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RESEARCH ARTICLE

REVISED How messages about behavioral genetics research can impact on genetic attribution beliefs [version 2; peer review: 3 approved with reservations, 1 not approved]

Previously titled: Communicating behavioral genetics: Charting the limits of the genetic interpolation effect

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Abstract

Science communication has the potential to reshape public understanding of science. Yet, some research findings are more difficult to explain and more likely to be misunderstood. The contribution of this paper is threefold. It opens with a review of fascinating interdisciplinary literature on how scientific research about human genetics is disseminated in the media, and how this type of information could influence public beliefs and world views. It then presents the theoretical framework for my research program, providing a logical basis for how messages about human genetics may influence people's beliefs about the role of genes in causing human traits. Based on this reasoning, I formulate the genetic interpolation hypothesis, which predicts that messages about specific research findings in behavioral genetics can lead members of the public to infer greater genetic causation for other social traits not mentioned in the content of the message. While this framework offers clear, testable predictions, some questions remain unaddressed. For instance, what kind of message formats are persuasive enough to alter people's views? The third contribution of this paper is to begin to address this question empirically. I present the results of a survey experiment that was designed to test whether a simple, short paragraph about behavioral genetics is a powerful enough stimulus to cause the genetic interpolation effect.

Keywords

science communication, public understanding of science, behavior genetics, genetic attribution, biased assimilation, genetic interpolation effect, media effects, survey experiment

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REVISED Amendments from Version 1

I am grateful to both reviewers for presenting me with their views on my paper and with propositions on how to improve it. As KK and BMD will find out, answering their queries required a major revision of the paper. Critics are addressed at different places in the paper. A new literature review illustrates clearly the place of this contribution in the current debates and the direct links with my previous contribution (KK). The paper carries on and elaborates a new theoretical framework from which clear hypotheses are derived, providing more information about the anchoring and adjustment heuristic and the schema theory (KK), thus clarifying how this theoretical framework sets people's general prior belief structure as an assumption (BMD). **Figure 1** was added to help readers understand the expected "chain reaction". This section also eliminates the confusion regarding word choice (interpolation vs extrapolation) and whether some hypotheses are conditional on others (KK). The empirical section now justifies the relevance of studying the impact of a short paragraph on behavioral genetics (BMD), a clarification that later offers guidance for interpreting the null-finding (BMD). The empirical section also addresses other problems raised by reviewers: the number of participants and their spread across the experimental groups (KK and BMD); specifying which tests are confirmatory or exploratory and, accordingly, using the appropriate statistical method (BMD); presenting additional statistics to compute effect sizes (BMD); secondary analyses further exploring the distribution of response patterns in the control group (BMD). Finally, the discussion section highlights the limitations of the current work, such as the inability to control for previous beliefs (BMD) and presents a new paragraph speculating on the reasons that might account for the unexpected finding (BMD). Finally, the discussion reemphasizes the importance of this contribution (KK and BMD) and lists directions for future research.

See referee reports

Introduction

How do laypeople—as opposed to experts—understand human genetics? This subject has been approached from a diversity of angles. For example, one stream of research focuses on clinical contexts to assess public understanding in situations where, for example, patients are informed about their own genetic predispositions or the predispositions of their relatives¹⁻³. Another stream of research examines how attributing certain conditions such as schizophrenia, obesity or sexual orientation to genetic influence correlates with or impacts on stereotyping attitudes⁴⁻⁸. Moreover, a group of studies tests how different types of claims about the existence or non-existence of significant genetic differences between genders or ethnic groups impact on individual perceptions and intergroup relations⁹⁻¹¹.

My research contributes to closely-related literature on how people develop, reinforce or update their beliefs about the influence of genetics on various human traits. What is it that makes people think that some traits are more or less affected by genetic predispositions? The existing literature points to different sources of influence. Research in psychology indicates that basic intuition about the influence of biology on physical and social traits is rooted in childhood^{12,13}. Some research also verifies whether participation in an introductory biology class or

social science programs influences students' views on the role of genetics¹⁴⁻¹⁶. The present study speaks to a third branch of research that investigates the roles played by the news media.

The contribution of this paper is threefold. It begins with a review of fascinating literature on how human genetics is disseminated in the media and how this type of information could influence beliefs and world views. This literature is produced by intellectuals and social scientists from a variety of disciplines such as sociology, ethics, psychology, and communication studies. My review highlights the milestones in the evolution of this branch of research. With this map in hand, I then present the theoretical framework for my research program, providing a logical basis for how messages about human genetics may influence people's beliefs about the role of genes in causing human traits. The central argument is that people's reactions vary depending on whether the presented information is from medical/physical genetics or behavioral genetics research findings. The main hypothesis that can be derived from this framework is the genetic interpolation hypothesis, which predicts that messages about how genes impact on specific behavior can lead the public to infer greater genetic causation for social traits that are not even mentioned in the content of the message.

The main strength of my framework is that it offers clear, testable predictions. However, it is not without limitations and some questions remain unanswered. One of these questions is: what kind of message formats can succeed at altering people's views about the influence of genes on human beings? The third contribution of this paper is to begin to address this question empirically. I present the results of a survey experiment that was designed to test whether a simple, short paragraph about behavioral genetics is a powerful enough stimuli to cause the genetic interpolation effect.

Genetics in news media: A literature review

During the '90s and early 2000s, an important group of intellectuals expressed concern about how genetics research is communicated to the public¹⁷⁻²¹. The blame was divided between three culprits: overenthusiastic scientists, fantasizing pop culture, and misreporting news media. The news media were also charged for reporting inflated optimism about genetics research, over-emphasizing benefits and ignoring or downplaying risks. But the media was mainly criticized for how they described the influence of genetics as deterministic, that is, as if genetics were the only, or at least, the most important factor at play. It was argued that, while this depiction might be accurate for a few distinct physical traits or rare mono-genetic diseases, such a deterministic outlook is largely misleading for describing the influence of genetics on most human traits, especially social traits like sexual orientation, intelligence, personality traits, or other complex social behaviors.

Among academics, this critique found both supporters and skeptics. The debate was partly fueled by the fact that most of the evidence gathered to support these claims rested on anecdotes and case studies. A number of social scientists decided to contribute to this debate by systematically analyzing the

contents of media coverage. In a paper published in 1998, Condit, Ofulue and Sheedy²² test whether, as argued by critics, the advent of molecular genetics is associated with an increase in genetic determinism in news coverage. Their results refuted expectations, indicating that there had been a small decrease in deterministic framing during the 1985–1995 decade compared to previous periods. However, the study also reveals that in approximately a third of news article, causation is attributed solely to genes. Regarding this finding, the authors preferred not to adopt a normative stance: “Whether this is an appropriate balance or whether it instead represents excessive determinism will depend on one’s own view of the role of genes.” (p.981)

This quote emphasizes the intrinsic difficulty of evaluating the quality of news coverage: that of choosing which standards to use. Many other studies have assessed how genetics research is covered in the media. Often, the focus of these analyses varies according to the type of research presented in the news: medical genetics^{23–25}, psychiatric genetics²⁶, genetics of addiction^{27,28}, or behavioral genetics²⁹. In line with critiques³⁰, claims, optimism is found to be prevalent, and the language used to describe the effect of genes on human traits often suggests that the role of genetics is predominant. (For an interesting exception, see 24.) However, each of these works sets its own standard for evaluating whether a claim is too optimistic or too deterministic. The challenge is to find a discriminant benchmark that can be replicated.

In their article published in 2004, Bubela and Caulfield³⁰ present an innovative approach. Their clever study design consists in gathering 627 news stories from 26 newspapers published in 4 countries. Their work compares each of these news stories with the content of the original research article on which it is based. This comparison allows the authors to assess the prevalence of exaggerated claims, and to explore which factors are associated with these claims. The authors summarize their results in a few words:

[...] most newspaper articles had no exaggerated claims (63%) or only slightly exaggerated claims (26%). However, the media do seem to over-emphasize particular topics, such as behavioral genetics. The high profile of these types of stories may be one reason for the perception that newspaper stories are often hyped. In other words, although we found that only 11% of the newspaper articles had moderately or highly exaggerated claims, these few stories might have a significant impact on public perceptions³⁰.

The literature studying how genetics is presented in the media is far too broad for a fair review [see also 31,32]. But the last sentence from Bubela and Caulfield’s quote points us to the second and key component of this debate: whether or not media coverage of genetics research has a significant impact on public beliefs. Indeed, from a professional perspective, science journalists’ tendency to inflate the influence of genes is a questionable practice. However, this exaggeration would be of

less concern if it was found that public beliefs remained largely unaffected by the message.

This leads us to a closer review of the empirical literature on media effects, to which the present paper intends to contribute. The predominant approach has thus far consisted in testing specific hypotheses using psychology experiments where different groups of participants are randomly exposed to one of several messages about the influence of genes before being asked to report their beliefs about genetics. Provided a rigorous implementation and sufficient statistical power, this research design allows researchers to be confident that the differences that may emerge between the experimental groups following message exposure result from the influence of these messages on participants’ beliefs^{33,34}.

Most of the empirical work produced thus far tests whether messages about *medical genetics* have an impact on public beliefs about the influence of genes on human traits. The evidence gathered in the literature indicates that this type of information has limited influence. In their 2001 study involving approximately 100 college students, Condit and colleagues test whether using or not using a deterministic headline influences participants’ reading of a 500-word, non-deterministic news article about the influence of genetics on diabetes³⁵. Following message exposure, participants were asked: “How significant a role do you believe that genes play in human health generally?” The study finds no significant post-exposure difference between the groups. In their 2008 paper, Lynch and colleagues describe a study where they recruited a similar sample of participants (n=104) and conducted a quasiexperiment to test whether repeated exposure to news headlines, news summaries and video advertising about medical genetics can, among other things, increase genetic determinism as measured using a 7-item battery of questions³⁶. The results show no statistically significant increase.

These two studies converge in suggesting that participants do not mechanically endorse genetic determinism in response to messages about medical genetics. Still, it would be premature to conclude that the public remains unaffected by this type of message. Indeed, these experiments included only a small number of participants, thus offering low statistical power. Additionally, the outcome measures were designed to test if message exposure causes an overall change in views about the influence of genetics. One might expect that these two studies would fail to detect more nuanced or subtle effects.

Lynch and colleagues conclude their paper by recognizing that people likely “assign different levels of [genetic] causation to different characteristics” (p.51) and therefore, they suggest that future research should measure perceptions of genetic influence with regard to specific traits. Smerecnik’s paper, published in 2010, follows this recommendation³⁷. In the literature, the term “genetic attribution” refers to the level of importance people attribute to genetics in explaining a particular trait or group difference³⁸. Smerecnik’s study tests if messages about health genetics impact on genetic attribution. The 235 students and members

of the public who participated in the experiment were randomly exposed to one of two short—approximately 140-word—health messages. One was a paragraph about how salt intake can impact blood pressure; the other was the same paragraph complemented with an indication about a gene-environment interaction: “People without a genetic predisposition to salt-sensitive blood pressure may consume salt as usual.” Following message exposure, participants were asked to use a 7-point scale to report how much they believe genes impact on each of 11 health conditions (e.g., obesity, lung cancer, hypertension). Results reveal that the group exposed to the GxE interaction version of the stimuli showed a statistically significant increase in the influence they attributed to genetics in causing salt sensitivity, while the other group did not. But more crucially, participants “did not extrapolate these implications to other diseases.” (p.390)

This experiment offers more than a conceptual replication of previous studies. Indeed, the results reinforce the conclusion that messages about medical genetics do not cause genetic determinism. But more importantly, Smerecnik’s research design also offers a novel, quantifiable benchmark to evaluate one potential type of interpretation bias: the risk that a message about the role of genetics in causing one characteristic could lead the audience to infer greater genetic causation for other traits that are not the object of the message. His work inspired me to apply the same benchmark in order to address a related, much discussed, but until then unexplored research question: How do members of the public react when exposed to news about *behavioral genetics*?

In my study, published in 2014³⁹, I divided a large convenience sample of approximately 1400 respondents into three groups. The control group, used as a baseline for comparison, was exposed to a news article about medical genetics, more precisely about the recent discovery of a key gene involved in the development of breast cancer. The second group was exposed to a news article claiming that scientists have found a gene that causes liberal ideology. The third group was presented with a news story claiming that credit card debt is linked to a particular gene. All three stimuli were real news articles published in well-known newspapers or magazines from the United States, Canada, and the United Kingdom. Immediately following news exposure, participants were asked to report how much influence they attribute to genetics in explaining 14 body, medical, psychological and behavioral traits on an 11-point scale ranging from 0% to 100%.

Responses from the control group reveal that the average genetic attribution for political ideology and credit card debt is low, 10.6% and 7.2% respectively. Comparison with the two other groups shows statistically significant differences in genetic attribution for the specific trait described in the news article. For those exposed to the story about a liberal gene, genetic attribution for ideology doubled to 21.5%; for those exposed to the story about the debt gene, genetic attribution for credit card debt showed a threefold increase, 24.2%.

Therefore, as was the case for salt sensitivity in Smerecnik’s experiment, results show that participants tend to accept the

specific genetic argument presented to them, and to adjust their genetic attributions accordingly. However, in contrast to the salt experiment, my study reveals the presence of a side effect. Groups exposed to behavioral genetics report higher genetic attribution for other traits not mentioned in the new articles. The size of these increases in genetic attribution may seem relatively small, ranging from approximately 3 to 8 percentage points on the 0% to 100% response scale. But what is more striking is that traces of this side effect were found in genetic attribution for most, if not every other social trait: intelligence, obesity, gambling addiction, violence, alcoholism, mathematical ability, and sexual orientation. Interestingly, exposure to news about behavioral genetics has no significant additional effect on genetic attribution for height, skin color and skin cancer.

This study offers support for some of the concerns expressed by intellectuals during the ’90s and early 2000s: news about human genetics can impact on people’s beliefs. Yet, this media effect is much more subtle than anticipated. Firstly, interpreting my research in light of the literature suggests that news articles impact views about the influence of genetics when they cover research on behavioral genetics, but not when they cover research on medical genetics. And secondly, this impact is not an instant endorsement of genetic determinism, but a marginal upward adjustment in genetic attribution for social traits.

While my 2014 paper offers an important empirical contribution, the issue of why people react the way they do was only superficially addressed, due to space limitations. The next section presents a theoretical framework for cognitive mechanisms that could account for why people generalize particular findings from behavioral genetics to other social traits.

The genetic interpolation hypothesis: A theoretical framework

Psychological essentialism can be observed when people tend to associate the characteristics of an individual, group or phenomenon to its “deep-seated hidden essence” [40, p.4]. There exist different types of essentialism. For example, in many religions or sects, prophets are believed to be the embodied manifestation of divine intervention. This belief is a manifestation of spiritual essentialism. Another example is nationalist essentialism, where a particular nation is, in its history, ethnicity and culture, seen as fundamentally unique and therefore different from other nations.

The present framework is concerned with a particular type of essentialism. Genetic essentialism is a world view that holds a particular set of assumptions about the origins and characteristics of human traits⁴¹. In its most drastic form, often referred to as genetic determinism, this world view perceives most of the features that define human beings and their interactions as innate, shared by family members, immutable, predetermined and therefore independent of one’s own will. While very few people adhere to this extreme view, research shows that there is substantial disagreement among members of the public regarding the extent of genes’ impact on humankind⁴².

From this perspective, genetic essentialism should be perceived as a continuous dimension bounded by a bottom limit – genes have no influence whatsoever – and a top limit – genes explain everything – with most people positioning themselves somewhere in between. Research also indicates that genetic essentialism may coexist with some world views or ideologies (e.g., creationism) and conflict with others (e.g., constructionism)⁴².

While the social implications of genetic essentialism have been the object of much scholarly attention^{43–45}, relatively little is known about the reasons why some people come to endorse or reject this world view, partially or completely. Of course, some dimensions of genetic essentialism refer to properties that are partly observable in the real world (e.g., whether a trait is detectable in infancy, shared among relatives, and stable over time). It seems plausible that people use these features as proxies to assess the influence of biology and genetics¹². Members of the public may also form their beliefs by relying on knowledge acquired in college as part of the biology or social science curriculum¹⁶. The theoretical framework described in the paragraphs below leads me to predict that another aspect of their environment, namely media coverage of behavioral genetics, can also affect people's beliefs about the influence of genetics on human beings.

Defining whether and to what extent human traits are caused by genetics requires rigorous scientific investigation, and involves significant expertise and resources. Very few people will ever have the opportunity to empirically measure the real influence of genes. Still, this limitation does not prevent members of the public from forming their own beliefs and impressions on the matter. In many studies, participants were asked to estimate the extent of the impact of genetics on various human traits^{39,46–48}. A consistent pattern emerged. People tend to consider genetics to be a dominant influence on lasting and stable traits like physical characteristics, chronic diseases or persistent health conditions. Genes are perceived to play a weaker but still significant role in explaining various psychological traits like mental abilities and personality traits. Participants also believe genetics play a moderate or weak role in explaining impulsive or addictive behavior such as violence or gambling addiction. Finally, genetics attribution is very small or null for complex social preferences or behavior such as voter turnout or brand preferences.

To interpret this relationship, I mobilize the notion of *schema* used in cognitive and educational psychology. This notion was initially developed in the first half of the 20th century^{49,50} and later stimulated major theoretical innovations^{51,52}. However, the literature it inspired has been criticized for its contradictions, ambiguity and overstatements. It is therefore crucial to clarify the meaning I intend to invoke.

Schemata—plural for schema—are a form of knowledge that involves the organization of related mental objects into a coherent structure. The logical structure underlying this organization may take various forms, such as categories, geographical locations, hierarchy, chronology or associations,

to name a few. The logic of a schema offers a representation that connects mental objects together and helps to make sense of these objects. One example of a schema is a timeline representing various events organized logically in chronological order of occurrence. This information can be presented visually using a material or numeric support. But people may also store the chronological order that connects events in their memory. In this way, information about the chronology of events (mental objects) is organized in their mind and remains accessible even when the visual representation is not available. This coherent structure of mental objects is an example of a schema. We use schemata in everyday life. It has been argued that organizing mental objects into schemata facilitates the encoding of information in memory during learning, and eases memory recollection afterwards⁵³.

The schema theory posits that preexisting schemata are consciously or unconsciously activated when people try to interpret new pieces of information. Rumelhart compares the role of schemata in everyday life with the role of theories in science⁵²:

Theories, once they are moderately successful, become a source of predictions about unobserved events. Not all experiments are carried out. Not all possible observations are made. Instead, we use our theories to make inferences with some confidence about these unobserved events. So it is with schemata. We need not observe all aspects of a situation before we are willing to assume that some particular configuration of schemata offers a satisfactory account for that situation.(p.38)

People's assessments of genetic influence are not precise estimates but informed guesses. In my view, the concept of schema is reasonably well-suited to the genetic attribution pattern described earlier. The structure of this schema is organized such that *the more a trait is believed to be influenced by biology—as deduced from experience, observable features or as learned from other sources—the more genetics plays a predominant role*. I use the term *genetic attribution schema* to refer to this belief structure. I further argue that people are more confident about their guesses when it comes to assessing the influence of genetics on traits that are either very close to biology or very far from it. This assumption is partly derived from the fact that one of the features of genetic attribution is that it is constrained by a ceiling value (completely genetic) and a floor value (not genetic at all). **Figure 1**, Panel A, visually illustrates the genetic attribution schema.

What happens if a person comes across information about a case or event that presents characteristics that are inconsistent with the schema in which it should normally fit? The exact answer varies quite a bit from one person to another, and it also varies depending on the particular schema at stake. Rumelhart discusses three broad possible categories of reactions⁵². In a first scenario, the individual interprets this new information as an exception, a rare case that deviates from the general rule. Here, incoherent information is discarded and original schema remains intact. In a second scenario, the

Figure 1. Visual representation of the theoretical framework.

individual recognizes the new information as true or at least plausible, and adapts the original schema at its margins. This amended schema offers a way to interpret the new information without compromising the schema's fundamental logic. In a third scenario, the original schema is discredited. The individual either finds an alternative schema compatible with the new information, or accepts to live without a coherent schema to make sense of the phenomenon at stake.

I argue that some of the findings derived from behavioral genetics research and disseminated in the news media are inconsistent with the genetic attribution schema. This is especially the case when research suggests that genes play a non-negligible role in causing complex social behavior such as credit card debt, generosity, friendship or voting preference^{54–56}. Indeed, the genetic attribution schema posits that genetics has very little to no influence on these kinds of characteristics. Some people likely consider these findings to be exceptions to the rule, or may simply discredit the information presented to them. However, provided a credible source of information, such as a scientific study or a respected news outlet, other people will accept the scientific argument presented to them. Consequently, they will try to adapt their genetic attribution schema so that the new piece of evidence can fit into it. One way to do so is to raise the floor level of genetic attribution and to conclude that genetics has at least a small but non-null influence on almost every human trait. This change at the margin allows the new information to fit into the schema without compromising its basic structure: the negative association between genetic attribution and perceived distance from biology.

From this perspective, one can anticipate four consequences at the aggregate level. First, exposure to a specific finding from behavioral genetics will cause an increase in average genetic attribution for the particular complex social trait on which the message is focused. I refer to this as *the persuasion hypothesis*, since this effect reflects an acceptance of the specific claim presented in the message (see Panel B from [Figure 1](#)).

Two other hypotheses can be derived by mobilizing evidence from well-established works in the field of cognitive psychology. These works investigate the strategies and shortcuts humans beings employ in everyday life when they have to make guesses in situations of imperfect information^{57,58}. As Epley and Gilovich write, “one way to make judgments under uncertainty is to anchor on information that comes to mind and adjust until a plausible estimate is reached.” [59, p. 311] The anchoring and adjustment heuristic is commonly used in tasks that require guessing the characteristics of an element. It consists in searching in memory or nearby environment for other elements that share similarities with the element at the center of the task, and to use them as anchors for comparison. The individual then proceeds by estimating how and to what extent the element under study differs from or is comparable to the anchor elements, and adjusts their final estimate accordingly.

For example, a question in a history exam may ask what year the Geneva Conventions were ratified. A student may recall that these international treaties were written

to define the basic rights of wartime prisoners in response to abuses witnessed during the Second World War. The student may infer that the convention was signed after the end of the war (anchoring), following lengthy diplomatic negotiations (adjustment), and write 1951 as a best guess (actual answer: 1949). The broad literature on anchoring and adjustment heuristics has shown that people use this shortcut in a variety of everyday situations⁶⁰, but it can also be deployed when making guesses about scientific phenomena (e.g., estimating the freezing point of vodka⁶¹).

The task of estimating the influence of genetics involves a great degree of uncertainty. In this context, people will use the anchoring adjustment heuristic to reduce their efforts. I argue that the information presented in conventional news stories about how genes influence a particular complex social behavior provides people with an anchor information. This anchor indicates that the influence of genetics on this complex behavior is not null, and in fact is significant enough for scientists to investigate it. This anchor can later be used when estimating the influence of genetics on other complex social traits that are perceived as being at a similar distance from biology as the particular social trait covered in the message. Accordingly, exposure to the news story will cause an increase in average genetic attribution for other complex social traits. I call this second prediction *the similitude hypothesis* (see Panel C in [Figure 1](#)).

Finally, the anchoring and adjustment heuristic can also be used when estimating genetic attribution for traits that are seen as moderately distant from biology, such as mental abilities, sexual orientation, or vulnerability to addiction. As [Figure 1](#), Panel A illustrates, I argue that people are even more ambivalent about the extent to which genetics impact on these traits than they are for complex social traits, on one hand, or medical and physical traits, on the other. When estimating the influence of genetics on traits that are moderately distant from biology, people thus think of traits about which they are more confident. They then use these traits as anchor points, and adjust their estimate depending on how close to or far from biology they believe the trait is.

Yet, suppose that some of these people are exposed to information about behavioral genetics research and, as a result, now believe that genetics does have a non-negligible impact on complex social traits. Implementing this calibration at the margin of the genetic attribution schema implies increasing genetic attribution values used as anchor points for estimating the influence of genetics on other traits. As illustrated in [Figure 1](#), Panel D, this will cause people to increase their genetic attribution for social traits that are moderately distant from biology. I refer to this collateral impact as *the genetic interpolation hypothesis*, in reference to the mathematical operation which consists in estimating unknown mid-range values that are located between known values.

To summarize, some people react to news about behavioral genetics by thinking that “if genetics is strong enough to have a significant influence on complex social trait X or Y presented in the news story, then the general role of genes in causing other

social traits must be stronger than I had originally imagined". This reasoning leads them to infer greater genetic influence for various social characteristics. Therefore, compared to the original belief system, the resulting one presumes that genetics has, on average, a greater influence on human beings. This reasoning provides a basis for one last hypothesis: the dissemination of news about behavioral genetics can cause greater endorsement of genetic essentialism as a world view. The logic behind this hypothesis is the same as for the genetic interpolation hypothesis. People will read a message about behavioral genetics and think to themselves: "If genetics is strong enough to have a significant influence on X or Y, then its the general role in causing racial differences, gender differences or family similarities must be stronger than I thought." To be clear, we should not expect that exposure to one message or news story will lead people to instantly embrace genetic determinism. But this stimulus will effectively leave the individual with the impression that genetics is more powerful than they initially thought.

In addition to offering a rationale for predicting how people react to behavioral genetics, this theoretical framework accounts for the null finding revealed in previous studies on how people react to medical genetics. Messages claiming that genetic predispositions influence a medical or biological condition, such a salt sensitivity in Smerecnik's study, convey information that easily fits into people's preexisting belief structure. Indeed, the genetic attribution schema already presumes that these traits are significantly influenced by genetics. While people may adjust their specific genetic attribution for salt sensitivity upward as a response to message exposure, this change does not challenge the general logic of this schema, which therefore remains unaffected.

Charting the limits of the genetic interpolation effect

There are many ways in which the public can learn about behavioral genetics, and it seems reasonable to expect that some stimuli will be more persuasive and will have a stronger influence than others. For example, when reading a whole book about behavioral genetics or participating in a university lecture focused on this topic, people may become aware of many research studies supporting the argument that genetics has a non-negligible impact on social characteristics. Also, such activities take hours to complete and require a significant level of intellectual involvement. For these reasons, books and lessons about behavioral genetics can be considered strong stimuli. In this context, we could expect that a great number of people will accept the behavioral genetics argument and its implications, and will thus perform genetic interpolation. Moreover, as I have shown in my previous study³⁹, news about behavioral genetics can cause genetic interpolation. However, compared to strong treatments like lessons or books, a news story or a magazine article requires less time and presents less evidence. From this perspective, this type of media material appears more like moderate stimuli.

But there also exists other arguably weaker stimuli. In journalism, a "nutshell paragraph," sometimes called "nutgraph," refers to a short paragraph that summarizes the main point of a news story and explains why it is newsworthy. Traditionally, the nutgraph

appears early in a news article, after the lead⁶². Yet, in some contexts, this type of news summary is the only information the public is exposed to. This may be the case for users of news feed applications, such as those designed by many news providers (e.g., BBC, CNN, Al Jazeera), where users can see lists presenting article titles along with a summary sentence and a URL link leading to full-length news articles. Additionally, many websites and tabloids now present daily news using a "news in brief" format, basically publishing nutgraphs without offering access to additional information. This short format fits with the time constraints of the free metropolitan newspapers' target audience, where the common reader only takes a few minutes to browse through the news while using public transportation. Also, since an increasing number of customers read news using smart phones or tablets, it should be no surprise to see the emergence of applications specializing in this kind of news format (see for instance brief.news or newser.com).

Many findings derived from behavioral genetics research are eye-catching, and their main conclusions are often oversimplified though exaggerated claims. For these reasons, it would not be surprising to see these findings being summarized and disseminated to the public using the nutgraph news format. These short stimuli offer a treatment that is likely even softer than conventional news articles. However, tabloids and news apps have the potential to reach a large audience, certainly much larger than the audience of books, academic lessons, specialized magazines or broadsheet newspapers. For this reason, it is crucial to assess how non-experts react when exposed to this information. The third contribution of this article is to test whether a short paragraph about behavioral genetics research suffices to cause the genetic interpolation effect.

I designed my experiment with three goals in mind. As seen earlier in the literature review, Smerecnik's study³⁷ suggests that a soft stimulus pointing to the influence of genetics in causing a medical condition does not lead people to increase genetic attribution for other medical traits. Firstly, my experiment attempts to replicate this null-finding, this time deploying greater statistical power and using a paragraph about how genes impact on physical characteristics. Secondly, the experiment verifies if being exposed to a short paragraph about how genes can influence a complex social trait is sufficient to cause the genetic interpolation effect. Finally, one interesting aspect of the news coverage is that some news stories are clear about the fact that the role of genetics is not deterministic, while others remain ambivalent. The third objective of my study is to explore whether clarifying that genes have a probabilistic influence impacts on the way people react to the stimulus.

Discovering that a single paragraph about behavioral genetics can cause genetic interpolation would have at least two important implications. Firstly, this finding would reinforce the position held by many intellectuals and social scientists who are critical about media coverage of human genetics, and express concern about how this coverage might impact on public understanding of genetics. Finding that even a soft stimulus on behavioral genetics conveys an image that is strong

enough to reshape public understanding of genetics would offer a reason to question the relevance of disseminating behavioral genetics using such short and overly simplified communication devices. Moreover, from a psychological perspective, this conclusion would suggest that the genetic attribution schema is a belief structure that is less rigid than one might initially expect. If, however, no side effects emerge, the results would encourage future work studying the genetic interpolation effect to narrow the scope of investigation and focus efforts on testing the influence of stronger stimuli.

Hypotheses

Leaning on the theoretical framework presented above, I predict that:

- H1: Exposure to a short paragraph claiming that genetics can impact on physical characteristics will have no average influence on the genetic attribution schema.
- H2: Exposure to a short paragraph claiming that genetics can impact on a complex social behavior will increase the average genetic attribution for this specific behavior (persuasion effect).
- H3: If H2 is confirmed, the desire to integrate this piece of information into the genetic attribution schema without compromising its fundamental logic will lead to an increase in genetic attribution for other social traits (genetic interpolation effect). The size of the interpolation effect will be smaller than the persuasion effect.
- H4: Exposure to a short paragraph claiming that genetics can impact on complex social behavior will have no significant effect on genetic attribution for physical traits.

In addition to testing these hypotheses, this study will further explore whether the effects predicted in H2 and H3 vary when the paragraph clarifies that the influence of a specific gene is probabilistic and relatively small.

Methods

This study was conducted as part of the Short Study Program of Time-Sharing Experiments for the Social Sciences (TESS). TESS invites social scientists to submit their experimental protocol. If the protocol receives a positive evaluation, the experiment is funded and fielded inside a web survey on a sample of approximately 2000 American subjects along with other research projects from other researchers. To be eligible for the Short Study Program, projects must involve stimuli that take no more than 90 seconds to administer and require no more than three question items. Researchers are provided with basic sociodemographic information on each individual subject. My experiment was fielded by the firm Government for Knowledge during the month of August 2013. Further details about the sampling methodology can be found on the Open Science Framework page where the data are archived⁶³. This study leans

on a final sample of 2080 subjects. No participants were excluded from the data provided by the survey firm.

Subjects were randomly assigned to one of four experimental conditions without their knowledge. Table 1 reports the stimulus for each experimental condition. Group 1 was exposed to no information and is used as our control group. Group 2 was presented with a short paragraph about the genetics of body traits. This paragraph begins by reminding subjects about the advent of the Human Genome Project (HGP), before pointing their attention to a general conclusion about the strong influence of genetics on physical conditions. “Coding the chemical components” may not be the most appropriate expression to accurately describe what researchers involved in the HGP were actually doing. This ambiguity—for which I am the only person to blame—may have caused confusion among some of the participants. Luckily, this mistake does not blur the key argument of this stimulus: that some “physical traits are strongly influenced by genetics.” In contrast, the stimulus to which Group 3 was exposed argues that genes can impact on voter turnout. This influence is possible because genes impact on some of the personality traits that motivate some citizens to vote. The content of this paragraph reflects the causal framework that emerges from a number of studies in genopolitics, an interdisciplinary field that applies behavioral genetics research methods to study political behavior and orientation⁶⁴. Finally, Group 4 was given the same material as Group 3, plus a sentence presenting one of the genes involved and clarifying that its influence is probabilistic and rather small.

Following treatment exposure, subjects were asked how much they believe genetics (as opposed to the environment) impacts on three human traits: “Turning out to vote,” “intelligence,” and “natural hairstyle (curly or straight).” These items were presented in random order. Table 2 shows the 11-point response scale. This experiment included no other question items and no other treatment groups.

Figure 2 presents a Consort Diagram showing how many participants were initially contacted, how many participated in the study, and how many were assigned to each experimental group. Table 2 reports a randomization check. All four groups are very similar in terms of sociodemographic characteristics, suggesting that randomization succeeded in producing comparable groups.

The final sample size for this experiment was imposed by TESS design constraints for the Short Study Program. However, the four experimental groups (see Table 3, bottom row) are similar or greater in size to those I recruited for my previous study during which subjects were exposed to real news articles. It follows that this experiment would have sufficient statistical power to detect the treatment effects of similar sizes than those I found in this earlier study (between 3 to 8 percentage points on the 0% to 100% response scale).

Table 1. Experimental conditions.

Group	Hypotheses	Stimuli
1		No stimulus
2	H1	"Between 1993 and 2003, scientists from around the world have worked on coding the chemical components of human DNA. Now that the Human Genome Project is complete, geneticists strive to understand the roles played by specific genes. For example, research finds evidence that eye color, hair color and whether someone has attached or free hanging earlobes are physical traits that are strongly influenced by genetics."
3	H2, H3 and H4	"A new field of research, called genopolitics, investigates the influence of genetics on political attitudes and behaviors. Political scientists have found the act of voting to be associated with prosocial personality traits like altruism and open-mindedness. Geneticists have further uncovered that these traits are themselves partly shaped by one's genetic makeup. This research has led to a growing number of studies showing that genes indirectly influence political participation in general, and voter turnout in particular."
4		Same as Group 3 + "For example, one study shows that turnout is 5% higher among people with one form of the MAOA gene than among people with the other form of the gene."

Table 2. Response scale used for each question.

Influence of genetics	0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
Influence of the environment	100%	90%	80%	70%	60%	50%	40%	30%	20%	10%	0%
	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Figure 2. Consort Diagram.

Table 3. Randomization check.

Variable	Gr 1	Gr 2	Gr 3	Gr 4	All Sample
	%	%	%	%	%
Male	49.0	48.1	46.8	50.4	48.6
Female	51.0	51.9	53.2	49.6	51.4
18–24	8.5	7.4	7.6	8.0	7.9
25–34	12.3	14.2	13.5	13.4	13.4
35–44	15.2	13.9	14.7	17.3	15.2
45–54	18.6	19.8	19.1	17.7	18.8
55–64	22.4	22.2	22.7	21.6	22.2
65–74	16.3	15.3	17.7	15.4	16.2
75+	6.6	7.2	4.8	6.6	16.4
Less than HS	7.6	8.1	8.0	8.4	8.0
High School	29.2	28.7	29.7	28.2	28.9
Some College	28.7	29.2	28.3	27.2	28.4
Univ. Degree	34.5	34.0	33.9	36.2	34.7
Less than 25K	16.7	16.8	15.1	15.4	16.0
25K to 50K	23.0	22.2	24.3	21.2	22.6
50K to 75K	20.1	18.3	20.9	19.5	19.7
75K to 100K	13.5	16.3	12.9	15.2	14.5
100K to 125K	11.6	13.3	12.7	14.0	12.9
125 and +	15.2	13.1	14.3	14.8	14.3
Extr Lib	3.2	3.9	3.2	3.3	3.4
Liberal	14.2	12.6	12.7	14.0	13.4
Slightly Lib	8.2	12.4	11.2	11.7	10.9
Moderate	32.1	31.6	32.7	36.0	33.1
Slightly Cons	11.8	13.1	14.1	13.0	13.0
Conservative	23.9	21.4	20.5	17.1	20.8
Extr Cons	5.9	3.7	3.4	3.7	4.2
Refuse	0.8	1.3	2.2	1.2	1.4
N of obs.	527	541	498	514	2,080

The analyses that follow are divided into three steps. Firstly, for each dependent variable, a one-way ANOVA will assess if there are significant differences between experimental groups. Secondly, those mean differences which are directly related to the hypotheses will be calculated using Dunnett's test which corrects for the greater error risk induced from multiple simultaneous comparisons. These tests will exclude data from Group 4 since the aim of this group is not to test a particular hypothesis but to explore if participants react differently than those in Group 3. The third step of the analysis will explore the differences between Group 3 and Group 4 using tests that do not correct for multiple comparisons. For all the analyses presented in this paper, the statistical threshold is set to $p < 0.05$, two-tailed.

Results

Table 4 reports the number of observations, the mean value and the standard deviation for the three dependent variables in each group. In line with the genetic attribution schema, respondents from Group 1 (the control group, without stimulus) report, on average, a higher genetic attribution for “natural hair style”

than for “intelligence,” and higher genetic attribution for “intelligence” than for “turning out to vote.” Next, three ANOVAs are performed, one for each variable, in order to test for whether there exist significant group differences. For the item “turning out to vote,” the ANOVA analysis shows that there are statistically significant differences between groups ($F(3,2012) = 6.77$, $p < .001$, $\eta^2 = .010$). In contrast, no group differences are found for “intelligence,” ($F(3,2015) = 0.68$, $p = .565$, $\eta^2 = .001$). While the theoretical framework predicts no effects for the item “hair style,” the ANOVA does indicate the presence of significant group differences ($F(3, 2024) = 5.50$, $p < .001$, $\eta^2 = .008$).

Following each ANOVA, means for Group 2 and 3 are compared to Group 1 using Dunning's method for multiple comparisons. Table 5 organizes results by group to facilitate discussion. Hypothesis 1 predicts that subjects exposed to a paragraph about how genes can influence physical traits (Group 2) will report genetic attribution levels that are similar to those found in the control group (Group 1). The results displayed in the three lines at the top of Table 5 show support for this hypothesis. The observed group differences are small, and none of them reach statistical significance.

Hypothesis 2 focuses on the persuasion effect, and predicts that exposure to a paragraph about genopolitics (Group 3) will cause respondents to increase their genetic attribution for “turning out to vote” compared with the control group (Group 1). The first line of the bottom part of Table 5 confirms this hypothesis. In Group 3, the average genetic attribution for voting is 4.1 percentage points higher than in Group 1. While statistically significant, this persuasion effect is much weaker than the one found in my previous study, where respondents were exposed to a real article about genopolitics or genoeconomics (between 11 and 17 percentage points).

Hypothesis 3 focuses on genetic interpolation. It predicts that, in response to the paragraph about genopolitics, subjects from Group 3 will infer greater genetic causation for intelligence. The theoretical framework also predicts that the genetic interpolation effect will be smaller in size than the persuasion effect. Table 5 shows that the mean genetic attribution for intelligence in Group 3 is not statistically different from the one found in Group 1.

Finally, Hypothesis 4 predicts that exposure to the paragraph about genopolitics will not affect the average genetic attribution for natural hair style. However, the bottom line of Table 5 reveals a statistically significant negative difference for this mean comparison. Genetic attribution for natural hair style is 4.2 percentage points lower in Group 3 than in Group 1. Therefore, H4 is not supported by the data.

Group 4 was designed to explore whether clarifying that the effects of a single gene on political behavior is probabilistic in nature could change how people react to this information. The results, reported in Table 6, show that none of the genetic

Table 4. Aggregate statistics by group and dependent variable.

Trait	Group 1			Group 2			Group 3			Group 4		
	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD
Turning out to vote	514	27.2	26.2	523	25.1	27.4	480	31.4	27.4	499	31.4	26.9
Intelligence	518	56.9	23.8	526	58.7	22.7	481	58.0	22.2	494	57.1	23.4
Natural hair style (curly or straight)	517	80.9	24.5	529	82.1	24.3	483	76.6	28.3	499	76.9	29.0

Note: Genetic attribution values are rounded at the first decimal.

Table 5. Effect of treatment exposure on average genetic attribution.

Group Comparison	Dependent Variable	Contrast	Std. Err.	t	P> t	[95% Conf. Interval]	Cohen's d
Gr 2 - Gr 1	Turning out to vote	-2.1	1.7	-1.262	0.345	[-5.8 ; 1.6]	0.08
	Intelligence	1.8	1.4	1.266	0.342	[-1.3 ; 4.9]	0.08
	Natural hair style	1.2	1.6	0.772	0.657	[-2.3 ; 4.7]	0.05
Gr 3 - Gr 1	Turning out to vote	4.1	1.7	2.404	0.030	[0.3 ; 7.9]	0.16
	Intelligence	1.1	1.5	0.739	0.680	[-2.1 ; 4.3]	0.05
	Natural hair style	-4.2	1.6	-2.595	0.018	[-7.8 ; -0.6]	0.16

Note: Genetic attribution values are rounded at the first decimal.

Table 6. Exploring the effect of specifying that the influence of a particular gene is small and probabilistic.

Group Comparison	Dependent Variable	Contrast	Std. Err.	t	P> t	[95% Conf. Interval]	Cohen's d
Gr 4 - Gr 3	Turning out to vote	0.0	1.7	0.016	0.987	[-3.4 ; 3.4]	0.00
	Intelligence	-0.9	1.5	-0.615	0.538	[-3.8 ; 2.0]	0.04
	Natural hair style	0.3	1.8	0.147	0.883	[-3.3 ; 3.9]	0.01

Note: Genetic attribution values are rounded at the first decimal.

attribution mean values observed in Group 4 are significantly different from those of Group 3 Overall, this exploratory analysis offers no indication that this clarification changes how subjects interpret the message about genopolitics.

Secondary findings

The theoretical framework behind the genetic interpolation hypothesis assumes that most people adhere to the genetic attribution schema, where genetics is seen as having a greater influence on physical traits, a weaker influence on psychological traits, and an even weaker influence on complex social behavior. As presented earlier in this text, various studies support this assumption, at least in the aggregate. However, so far, no study has explored how genetic attributions are structured at the individual level.

For the purpose of this secondary analysis, only participants from the control group are considered. Table 7 presents their distribution among nine possible configurations of beliefs. In the

top left cell, we can see that, out of the 508 respondents who offered a valid answer to all three response items, 282 (56%) show a response pattern that is generally consistent with the genetic attribution schema.

The other cells report the distribution of responses among alternative belief configurations. When looking at the cell located in the middle of the table, we can see that 29 respondents assigned the same genetic attribution for all three characteristics. Among these, only six respondents (1%) adopt genetic determinism as a world view and report that genetics account 100% for hair style, intelligence, and voting at elections. At the extreme opposite, only 2 respondents (.3%) report believing that genes play no role whatsoever in explaining any of these traits.

It is not difficult to imagine a scenario where the genetic interpolation hypothesis would occur, but only among the subgroup of participants whose pre-treatment configuration of

Table 7. Configurations of genetic attribution beliefs in the control group.

	$GA_{intelligence} > GA_{voting}$	$GA_{intelligence} = GA_{voting}$	$GA_{intelligence} < GA_{voting}$	Total
$GA_{hair} > GA_{intelligence}$	282 (55.5%)	44 (8.7%)	37 (7.3%)	363 (71.4%)
$GA_{hair} = GA_{intelligence}$	54 (10.6%)	29 (5.7%)	6 (1.2%)	89 (17.5%)
$GA_{hair} < GA_{intelligence}$	43 (8.5%)	7 (1.4%)	6 (1.2%)	56 (11.0%)
Total	379 (74.6%)	80 (15.7%)	49 (9.6%)	508 (100.0%)

beliefs fit with the genetic attribution schema. The effect among this subgroup would then become diluted and lose significance when all participants are combined, accounting for the null finding observed in H3. Unfortunately, the current data leans on a cross-sectional survey, and therefore, it is impossible to measure treatment effects conditioned by preexisting beliefs.

Discussion

One interdisciplinary branch of research investigates how people form their beliefs about the influence of genetics on human traits. A potential source of influence is the information disseminated in news media coverage of genetics research findings. In fact, many intellectuals have expressed concern about the risk that news about human genetics would lead the public to believe that genes play a dominant role in individual differences. My review of the literature shows that researchers have addressed this issue empirically by testing how people react to news about medical genetics. Results of various studies have largely failed to support original concerns and instead showed that people tend to interpret this information with caution.

While this conclusion seems to be robust, my own personal contribution to this literature, published in 2014, reanimates the debate. My study suggests that people react differently to behavioral genetics than they do to medical genetics. It showed that news stories about how genes impact on a specific behavior can lead people to generalize this finding to other social traits. The results further demonstrate that people's reactions are not a sudden endorsement of genetic determinism. Instead, participants show a modest but systematic upward adjustment of their genetic attribution for other social orientations and behaviors. However, due to space limitations, this 2014 contribution did not elaborate extensively on the potential psychological mechanism that may account for this phenomena.

The present article is a direct follow-up to my previous study³⁹, and copes with some of its limitations. The core contribution of this article is to present an original theoretical framework that offers clear hypotheses predicting how people react when exposed to news about behavioral genetics research findings. This framework combines two theoretical elements that are well-established in the literature. The first is the concept of schema. This concept is mobilized to account for people's pre-treatment genetic attribution belief system, and how they view a positive relationship between genetic causation in human traits, and the perceived distance of these traits from biological roots.

The second concept is the anchoring and adjustment heuristics. This cognitive shortcut accounts for how individuals react when exposed to behavioral genetics.

This framework leads me to make some predictions about how people react when exposed to information suggesting that genes are powerful enough to have a non-negligible impact on complex social traits. The central prediction is the genetic interpolation hypothesis, which predicts that people will adapt their genetic attribution schema in an attempt to integrate this new piece of evidence, but will try to do so without compromising the basic logic of their belief system. As a result, people will incrementally increase genetic attribution for various other social traits that are not mentioned in the information material. Furthermore, this reasoning leads me to predict that exposure to news about behavioral genetics will increase genetic essentialism as a world view.

In addition to this theoretical development, the present article also contributes to the literature by attempting to evaluate empirically whether exposure to a weak stimuli suffices to trigger the genetic interpolation effect. The relevance of this test should not be understated. Imagine that this experiment had found that even such a soft treatment could lead people to adjust their belief system. First, this finding would have suggested that the genetic attribution schema can be altered relatively easily. And second, one could likely infer that almost any other stimuli reporting about behavioral genetics would succeed at reshaping attribution beliefs.

In contrast, the results of this survey experiment suggest that a short paragraph, presented out of context, is not sufficient to cause the genetic interpolation effect. While participants do increase their genetic attribution for the particular behavior presented in the stimuli – turning out to vote – their genetic attribution for intelligence remains unaffected.

This experiment also presents a finding that my theoretical framework fails to account for. Exposure to behavioral genetics caused participants to modestly reassess downward their genetic attribution for a physical trait: natural hair style. The exact reasons for this unexpected finding remain open to speculation. One possibility, consistent with the patterns observed in Table 5, is that the treatment caused ambivalence or confusion about how genetics impact on human beings, leading some participants to select middle-range response values to express their uncertainty⁶⁵. This experiment did not include questions

asking respondents how confident they are about their response, and therefore it is not possible to test whether this interpretation is accurate.

Altogether, the theoretical framework and the experiment presented in this article offer many interesting directions for future research. For instance, researchers could design new studies to address one or many of the following research questions:

1. To what extent does the genetic interpolation effect vary depending on pre-treatment genetic attribution beliefs?
2. Does genetic literacy moderate the genetic interpolation effect?
3. How do people react when exposed to other, arguably stronger types of treatments (e.g., attending a lecture, reading a book, being exposed to a live or web conference)?
4. Are some types of scientific evidence more likely to trigger the genetic interpolation effect? Would behavioral genetics findings involving GxE interactions or epigenetics also cause the same effect?
5. Are genetic attribution beliefs impacted permanently, or do they return to their original level after some time, and in the latter case, after how long?
6. Does repeated exposure to findings about behavioral genetics impact on the strength of effects or their duration?

Answering these questions will certainly require the mobilization of other theories relevant to the particular dimensions investigated. But in the end, we can hope that the collected evidence will help design new guidelines for communicators, be they science journalists, educators or behavioral geneticists worried about the implications of their works for society.

Ethical approval

The study was approved by the Comité d'éthique de la recherche en arts et en sciences of the Université de Montréal. The firm Gouvernement for Knowledge obtains and documents informed consent and agreement to the study's Privacy Policy and Terms and Conditions during the registration process. Participants are also reminded that their participation to the panel can be interrupted at anytime. The Comité d'éthique did not require the collection of additional consent for participation in the present study because the stimuli pose minimal risks to the participants and involve no deception.

Data availability

The dataset and the pollster report for this study are hosted on the Open Science Framework: DOI: [10.17605/OSF.IO/2UBP2](https://doi.org/10.17605/OSF.IO/2UBP2).

Author contributions

AMC conducted all of the work related to this study.

Competing interests

No competing interests were disclosed.

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Version 2

Reviewer Report 13 April 2018

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Fabien Medvecky

Centre for Science Communication, University of Otago, Dunedin, New Zealand

This manuscript aims to do three things. It 1) offer a review of the literature on the effects of news articles about genetics on lay people's beliefs in genetic determinism, 2) presents a framework for understanding and predicting this interaction, and 3) tests this framework through a (very) brief empirical study. This is an interesting subject of research, though I fear it tries to do too much and ends up not doing any of it particularly well. I think the article will require substantial revisions before it can be considered for publication.

I have two main concerns. Firstly, I think the literature review is too long, yet not thorough enough. While the paper makes claims about science communication generally, and about the effect of media article on public attitudes and public understanding of science, the literature that is actually reviewed is very limited and specific, but is discussed at unnecessary length. For example, there are virtually no references to science communication generally, and more importantly, there is no review of the very extensive general literature on the effect of media discussion of science on public attitudes. This seems a large omission, and picking up the small handful of papers that specializes on the issue of the effects of news articles about genetics on lay people's beliefs is simply too small a field to allow for a meaningful review. Specifically, the literature on framing would be helpful here, as would a more broadly researched, but more succinctly presented discussion on the role and capacity to the media to influence attitudes.

My second concern is that I don't fully see the point of the proposed framework given the results from the survey. As mentioned by another reviewer, "the results of [the] brief study [...] seem to disprove such hypothesis." If the framework is not supported by the study, I'm unsure what the value is spending so much time in developing it and presenting it in the first place. I wish I could be more positive, but I'm not sure what this proposed framework does to advance our understanding in light of the study.

I'm not quite sure what would make this a stronger study as I'm unsure of its purpose. Perhaps further empirical tests might reveal that this framework is, indeed, helpful and accurately representative, and this might be a helpful way to move the idea forward.

Is the work clearly and accurately presented and does it cite the current literature?

No

Is the study design appropriate and is the work technically sound?

Partly

Are sufficient details of methods and analysis provided to allow replication by others?

Yes

If applicable, is the statistical analysis and its interpretation appropriate?

Partly

Are all the source data underlying the results available to ensure full reproducibility?

Yes

Are the conclusions drawn adequately supported by the results?

No

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: science communication, philosophy of science

I confirm that I have read this submission and believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.

Author Response 13 Apr 2018

Alexandre Morin-Chassé, Université de Montréal, Montréal, Canada

Dear Reviewer,

Thanks for the time you spend working on this review.

Regarding your first concern, I believe it is unfair to reject a paper for this. My contribution is narrow in focus, and this is also the case of many research in the field of science communication. Your review argues that "the paper makes claims about science communication generally, and about the effect of media article on public attitudes and public understanding of science". I have been very cautious not to make general claims about science communication. To only direct mention is *in the abstract*, to introduce my work as belonging to this field.

"My second concern is that I don't fully see the point of the proposed framework given the results from the survey."

As I carefully explain in my text, there already exists robust evidence supporting the

theoretical framework I propose. This evidence is found in my previous study, Morin-Chassé (2014), a work I take time to explain in my paper. However, Morin-Chassé (2014) was an exploratory work largely based on intuition. The theoretical section of my manuscript offers a deductive framework accounting for my previous finding, a framework from which hypotheses can be derived and tested. This is a significant contribution to the literature.

As I also explain in my text, the aim of the empirical study is to test if these hypotheses hold under extremely soft conditions, i.e. when respondents are exposed to a short paragraph about behavioral genetics rather than a whole news article. The answer is no. This empirical contribution helps to chart the limits of my framework. This kind of null finding is essential to orient future work, and it shows that a simple paragraph is not enough to move people's beliefs.

I am sorry to see that my work is rejected in these grounds.

Alexandre Morin-Chassé

Competing Interests: No competing interests were disclosed.

Reviewer Report 16 February 2018

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Matteo Farinella

Columbia University in the City of New York, New York, NY, USA

The manuscript titled "**Communicating behavioral genetics: Charting the limits of the genetic interpolation effect**" provides a review of existing literature on how the effects of exposure to news articles about genetics may affect people's beliefs in genetic determinism. The author also introduces theoretical constructs such as *genetic attribution schema* and the *genetic interpolation hypothesis* and finally presents the results of a brief study which seem to disprove such hypothesis.

I think this is an interesting subject of research and the article has considerably improved from the previous version. The first section provides a good theoretical background and many useful references were added. The introduction of graphics also help clarify the *genetic interpolation hypothesis* and the study design. However, I think the article still requires some major revisions in order to be published.

In particular, I think the introduction is too long and follows an odd structure. The literature presented in the first section seem to suggest that exposure to short news articles does *not* in fact affect people's belief in genetic determinism. Nonetheless, the next two paragraphs proceed to

introduce the genetic interpolation as a valid hypothesis.

I think a more logical structure would be:

1) Theoretical background for the *genetic interpolation hypothesis*.

2) Literature review: does previous research support the hypothesis or not and, if not, why the author still thinks this is line of research worth pursuing. In particular, if exposure to articles claiming that genes affect physical traits has no influence on the genetic attribution schema, why should we expect a different result for behavioural genetics articles? Is there any rationale behind this, beyond the 2014 paper by the same author?

3) Justification for the new study: how does the current study improves or expands upon previous research (i.e. it has greater statistical power and explores beliefs in the subfield of behavioural genetics). The author clearly state this at page 9 of the manuscript but I think it should come much earlier in the paper, possibly even in the abstract:

"My experiment attempts to replicate this null-finding, this time deploying greater statistical power and using a paragraph about how genes impact on physical characteristics. Secondly, the experiment verifies if being exposed to a short paragraph about how genes can influence a complex social trait is sufficient to cause the genetic interpolation effect. [...] The third objective of my study is to explore whether clarifying that genes have a probabilistic influence impacts on the way people react to the stimulus."

On the same note, at the moment the abstract reads more like a truncated introduction and I think it could be rewritten in a more clear concise way. In particular, it should clearly state the null results of the study, rather than ambiguously ending with "I present the results of a survey experiment that was designed to test whether a simple, short paragraph about behavioral genetics is a powerful enough stimulus to cause the genetic interpolation effect." without telling us whether it does or not in fact confirm the hypothesis.

I also agree with previous reviewers that although the experimental design seems appropriate, the procedures could be described in more detailed. For example, how did the 'survey firm' contacted the participants? Was the participation purely voluntary or were the participants rewarded for their participation? How were the different stimuli presented and which software was used to collect and analyse the responses? I am also concerned about the fact that previous concerns of 'straightlining' are completely dropped in this version of the paper. Has the author somehow addressed these somehow?

Finally, I have some concern regarding the choice of 'voting' as the behavioural trait claimed to have genetic influence. The mentioning of politics or voting may elicit strong feelings in participants which have nothing to do with their scientific beliefs. Therefore, I don't think we can draw any general conclusions from the exposure to a single article on such a specific subject matter. I am all in favour of publishing negative findings but to confidently reject the genetic interpolation hypothesis it would be appropriate to test exposure to several different articles, claiming genetic influence on other types of complex behaviours, more or less believable (e.g. marriage, travelling, physical activity) and see if these also have no effect on participants' beliefs. At the very least this obvious limitation should be addressed in the Discussion.

In conclusion, I think at the moment the paper reads as an odd mix of a very long and detailed literature review and a very brief - not incredibly well justified - experimental report. I think it would greatly benefit from additional empirical evidence (in order to firmly dismiss the genetic interpolation hypothesis) or it should be otherwise rewritten as a much shorter report, clearly describing the study as a replication of previous null-findings, rather than arguing at length for an hypothesis with little or none empirical support.

Other comments:

- When discussing existing literature it would be better to summarize the conclusions, rather than stating the general goal of the study, so that readers do not need to look up the original reference. For example, instead of saying: "Some research also verifies whether participation in an introductory biology class or social science programs influences students' views on the role of genetics", clearly say if such studies found an influence or not.
- Similar to previous referees, I also find slightly confusing how sometimes the *genetic interpolation hypothesis* is presented as a somehow established phenomena, instead of an hypothesis formulated by the author. I think this should be more clearly stated throughout the paper.
- In fact, all the empirical studies cited by the author (Condit *et al.* 2001¹ and Lynch *et al.* 2008²) show no evidence that message exposure produce deterministic beliefs. Also Smerecnik *et al.* 2010³ found that when beliefs were changed these did not "extrapolate" to other diseases (I think the appropriate term here should be 'generalize'). Then why does the author still think the genetic interpolation hypothesis is a viable hypothesis? The author claims that "these two studies would fail to detect more nuanced or subtle effects." but that is a very subjective statement. Why exactly would they fail? The author should clearly discuss the limitations of previous studies (if there are any, beyond the statistical power) or otherwise present his findings as a replication of previous work, in the specific subfield of behavioural genetics. Again, this becomes clearer later in the paper but could be more explicitly stated to begin with.
- The discussion of the 2014 study by the same author is also too long and detailed. I think the main finding as clearly summarized at page 5: "news articles impact views about the influence of genetics when they cover research on behavioral genetics, but not when they cover research on medical genetics." This is an important finding and should be mentioned earlier in the introduction as the rationale behind the current study.

Minor comments:

- I think some of the data would be more readable as histogram plots, rather than numeric tables.
- I personally dislike the term "lay people" for historical/cultural reasons, why not simply ask "How do non-experts understand human genetics?" in the opening paragraph.
- Who exactly are these "intellectuals" mentioned throughout the paper? Maybe "public figures" or "other authors" would be more appropriate.

- If possible, please avoid using subjective terms such as ‘fascinating’ and ‘clever’.
- To say that “my 2014 paper offers an important empirical contribution” is slightly biased in my opinion. We clearly all believe that our contributions are important, but it is not up to us to decide that.

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Is the work clearly and accurately presented and does it cite the current literature?

Partly

Is the study design appropriate and is the work technically sound?

Partly

Are sufficient details of methods and analysis provided to allow replication by others?

Partly

If applicable, is the statistical analysis and its interpretation appropriate?

I cannot comment. A qualified statistician is required.

Are all the source data underlying the results available to ensure full reproducibility?

Yes

Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Reviewer Report 13 February 2018

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Brian M. Donovan

Biological Sciences Curriculum Study, Colorado Springs, CO, USA

The author has adequately addressed some of the methodological issues raised in my last review. Most importantly, however, (s)he has not addressed the post-hoc power analysis. The author found evidence that priming a genetic attribution for voting increases genetic attributions for voting whilst decreasing genetic attributions for natural hair style. I think the discussion could unpack these (null) findings more. Is it possible that both findings are merely noise, that is to say Type I errors? Or are they Type II errors? The significant findings in this study have a standardized effect size of $d = 0.16$. But, this study may only be able to detect effects that are $d = 0.2$ or greater. So, the null findings may be a Type II error and/or the significant findings may be a spurious finding (Type I error). The author could strengthen the paper by discussing the significance of the null and significant results in light of their own post-hoc power analysis. I suggested this in my last review, but I do not see anything in the manuscript about the achieved power of their trial for each contrast. Nor was there any information reported about the planned power analysis for the experiment, which I also requested in my last review. I cannot approve the paper until the author discusses the null and significant findings in light of the achieved statistical power for any single statistical contrast between conditions.

Given that the author has substantially added to the conceptual framework in a way that draws upon my own area of research I also wanted to make the following suggestions to further improve the paper.

This is really two different papers, a theoretical one and an empirical one. Currently, a theoretical paper is crammed together with an empirical paper. Of course, the problem with doing this is that the null findings do not support the assertions laid out in the theoretical portion of the manuscript and the significant findings that do occur are not consistent with the genetic interpolation hypothesis. This raises the question, is the conceptual framework, as written, the appropriate framework for this paper?

I think the author needs to cut much of the new content added into the introduction after the first review and hone down this paper to the core argument needed to justify his priming experiment and why there might be null results. As I state in **bold** below, I think that argument needs to be about the boundary conditions of the genetic interpolation hypothesis. Then, (s)he can turn much of the content added into the conceptual framework after the first review into a separate stand-alone theoretical paper. At present, the paper unpacks a very large hypothesis that about the communication of science and the psychology of essentialist thinking, but then it offers very little direct evidence from the null experiment to support that very hypothesis.

Suggestions to improve the conceptual framework:

There is a need to address the issue of whether the genetic interpolation hypothesis applies to conceptions of individual difference versus group level difference. The conceptual framework now

ties the genetic interpolation hypothesis to the field of psychological essentialism. Genetic essentialist thinking can be applied to groups (gender, race, sexuality) and also behaviors believed to be under volitional control. The literature here is very complex because genetic attributions about group behavior versus individual behavior have different effects on social behavior. These differences are explained in the Dar-Nimrod and Heine (2011) paper cited by the author. For example, genetic attributions of gender or racial difference tend to be associated with increased racism and sexism whereas genetic attributions of homosexuality tend to be predictive of decreased homonegativity (see Jayaratne et al. 2006 or work by Nick Haslam). Furthermore, a variety of field experiments conducted by Donovan (2014, 2016, 2017) have found that priming adolescents to believe that races differ in genetic disease prevalence also causes increased belief that races differ in intelligence. Is this a genetic interpolation effect in the context of cognition of group difference? I would think so. And, if it is, then why does the genetic interpolation effect show up when priming adolescents about race and genetic diseases with a 2-page reading in the context of the science classroom among adolescents, but not in this paper?

It would be good for the author to state whether the genetic interpolation hypothesis applies only to the domain of cognition of individual difference, or whether it also applies to cognition of group difference. If it applies to both domains of thinking (i.e. differences between individuals and differences between groups) then the author needs to craft an argument about how his genetic interpolation hypothesis is similar to, or different from, genetic essentialism theory. Is it a refinement of the genetic essentialism hypotheses? An addition?

In fact, I find the conceptual framework to be too broad and think much of it could be cut to create more space to unpack the genetic interpolation hypothesis and the boundary conditions under which it will operate (or not). Will it operate only in some contexts or with regards to some beliefs about groups, individuals or traits? Boundary conditions are critical because of the null findings. And, the distinction between cognition of individual difference and group difference could be one such boundary condition. Thus, I think this paper needs to be more about the boundary conditions of the genetic interpolation hypothesis. The conceptual framework includes a lot of ancillary information about particular studies that could be cut to streamline the argument a bit more to focus on this issue. If that conceptual work is done on boundary conditions, and if an achieved power analysis is added, then the author may be able to draw some conclusions about the null and significant findings in this study.

Toward that end, the author can also explain how it is possible that people develop a belief that some traits are more under the control of biology than others. The whole basis of the genetic interpolation effect is that individuals are more likely to believe that some traits are more influenced by biology than others. Why is this the case?

The author states, "The structure of this schema is organized such that *the more a trait is believed to be influenced by biology—as deduced from experience, observable features or as learned from other sources—the more genetics plays a predominant role*. I use the term *genetic attribution schema* to refer to this belief structure." This raises the issue as to what are the experiences, observable features or learning experiences that give rise to genetic attributions? The author appears to claim that media exposure to behavioral genetics research is a source of schema production. But, this cannot be the only source. And, if it is, then such media exposure cannot explain how people develop genetic attributions for non-behavioral traits. Also, I am left wondering whether the genetic interpolation process is a hypothesis that explains belief updating or belief formation? It

would seem that it only explains the former, because of the anchoring aspect of it, which might be a boundary condition on where one can detect the genetic interpolation effect, or in which populations it might be present.

In sum, the author has not explained how or why people attribute some traits to genetics more than others. This seems like a critical flaw in the argument. For there is no basis for the genetic interpolation hypothesis unless individuals believe that some traits are more under the control of biological factors than others. Otherwise there can be no anchoring and re-adjustment after exposure to a behavioral genetics text. Thus, there is a need to justify why, empirically, we would predict that people believe that genes play less of a role in credit card debt than they do in intelligence or hair texture at outset, prior to this experiment, as they did in condition 1. I would argue that most of our prior beliefs about genetic attributions are influenced by sociocultural factors, such as educational experiences (i.e. Mendelian genetics education) and stuff like television. Indeed, the literature on genetic reasoning suggests that DNA fingerprinting technology in crime scene investigation television shows influences genetic attributions for behavior and identity (see Venville et al., 2005).

Also, Figure 1 needs to be better explained. Fig. 1d made sense as what we would predict if the genetic interpolation hypothesis is correct. However, Fig 1a-c are not very clear and could be explained better.

In short, I think the author could improve the theoretical portion of the manuscript by addressing the following questions:

- How do people develop genetic attributions for different traits?
- Is the genetic interpolation hypothesis an explanation for conceptions of individual difference, group difference, or both?
- How is the genetic interpolation hypothesis any different from genetic essentialism theory?
- What are the boundary conditions to the genetic interpolation effect?

The following papers show that people develop genetic attributions from educational experiences and also that the genetic interpolation hypothesis might influence how people make sense of group differences in behavior in some contexts but not others:

- Sheldon, J. P. (2018). Incorporating a Discussion of Genetic Attributions Into Psychology Courses¹.
- Donovan, B. M. (2016). Framing the Genetics Curriculum for Social Justice: An Experimental Exploration of How the Biology Curriculum Influences Beliefs About Racial Difference².
- Donovan, B. M. (2017). Learned inequality: Racial labels in the biology curriculum can affect the development of racial prejudice³.
- Donovan, B. M. (2014). Playing with fire? The impact of the hidden curriculum in school genetics on essentialist conceptions of race⁴.
- Jamieson, A., & Radick, G. (2017). Genetic Determinism in the Genetics Curriculum: An Exploratory Study of the Effects of Mendelian and Weldonian Emphases⁵.
- Jamieson, A., & Radick, G. (2013). Putting Mendel in His Place: How Curriculum Reform in Genetics and Counterfactual History of Science Can Work Together⁶.
- Stern, F., & Kampourakis, K. (2017). Teaching for genetics literacy in the post-genomic era⁷.

Suggestions to improve the discussion:

In the discussion the author says that the following questions are unanswered:

1. To what extent does the genetic interpolation effect vary depending on pre-treatment genetic attribution beliefs?
2. Does genetic literacy moderate the genetic interpolation effect?
3. How do people react when exposed to other, arguably stronger types of treatments (e.g., attending a lecture, reading a book, being exposed to a live or web conference)?
4. Are some types of scientific evidence more likely to trigger the genetic interpolation effect? Would behavioral genetics findings involving GxE interactions or epigenetics also cause the same effect?
5. Are genetic attribution beliefs impacted permanently, or do they return to their original level after some time, and in the latter case, after how long?
6. Does repeated exposure to findings about behavioral genetics impact on the strength of effects or their duration?

However, preliminary answers to most of these questions can be found in the papers above. The author could craft a more thorough discussion that deals more substantively with the issues raised in the introduction.

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4. Donovan B: Playing with fire? The impact of the hidden curriculum in school genetics on essentialist conceptions of race. *Journal of Research in Science Teaching*. 2014; **51** (4): 462-496 [Publisher Full Text](#)
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Is the work clearly and accurately presented and does it cite the current literature?

Yes

Is the study design appropriate and is the work technically sound?

Yes

Are sufficient details of methods and analysis provided to allow replication by others?

Yes

If applicable, is the statistical analysis and its interpretation appropriate?

Yes

Are all the source data underlying the results available to ensure full reproducibility?

Yes

Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Genetic essentialism

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Author Response 14 Feb 2018

Alexandre Morin-Chassé, Université de Montréal, Montréal, Canada

Thanks for sending your review so quickly! It is a real privilege to find a reviewer so motivated to making this paper a significant contribution to the literature. I will wait for the other reviews before formally replying to you. Then, if you agree, I would like to engage in a discussion about some the elements you raise in your review.

For the time being, I would like to clarify one point of critical importance. In your review, you suggest that I "turn much of the content added into the conceptual framework after the first review into a separate stand-alone theoretical paper." While I would argue against this proposition, I believe it is relevant to inform you that doing so would not be possible even if I wanted to. F1000 is an open publishing platform. In my view, it has clear advantages, not least of which are open access and open peer review. However, once a manuscript appears online, it cannot be retracted or deleted from the website, except under exceptional circumstances such a fraud. Previous versions are also archived on the website and easily accessible. It follows that it is not possible for authors to submit their manuscript/article to other journals and to argue that it has not been published elsewhere. In a certain way, this is a drawback of the post-publication peer review.

I'll reach back to you via this Comment zone when other reviews come in.

Competing Interests: No competing interests were disclosed.

Version 1

Reviewer Report 10 January 2017

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Kostas Kampourakis

Section of Biology & University Teacher Training Institute, University of Geneva, Geneva, Switzerland

This is a review of the manuscript titled "Communicating behavioral genetics: Charting the limits of the genetic interpolation effect". I have strong reservations about the manuscript on various grounds, and I hope that the author will manage to revise it.

First of all, the author needs to justify why we should care at all about what he calls "the genetic interpolation effect". With this, I do not mean to suggest that we should not care. Rather, I suggest that the author needs to make a stronger case for this. The author writes that "Experimental studies have tested how people react to this kind of news", but the author only cites his own previous work. Are these author's studies all the experimental studies on this topic? Or are there others that have not been mentioned? Is this effect well-established in research? Or is it something recently "discovered"?

The author writes that "This side-effect has been called the *genetic interpolation effect*." By whom? Why? How strong this effect is? How prevalent? No references are given and so we have no idea about these. I am also wondering about the name itself. The author writes that "However, the results also show that disseminating behavioral genetics leads some members of the public to infer greater genetic causation for other complex social traits, which were not the focus of the study presented." This looks more like *extrapolation* to me. Perhaps the author is right, and my point is not to question the choice of the name. Rather my point is to suggest that we need more information about this effect, which is the focus of the article.

There are studies by Ilan Dar Nimrod (<http://sydney.edu.au/science/people/ilan.dar-nimrod.php>) that I find very relevant and that should have at least been discussed in this article. Celeste Condit has also conducted a few studies on how people interpret news articles on genetics. Both of these scholars would be appropriate reviewers for this article. My suggestion would be for the author to briefly review the findings of their research, and relate his own research to those in order to make more explicit what his contribution is. Individual studies may make interesting contributions, but generalizations are not easy to make even if the sample is large.

Some more information about the anchoring and adjustment heuristic would be useful. These are not sufficiently described in the current article, and so one must read the cited articles in order to understand what it is about and how it relates to the research presented therein.

There are some problems with the hypotheses made by the author. Does H2 presuppose H1? This is not clear. My understanding is that it does, i.e. that people first have to increase the influence they attribute to genetics in explaining a characteristic after reading the article, in order to subsequently infer greater genetic causation for other complex social traits. This is at least the inference I make from author's description of H3 and H4. It is of course plausible that this is not

the assumption made by the author. But then the author should explain why this is the case. Even if I am wrong, the author should ensure that readers will not make the same mistake I have made.

Commenting on the statistical methods used falls outside my modest area of expertise. Therefore, I will only comment on the conclusions made assuming that the statistical analysis is appropriate.

The author writes: "A sample of 2080 respondents was recruited from a pre-existing panel, with a response rate of 62.6%" Does this mean that approximately 1302 people responded? If yes, why isn't this made clear so that we know what the sample of the study was? That 38% of those asked declined to respond may be interesting for those conducting this kind of surveys. However, in this case we only need to know the actual sample size; how many people were asked to participate is of secondary importance.

I am wondering how the reliability and the validity of the inferences made from participants responses was established. Did the author ask experts to read the stimuli and confirm that they are appropriate to measure what the author intends to measure? Were these stimuli used in a pilot study of some kind before this study? For instance the author writes that (Table 1) "Between 1993 and 2003, scientists from around the world have worked on coding the chemical components of human DNA." The expression "coding the chemical components" makes no sense to me. What happened during the HGP was that researchers developed methods to **find the sequence** of the chemical components (bases A,T,C,G in nucleotides) of the human genome. **Decoding** this sequence, i.e. finding the information encoded therein, is something that they are still working on. The expression "coding the chemical components" could be very misleading and I do not know if this was done in purpose or accidentally.

On the same topic, I do not see why the author would expect that H5 would be confirmed. The author concludes that "Finally, the fifth column of Table 3 suggests that, contrary to what H5 predicts, presenting a paragraph describing how genetics impacts on human physical traits is not sufficient to generate the genetic interpolation effect." There are numerous studies that provide evidence that people intuitively tend to think about biological characteristics as influenced, if not determined, by genes. People tend to take for granted that "physical traits" "are strongly influenced by genetics". Confusion begins when people mistakenly start wondering how much of a trait is due to genes or due to environment, as they often fail to understand that it is the interaction of the two that brings about a phenotypic outcome and thus they cannot be separated. Now, it seems to me that what the author is testing here is another extrapolation effect: whether a statement about biological traits would form the basis for inferences for a behavioural trait. This is interesting but seems to me to be a different effect than the one that the author wants to test, rather than "an alternative explanation for the genetic interpolation effect."

Based on all this, I suggest that the author carefully reconsiders his conclusions. I am not at all convinced that this conclusion is valid: "it seems plausible to interpret this null finding as an indication that a short paragraph on behavior genetics is a treatment condition that is not strong enough to cause people to update their general belief framework about the influence of genetics." I am inclined to think that when it comes to genetics even a replacement effect is possible, i.e. reading a highly deterministic headline could make people think in more deterministic terms about genetics. There is no research on that, but some relevant research suggests that this could be possible (e.g. Ecker, U. K., Lewandowsky, S., Chang, E. P., & Pillai, R. (2014). The effects of subtle misinformation in news headlines. *Journal of Experimental Psychology: Applied*, 20(4), 323

– 335.). This is just speculation based on personal experience, and we need research to draw conclusions. The conclusions of this article seem to point to the opposite direction, but I am not sure about their validity. The author should work more and reconceptualize the available data in order to be more convincing that his conclusions are valid.

Therefore, I have strong reservations about the quality and the conceptual foundations of this study, and I would like to see if the author will manage to address my concerns.

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Yes

Is the study design appropriate and is the work technically sound?

Yes

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Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Reviewer Report 08 December 2016

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Brian M. Donovan

Biological Sciences Curriculum Study, Colorado Springs, CO, USA

In “Communicating behavioral genetics: Charting the limits of the genetic interpolation effect” the authors report research from an experimental trial run online which explores how exposure to genetic information about human behaviors affects beliefs about the genetic (versus environmental) determinants of social behavior. This topic is an important area for research given the often widespread misunderstanding of genetics and behavior, which is both perpetuated through genotype in the media and also through science education. Furthermore, genetic determinism has been shown to be implicated in a variety of social prejudices by social psychologists. Consequently, the null findings reported in this research could be significant to many science communication scholars, science education researchers, or social psychologists interested in public understanding of genetics. To make that contribution more clear, however, the author needs to attend to some theoretical and methodological issues in this present draft of the manuscript.

Those issues are:

- A clearer and stronger theoretical and/or empirical rationale for testing a softer treatment to chart the limits of the genetic interpolation effect.
- Clearly defined confirmatory and exploratory hypothesis tests which are then mapped onto a post-hoc power analysis to demonstrate that the trial was well-powered, and thus, that the null findings are significant.
- More care needs to be taken in interpreting the null findings. At present, the authors provide a partial explanation for the null findings with the idea of straightlining. However, by their own account this explanation might only capture 6-10% of the null effect. Thus, a more convincing explanation for the null effect is needed to guide future research.

In sum, the significance of a null-experimental finding should be judged on its ability to guide future research. Null findings, when well powered, should help researchers decide which paths NOT to go down. And, they should provide some theoretical reason for why such paths lead nowhere. At present, the manuscript does not accomplish this task. But, with major revisions it might be able to make this contribution. Thus, I recommend that the study be approved with reservations. If all of the issues outlined below in my critique of the piece can be addressed by the author, then I think this piece will make a significant contribution to the public understanding of genetics.

Theoretical issues:

I would have liked to see a more nuanced discussion about the possible mechanisms which link the reading of behavioral genetics research to genetic attributions of complex human traits. For example, if the genetic interpolation effect is actually based on anchoring and adjustment, then one would expect that individual differences in people's prior conceptions about the genetic basis of human traits might moderate the activity of the genetic interpolation effect.

For example, the author hypothesizes that “Some people may react to behavior genetics by thinking to themselves that “if genetics is strong enough to have a significant influence on this complex social trait, then its general influence on other social traits must be stronger than I had

imagined," thus causing them to infer greater genetic influence for other complex characteristics not mentioned in the news article content."

But, what if people already strongly believe that social traits are caused by genes. In this situation, a ceiling effect might exist, thus preventing the statistical detection of the genetic interpolation effect. Under a ceiling effect model, we would expect a Treatment x Prior genetic belief interaction, where those who do not attribute social traits to genes before reading are affected by the experiment and those who do are not impacted by treatment. Conversely, it could also be the case that those who are generally averse to the idea that genes cause human social traits are inoculated against any impact that reading about behavioral genetics might have on genetic interpolation. Thus, the Treatment x Prior genetic belief interaction on the genetic interpolation effect could be the opposite of what I have previously stated.

The point is that prior knowledge and beliefs could have washed out the treatment effects and produced the null findings of this experiment. And, there is a large body of reading comprehension research which shows that the meaning people construct from readings varies with their prior knowledge. Hence, to interpret these null findings some discussion of the impact of prior knowledge is needed both in the theoretical motivation for the study and during the discussion. Indeed, the authors allude to this need when they discuss an unexpected finding in their results. They state:

"Noticeably, the results also present an unanticipated finding: compared to Group 1, Group 2 and Group 3 show lower average genetic attribution for natural hair style. The genetic interpolation hypothesis offers no explanation for this phenomenon, but something else may be at play here."

Perhaps the explanation is that individual differences in people's prior beliefs caused the null effect? The authors partly explain this finding with straightliners who varied significantly in proportion by condition. However, only 6-10% of straightliners could be inferred from their responses in each condition. So, the above issue – the issue of prior knowledge and beliefs affecting how people constructed meaning from the texts – might help to explain the other part. I would encourage the authors to analyze and report any findings they have (or have not reported) which explore how responses in the baseline data help to resolve the mechanism for the null results.

Methodological issues:

Because the researchers apparently did not measure prior knowledge of genetics or genetic beliefs prior to reading, then this could also be discussed as a methodological limitation of the present experiment.

The theoretical and empirical rationale for testing a shorter treatment given in the introduction is not convincing nor is it compelling. For example, the authors state, "The purpose of this study is threefold. First, the main goal is to test if the persuasion effect (H1) and the genetic interpolation effect (H2) also emerge following exposure to a softer treatment. Addressing this issue is necessary to better capture what kind of message can trigger the genetic interpolation effect". If this study is telling us anything about the world, then it should tell us what it is actually modeling in the world through the experimental design. For example, is the shorter reading on behavioral genetics more akin to what science students might read in a textbook, and thus, worth

investigating? Or, is it the kind of message that people might come across on PubMed? In short, why is the shorter reading important to investigate?

There are four conditions and 2080 participants. Given that the main finding in this study is a null finding, some care and effort needs to be taken to explain the a priori power analysis and post-hoc power analysis for the trial. For example, if the experiment is underpowered, then the significance of the null finding is diminished if not destroyed.

This issue is exacerbated by the multiple comparisons in the study. With four groups in the study there are three comparisons that can be made with the control group. Then, there are three different measures. So, in sum total there are at least nine-different statistical tests that could be run to test hypotheses. The authors should outline which of these tests are the confirmatory tests and which are the exploratory tests. Alpha values, and hence p-values, should be adjusted to account for Type-I error on the confirmatory tests. And, then, based on the actual effect sizes, and the adjusted alpha criterion, the authors should report what their post-hoc power was on each test. If the reach 80% power and their finding is still null, then such a null finding, in my mind, is worthy of indexing. However, if the authors were underpowered, then the contribution of these findings to the research community is less clear.

If we take the authors at their word, then with 2080 people in their panel and a response rate of 62.6%, there are about 1300 people enrolled in the experiment. If we divide that number in half ($n = 650$), that is the amount of people present in any single comparison with the control. Then, we can divide $0.05/3$ to account for the three different variables per treatment-control contrast. If we assume there is no baseline data which would improve the precision of the estimate by increasing the R^2 in the models, then any single treatment-control contrast could detect an effect size of $d = 0.25$ or greater. If we, instead, adjust alpha to account for all possible treatment control contrasts on all variables (i.e. 9 tests or $0.05/9 = 0.0055$), then effects of $d = 0.28$ or greater should be detectable. So, the question is, how big were the effects. Unfortunately, the standard deviation for the control group is not reported, nor are the pairwise effect sizes. If the authors provided that information, then the readers would have more confidence that the null effects are significant and merit indexing.

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Are the conclusions drawn adequately supported by the results?

Yes

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Author Response 08 Dec 2016

Alexandre Morin-Chassé, Université de Montréal, Montréal, Canada

M. Donovan, thanks for your careful reading of this paper. Your suggestions and critiques will help get the most out of this study. I will wait until I receive additional reviews and devote some time in January to work on writing a revised version which will address all of your concerns.

Competing Interests: No competing interests were disclosed.

Comments on this article

Version 1

Author Response 06 Feb 2017

Alexandre Morin-Chassé, Université de Montréal, Montréal, Canada

Dear reviewers, this is just a short message to inform you that I am currently working on writing a revised version of my paper that will address your reservations. I expect to submit this second version during the last week of February. Thanks for your insights and your patience.

Competing Interests: No competing interests were disclosed.

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