portrayals) of genetics research explaining important outcomes like health and violence can impact the public's racial attitudes—and even their policy preferences in Study 1. Although simply reporting on genetics research on obesity (whether deterministic or conditional) in Study 1 appeared to negatively influence anti-Black bias, we also observe some evidence that narrative nuance matters. Hinshaw and Stier (2008) suggest that genetic mechanisms tend to be prioritized over other explanations (such as the environment; see also Schmalor et al., 2021), but our results suggest this does not have to be the case. Rather, in Study 2, our results indicate that despite increased anti-Black attitudes following reports of racialized genetics research, the pernicious attitudinal effects of such reporting can also be mitigated with explanations of polygenic, environmental, and personal choice factors. Thus, our research joins a chorus of scientists and academics in cautioning against carelessness in reporting or tendencies to sensationalize such genetics research in the media.

Highlighting genetic contributions to health and behavior may render racially minoritized group members particularly more vulnerable to prejudice. Although our focus in this research was to examine the effects of genetic explanations on racial attitudes toward Blacks, it remains to be seen whether prejudiced and punitive attitudes would also emerge toward other social or racial groups, including racial majority group members, when described in genetically deterministic ways. Although some prior research suggests that genetic attributions are associated with *increased* tolerance for disadvantaged and vulnerable groups (e.g., homosexuals, drug addicts, obese people; Schneider et al., 2018), it may be that such tolerant attitudes are mostly reserved for groups that are explicitly or implicitly perceived as prototypically White (even if they are low status in other ways). Schneider et al. (2018) also find that genetic attributions for *individual* characteristics tend not to be associated with negative racial attitudes, but there may be a distinction to be made between genetic attributions for individuals versus groups. For instance, Suhay and Jayaratne (2013) find that genetic attributions for racial group (as opposed to individual) differences in characteristics like intelligence and violence are associated with ideological beliefs that served to reinforce existing racial inequalities. Another open question for future research is how genetic attributions for *positive* outcomes or stereotypes may affect racial attitudes, in part because research on positive stereotypes largely suggests that such favorable group stereotypes are societally pervasive and influential in perpetuating systemic inequality (Czopp et al., 2015). We believe our studies take one step toward disentangling the intersectional effects of race and genetic attribution on attitudes, but there is much to explore in future work.

Together, our studies explore both modern and old-fashioned forms of anti-Black racism as a function of racialized genetic attributions, and our results suggest that genetic explanations have the potential to impact different types of racial attitude expressions. But, of course, this is merely a starting point for future investigation into the conditions under which such connections are made, as well as what kinds of ideological consequences may follow. For example, Banks and Valentino (2012) show that old-fashioned racism is associated with feelings of disgust, whereas modern racism is associated with feelings of anger or resentment. Investigation of such discrete emotions may provide clues to how genetic attributions can be associated with general negative racial out-group attitudes versus identification with explicitly racialized ideology, like the alt-right's White nationalism.

It will also be important in future work to further examine the political and policy implications of genetic attributions in intergroup contexts. Here, consistent with prior research that suggests genetic framing of ethnic out-group members impacts support for conflict policies (Kimmel et al., 2016), we show some preliminary evidence that the way genetic explanations are framed can impact pertinent policy attitudes. However, much remains to be explored, as we believe, like Huddy and Feldman (2009), that “the political power of racial prejudice remains an important issue” (p. 441). We hope to engage in further research that not only probes the effects of genetic attributions in different outcome domains on broader policy attitudes, but also on perceptions of and adherence to racialized ideology itself. If, for example, learning about genetic science associated with racial categories is an initial step toward endorsement of White nationalist ideologies, then identifying how such genetic justifications contribute to the formation of such ideological beliefs will be particularly important to understand.

There are also implications of this research for both education and media communication. Not only has the frequency of genetic science reporting in the news media increased significantly since the start of the Human Genome Project—including reports focused on highlighting racialized genetic differences (Phelan et al., 2013)—but genetic science forms the basis for the reemergence of discussions of race in high school biology textbooks in the 1990s (Morning, 2008). Whereas there were no discussions of race in the context of health disorders in textbooks from 1952 to 1962, discussions of racial differences in biology appear in 93% of contemporary textbooks (from 1993 to 2002; Morning, 2008). Indeed, disputes about “racial superiority” based on biological justifications are occurring in contemporary high school classrooms where individuals typically have their first contact with scientific education about genetics. In this way, supposedly neutral scientific information may be forming the kernel for racialized ideology in adolescents, without clear scientific guidance on whether such genetic justifications can be debunked (Harmon, 2018a). Moreover, increasingly accessible and popular direct-to-consumer genetic ancestry tests have also been shown to reinforce essentialist views on racial differences in the general population (Phelan et al., 2014), as well among White nationalists (Panofsky & Donovan, 2019). For these reasons, we suspect that the wide availability of information on genetic science—in the absence of careful interpretation—has the potential to strengthen and mobilize negative racial attitudes in the broader public.

Finally, and relatedly, an important future direction for this work will be to probe what type of language is most effective in mitigating the negative effects of linking genetics to health or behavior. Schneider et al. (2018) break down the types of attributions that people tend to make for human characteristics as genetic, environmental, and personal choice. We would like to explore in subsequent studies how different types of conditional language in genetic narratives might contribute to prejudice reduction—for instance, by highlighting polygenic, environmental, and personal choice factors separately. Indeed, the relationship between genetic attributions, behavioral outcomes, and social group characteristics make for complex perceptions of and attitudes toward those social groups. It will be important as this research moves forward to examine the ways in which genetic attributions can contribute to continued and exacerbated social inequalities—as well as the ways in which illuminating the role of genes in human behavior may lead to greater tolerance, respect, and societal equality.

Data availability statement. This article earned the Open Data, Open Materials, and Preregistered open science badges. The data, replication code and materials, and preregistration for this article are available at <https://osf.io/59kqg/>.

Author contributions. H. Hannah Nam and Katherine Sawyer contributed equally to this article.

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Appendix A: Study 1 Survey Items

Symbolic Racism Scale (Henry and Sears 2002). We recoded the following items in accordance with the recommendations by Henry and Sears (2002): items 1, 2, 4, and 8 were recoded so that a 1 = 4, 2 = 3, 3 = 2, and 4 = 1. Item 3 was recoded so that 1 = 3, 2 = 1, and 3 = 2.

Please read the statements below and chose the response most similar to your own:

_____ 1. It's really a matter of some people not trying hard enough; if blacks would only try harder they could be just as well off as whites.

- 1 Strongly agree
- 2 Somewhat agree
- 3 Somewhat disagree
- 4 Strongly disagree

_____ 2. Irish, Italian, Jewish and many other minorities overcame prejudice and worked their way up. Blacks should do the same.

- 1 Strongly agree
- 2 Somewhat agree
- 3 Somewhat disagree
- 4 Strongly disagree

_____ 3. Some say that black leaders have been trying to push too fast. Others feel that they haven't pushed fast enough. What do you think?

- 1 Trying to push very much too fast
- 2 Going too slowly
- 3 Moving at about the right speed

_____ 4. How much of the racial tension that exists in the United States today do you think blacks are responsible for creating?

- 1 All of it
- 2 Most
- 3 Some
- 4 Not much at all

_____ 5. How much discrimination against blacks do you feel there is in the United States today, limiting their chances to get ahead?

- 1 A lot
- 2 Some
- 3 Just a little
- 4 None at all

_____ 6. Generations of slavery and discrimination have created conditions that make it difficult for blacks to work their way out of the lower class.

- 1 Strongly agree
- 2 Somewhat agree
- 3 Somewhat disagree
- 4 Strongly disagree

_____ 7. Over the past few years, blacks have gotten less than they deserve.

- 1 Strongly agree
- 2 Somewhat agree
- 3 Somewhat disagree
- 4 Strongly disagree

_____ 8. Over the past few years, blacks have gotten more economically than they deserve.

- 1 Strongly agree
- 2 Somewhat agree
- 3 Somewhat disagree
- 4 Strongly disagree

Support of universal health care

Some people say that health care should be a right for all people and not a privilege only for those who are insured by their workplace or participate in some other private plan. Others say that the tax burden in this country is already high and it is unreasonable to expect people who are paying a part of their own private insurance plan to also pay for other people. How do you feel about universal, guaranteed health care?

- 7 Strongly support universal, guaranteed health care
- 6 Somewhat support universal, guaranteed health care
- 5 Slightly support universal, guaranteed health care
- 4 Neither support or oppose universal, guaranteed health care
- 3 Slightly oppose universal, guaranteed health care
- 2 Somewhat oppose universal, guaranteed health care
- 1 Strongly oppose universal, guaranteed health care

Health treatment—Deterministic

If You're Obese, a Single Gene May Be to Blame

One gene "could be fueling the obesity epidemic," according to a study performed on mice

By DANIELLE ZICKL, *MEN'S HEALTH*
November 16, 2017, 6:00 AM



We all know about the obesity epidemic here in America. In fact, more than 20 percent of people in every state have a body mass index (BMI) of 30 or above. And while fast food diets and lack of exercise have taken most of the blame, new research on mice suggests that a single gene could be at the root of why some people are overweight.

The study, published in the *Proceedings of the National Academy of Sciences*, showed that forms of a gene called ankyrin-B cause fat cells to absorb glucose at a much faster rate than they should, which makes them more than twice the size of normal fat cells.

"We call it fault-free obesity," Vann Bennett, M.D., Ph.D., senior author of the study and George Barth Geller Professor of Biochemistry at Duke University School of Medicine, said in a statement. "We believe this gene might have helped our ancestors store energy in times of famine. In current times, where food is plentiful, ankyrin-B variants could be fueling the obesity epidemic."

Researchers found that 1.3 percent of Caucasians and 8.4 percent of African Americans carry forms of ankyrin-B, which include millions of Americans.

"This gene could enable us to identify at-risk individuals who should watch what kind of calories they eat and exercise more in order to keep their body weight under control," Bennett said.

And the risks that come along with obesity, as we all know, are serious. High blood pressure, high cholesterol, diabetes, and heart disease (the leading cause of death for American adults) are all possible.

Health treatment—Conditional

If You're Obese, Genes in addition to Diet and Exercise May Be to Blame

By DANIELLE ZICKL, *MEN'S HEALTH*
November 16, 2017, 6:00 AM



We all know about the obesity epidemic here in America. In fact, more than 20 percent of people in every state have a body mass index (BMI) of 30 or above. And while fast food diets and lack of exercise have taken most of the blame, new research on mice suggests that genetics could also affect obesity rates.

The study, published in the *Proceedings of the National Academy of Sciences*, showed that forms of a gene called ankyrin-B cause fat cells to absorb glucose at a much faster rate than they should, which makes them more than twice the size of normal fat cells.

Researchers found that 1.3 percent of Caucasians and 8.4 percent of African Americans carry forms of ankyrin-B, which include millions of Americans.

The study was done on mice, so there's a lot more research on the gene that still needs to be done. Researchers will need to look into the family histories, physical characteristics, and metabolism of those with forms of the gene in order to truly figure out how it will affect people in addition to their exercise and diet habits.

"This gene could enable us to identify at-risk individuals who should watch what kind of calories they eat and exercise more in order to keep their body weight under control," Bennett said.

And the risks that come along with obesity, as we all know, are serious. High blood pressure, high cholesterol, diabetes, and heart disease (the leading cause of death for American adults) are all possible.

Control condition

The Washington Post

OPINION
AUGUST 10, 2011

Summer Homework

Seeing vacation homework from the perspectives of educator and parent.

By [DON JOHNSTONE](#)

Teachers assign it. Kids hate it. Parents hate it more. But what if the parent and the teacher are the same person? Welcome to my summer of confusion.

For five years, I was an elementary school principal. I loved my job. Each June, I sent parents a heartfelt letter describing the learning lost when children don't read or do math over the summer. The teachers I worked with assembled math sheets, curated summer book lists and crafted assignments to keep students on track with their learning. Boy, did I love a good summer homework packet back then.

But now, I've opted out, temporarily stepping away from work to parent my two daughters, now in the fourth and seventh grades. So now I'm just the one policing its completion, which drastically decreases my motivation. Yes, *my* motivation. For, as teachers may not understand, but most parents know, it is the rare child who completes summer homework without serious adult intervention.

I made a chart for the kids to document their progress. This, of course, is not working. We watched the endless summer dwindle into a few weeks of August. A few weeks ago, at dinner, my younger daughter worried aloud, almost to the point of tears: "I have too much summer homework! I'll never finish." My older daughter averred that she too might be "oh, just a little behind."

Having stepped out of my old professional role, and into this wholly parental one, my focus has shrunk to two children, and my priorities for those two girls' learning have widened. During the summer, they will play harder, travel farther, sing louder and climb higher than they do the rest of the year. They will paddle canoes, take photographs, and write stories. And so they may end up doing what most kids do: spending the last days before school begins filling in empty packets and notebooks. Things could get ugly at our house around Aug. 29. But those notebooks will be complete on the first day of school.

As a former principal, I can say for certain that a three-day sprint in late August is not at all what the teachers had in mind. In my old role, I knew I was right to assign summer homework. But now, I'm equally sure that there are even better ways for my own girls to learn and grow in the summertime.

Mr. Johnstone is parent and former elementary school principal from Wisconsin.

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Research debriefing form

Thank you very much for participating in our study!

In this study, we are interested in examining whether media portrayals of genetics research has any impact on people's social and political attitudes. You may have read an excerpt of a article that described the genetic basis for some behavior or health outcome, or you may have read an article unrelated to genetics at all. The article excerpt presented in the study was an adaptation of real articles in the media but were edited to emphasize various aspects of the role of genetics and the environment. We expect that when genetic explanations for behavior are provided that may lead to differences in people's attitudes toward the described groups. We apologize for not explaining the study's primary intent at the outset; if participants had been alerted to the purpose, responding could have been biased by this knowledge. Your participation was helpful in increasing our understanding of how media portrayals of genetics research may affect social attitudes.

Thank you again for your valuable participation!

Appendix A. Study 1: Empirical analyses

Appendix A. Table 1. Distribution of health treatments across survey respondents

	Frequency	Percent	Cumulative percent
Health—Conditional	51	33.77%	33.77%
Health—Deterministic	50	33.11%	66.89%
Control	50	33.11%	100.00%
Total	151	100.00%	100.00%

Appendix A. Table 2. Descriptive statistics

Variable	Sample Size	M	SD	Min.	Max.
Asian	151	0.298	0.459	0	1
Black	151	0.093	0.291	0	1
Latinx	151	0.126	0.333	0	1
White	151	0.430	0.497	0	1
Conservatism	147	3.442	1.531	1	7
Single	146	0.932	0.253	0	1
Age	145	21.400	2.509	19	38
Female	147	0.463	0.500	0	1
Education	147	6.687	1.313	2	8
Income	131	56.817	36.06	1	120

Appendix B: Study 2 Survey Items

Overt Racism (Feldman and Huddy 2010). Reverse-code items 1, 2.

On average, African Americans have lower income and worse housing than white people. How much of the economic difference between blacks and whites:

Occurs because most Blacks do not have the chance to get a good education?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Can be explained by discrimination against blacks?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Occurs because most blacks just don't have the motivation or will power to perform well?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Occurs because most blacks do not teach their children the values and skills which are required to be successful in school?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Is due to racial differences in intelligence?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Occurs because of fundamental genetic differences between the races?

- None (1)
- A little (2)
- Don't know (3)
- Some (4)
- A great deal (5)

Essentialism Scale (Bastian and Haslam 2006). Reverse-code items 2,3,6,8.

Please indicate the degree to which you agree with the following statements from 1 (strongly disagree) to 7 (strongly agree):

The kind of person someone is can be largely attributed to their genetic inheritance.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

Very few traits that people exhibit can be traced back to their biology.

- Strongly disagree (1)
- Disagree (2)

- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

I think that genetic predispositions have little influence on the kind of person someone is.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

Whether someone is one kind of person or another is determined by their biological makeup.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

There are different types of people and with enough scientific knowledge these different 'types' can be traced back to genetic causes.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

A person's attributes are something that can't be attributed to their biology.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

With enough scientific knowledge, the basic qualities that a person has could be traced back to, and explained by, their biological makeup.

- Strongly disagree (1)
- Disagree (2)

- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

A person's traits are never determined by their genes.

- Strongly disagree (1)
- Disagree (2)
- Somewhat disagree (3)
- Neither agree nor disagree (4)
- Somewhat agree (5)
- Agree (6)
- Strongly agree (7)

Violence treatment—Deterministic, race

“Warrior gene” may contribute to violent crime, studies say

By Jane Roberts

July 19, 2018

Whether criminals are born with an innate tendency to hurt others, are prone to mental disorders, or are molded by factors such as childhood trauma, a history of abuse or too many violent video games is a persistent and complicated question.

Now, new research suggests that genetics may in fact contribute to a propensity for violent criminal behavior.

In a study published Tuesday in the journal *Molecular Psychiatry*, researchers examined the genes of 895 people found guilty of crimes ranging from non-violent offenses such as drug or property crimes to severely violent offenses such as homicide and battery. They found that a variant of the gene—called MAOA and dubbed the “warrior gene”—was linked to “extremely violent behavior,” defined as having committed at least 10 homicides, attempted homicides or batteries. The MAOA gene plays a role in the metabolism of the neurotransmitter of dopamine that helps regulate emotions and reactions to pleasure and rewards.

A strong relationship between behavior and the “warrior gene” was not present among non-violent offenders. Even when the researchers accounted for factors such as personality disorders, childhood maltreatment or substance abuse, the effects were still specific to violent offenders.

The relationship between genetics and violent behavior was strongest for the 78 people in the study who were classified as “extremely violent offenders.” The people in this group committed a total of 1154 murders, manslaughters, attempted homicides and batteries.

Other researchers have also reported that this variant of the MAOA gene was less common among Caucasians (34 percent) and more common among African Americans (59 percent). Such studies further suggest that the prevalence of the “warrior gene” is quite variable across the population.

Violence treatment—Conditional, race

“Warrior gene” may interact with environment to contribute to violent crime, studies say

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Revealing the true complexity of the issue, in another study, published in 2012 in the journal *Science*, investigators found a relationship between convictions of violent crimes and a combination of low-activity MAOA plus childhood maltreatment. That is, the low-activity MAOA gene was only associated with violent crime convictions when the individuals had also experienced a history of adversity and maltreatment.

Other researchers have also reported that this variant of the MAOA gene was less common among Caucasians (34 percent) and more common among African Americans (59 percent). Such studies further suggest that the prevalence of the “warrior gene” is quite variable across the population.

In an interview with the BBC, Jan Schnupp, a neuroscientist at the University of Oxford called for caution in interpreting such studies, saying that up to half the population—most of whom do not commit violent crimes—could have one of the genes that the studies linked to violent behavior.

“To call these alleles ‘genes for violence’ would therefore be a massive exaggeration,” Schnupp said. “In combination with many other factors these genes may make it a little harder for you to control violent urges, but they most emphatically do not predetermine you for a life of crime.”

Violence treatment—Deterministic, no race

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Violence control

Traumatic events may contribute to violent crime, studies say

By Jane Roberts

July 19, 2018

Whether criminals are born with an innate tendency to hurt others, are prone to mental disorders, or are molded by factors such as childhood trauma, a history of abuse or too many violent video games is a persistent and complicated question.

Now, new research suggests that experiencing traumatic events may in fact contribute to a propensity for violent criminal behavior.

In a study published Tuesday in the journal *Sociological Inquiry*, researchers examined 895 people found guilty of crimes ranging from non-violent offenses such as drug or property crimes to severely violent offenses such as homicide and battery. They found that prior life experience with trauma—described as “an event or set of circumstances that is experienced by an individual as physically or emotionally harmful or life threatening and that has lasting adverse effects on the individual’s functioning and well-being”—was linked to “extremely violent behavior,” defined as having committed at least 10 homicides, attempted homicides or batteries.

Trauma is a common experience for individuals in American communities and has no boundaries with regard to age, gender, socioeconomic status, race, ethnicity, or sexual orientation. Research has shown that traumatic experiences are associated with both behavioral and physical conditions, especially those traumatic events that occur during childhood. Because these events can have a negative impact not

only on the affected individual but also harm to the larger community, researchers note it is of great importance to understand the nature and impact of trauma, as well as avenues for healing trauma.

Research debriefing form

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Thank you again for your valuable participation!

Appendix B: Study 2 Empirical Analyses

Appendix B. Table 1. Distribution of violence treatments across survey respondents

	Frequency	Percent	Cumulative percent
Deterministic—Race	257	20.14%	20.14%
Conditional—Race	256	20.06%	40.20%
Deterministic—No race	249	19.51%	59.72%
Conditional—No race	259	20.30%	80.02%
Control	255	19.98%	100.00%
Total	1,276	100.00%	100.00%

Appendix B. Table 2. Descriptive statistics

Variable	Sample Size	<i>M</i>	<i>SD</i>	Min.	Max.
Asian	1276	0.049	0.215	0	1
Black	1276	0.103	0.305	0	1
Latinx	1276	0.088	0.283	0	1
White	1276	0.740	0.439	0	1
Conservatism	1276	3.966	1.795	1	7
Age	1273	50.472	15.680	19	92
Female	1276	0.527	0.499	0	1
Education	1276	6.943	1.776	2	9
Income	1275	3.042	1.446	1	6

Appendix B. Table 3. Overt racism and genetic explanations for violence, collapsed

Variables	(1)	(2)
	Overt racism	Overt racism
	0.198*	0.197*
Deterministic	(0.098)	(0.098)
	0.099	0.096
Conditional	(0.097)	(0.097)
		0.002
Age		(0.003)
		0.015
Education		(0.024)
		-0.033+
Conservatism		(0.020)
		-0.002
Female		(0.081)
		-0.201
Black [†]		(0.126)
		-0.208
Asian		(0.187)
		0.102
Latinx		(0.134)
		-0.001
Income		(0.028)
	0.004	-0.025
Constant	(0.080)	(0.235)
Observations	1,273	1,269
R ²	0.003	0.010

Note: Robust standard errors in parentheses.

[†]White is the reference category.

***p* < .01; **p* < .05; +*p* < .1 (two-tailed).

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