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# Believing in both genetic determinism and behavioral action: a materialist framework and implications

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**Abstract**: A disparity exists between studies reporting that genetics discourse produces deterministic or fatalistic responses and studies reporting that the majority of laypeople do not hold or adopt genetically deterministic views. This article reports data from an interview study (n = 50), and an interpretation of those data grounded in materialist understandings of discourse, that explains at least part of the disparity. The article employs a detailed reading of an illustrative transcript embedded in a quantitative content analysis to suggest that laypeople have incorporated two sets of public discourses—one that describes genetic causation and another that describes behavioral causation. These different discourse tracks are presumed to be encoded in different sets of neural networks in people's minds. Consequently, each track can be articulated upon proper cueing, but the tracks are not related to each other to produce a discourse for speaking about gene–behavior interactions. Implications for the effects of this mode of instantiation of discourse in human individuals with regard to genes and behavior are discussed, as well as implications for message design.

**Keywords**: genetic determinism, materialism, neural networks, public understanding of genetics

## 1. Introduction

There has been widespread and persistent concern that the keen attention given to genetics research in the past forty years will produce deterministic understandings of human actions and motivations as being grounded primarily or exclusively in biological factors that are not amenable to individual influence or control. Both critical analysis and empirical research have supported the idea that genetic understandings might be coterminous with genetically deterministic understandings. On the other hand, at least thirty years of polling research, along with detailed analyses of laypeople's understandings of genetics from focus groups, interviews, and experimental studies have shown that, in spite of extensive social attention to genetics, the attitudes of the general publics of Western nations continue to be rather persistently located in a middle-ground position, which grants some influence to genes, but does not see genes as sole actors.

This paper reports data from an interview study (n = 50), in the form of both an extended reading of a single transcript and quantitative content analytic data indicating that transcript's illustrativeness of the interviews. The interpretative frame employed is a materialist understanding of discourse and belief, which employs a distributed neural network model. We suggest that most laypeople

harbor two separate discourse "tracks" or neural networks for explaining health outcomes. One track explains outcomes based on genetics (or family history), and this track is generally highly deterministic. The other track or neural network explains outcomes based on behavior. The behavioral track is used more frequently and spontaneously by most people in articulating their sense of health causation. This track can also be deterministic with regard to the impact of past actions, but it is more frequently marked by uncertainty. Other explanatory tracks exist, including chance, God, and sociocultural factors, but are more rare. Laypeople shift from track to track in accounting for and predicting health outcomes, based on a variety of factors, including contextual cues, understandings of specific diseases, and motivations to protect health optimism or desired behaviors. Few people articulate an understanding of any relationship between these factors, but instead they operate on one track at a time. Different contexts elicit one track or another, giving rise to support for both streams of research on lay understandings of genetics.

To develop this explanation, we first outline the details of the two existing, apparently contradictory research streams. We then describe the components of materialist theories necessary to understand the discursive and mental processes that produce the data that have been identified by both these streams. We next describe our interview methodology, followed by our results. In the discussion section we explore some potential implications for lay understandings of health causation and for combating genetic determinism.

## 2. Conflicting understandings of lay beliefs in genetic determinism

The idea that discourse about genes is tantamount to genetic determinism has been backed both by critical analyses and by empirical research with laypeople. Although genetic determinism may not be uniformly defined among these works, early arguments linking awareness of genetics to determinism and to negative outcomes such as fatalism, discrimination, and failure to attend to social factors were developed by Conrad and Weinberg (1996), Duster (1990), Hubbard and Wald (1993), Katz Rothman (1998), Lippman (1993), Murphy and Lappé (1994), Nelkin and Lindee (1995), and Spallone (1992), among others. More recently, empirical research has been added to these critical analyses. For example, Senior et al. (1999) found that parents who perceived testing for familial hypercholesterolemia as genetic, as opposed to focusing on raised cholesterol levels, tended to see the disease as uncontrollable and more threatening. Similarly, in an analogue study, Senior et al. (2000) found that presenting risk information with genetics indicated as a cause (as opposed to no causal source) resulted in a perception of arthritis and heart disease as less preventable.

Taken as a whole, this research stream has strongly suggested that to talk about "genes" may produce reactions that are consonant with a biologically deterministic view of human outcomes and characteristics. Nonetheless, there is an equally well-developed second stream that indicates that when laypeople are explicitly asked

how they understand the role of genes in most human outcomes, only a minority give a genetically deterministic answer. The majority indicate that genes play a role, but that other factors play an equal or greater role. Public opinion polls show, for example, that mental illness is thought to be due to "heredity and genes" by the following percentages of people "completely" (10%), "mostly" (24%), "somewhat" (52%), and "not at all" (11%) (Singer et al., 1998). Responses vary by poll options and by particular outcome. For example, with regard to "whether someone gets cancer," the percentages assigning primary causality are 35% (heredity), 45% (environment), and 22% (both equally). However, almost no conditions assign heredity an exclusive or even overwhelmingly dominant role. Similar results clearly indicating mixed, rather than simply genetically deterministic views, have been found in focus groups (Bates et al., 2003; Parrott et al., 2003). Several experimental studies comparing responses to different messages or perceptions of disease have also found moderate attitudes about genetic causation (Condit et al., 2001), a positive association between perceptions of genetic causation and adherence to treatment plans (Senior et al., 2004), or no perception of differences in controllability (Marteau et al., 2004). Recent multinational reviews suggest the pattern among Western nations may not be widely disparate (French et al., 2001; Walter et al., 2004).

There are ongoing efforts to explore these conflicting and confusing results as a product of measurement challenges (French et al., 2005), as related to psychological factors such as threat avoidance (Senior et al., 2002), and as related to confidence in and understanding of tests (Michie et al., 2003), among other approaches. However, some of the conflict in these findings was resolved for us when we conducted 50 interviews with laypeople in the United States. As we will detail below, in the interviews the majority of participants exhibited what was to us, at first, a bewildering manifestation of blatant inconsistency. They would at one point articulate a stark belief in genetic determinism, only moments later to affirm that undertaking a particular behavior could prevent or contribute to delaying the problem. To come to an understanding of this strong pattern required that we check our assumptions about discourse processes and rationalistic assumptions about "consistency" and look for a theoretical framework that could account for the pattern. We found this theoretical account by linking materialist theories of discourse with Friedemann Pulvermüller's (2002) distributed neural network theory of discourse and the human brain.

## 3. A materialist theory of human discourse

Our initial understanding of our participants' comments as "contradictions" comes from a particular theory of language, one that holds that language is a representational device. From this theoretical perspective, statements are judged by whether they accurately mirror the world, and accurate mirrors of the world can never be mutually contradictory. Thus, the impetus of apparently contradictory statements must be understood as arising from a fault in the persons pronouncing

them. The appropriate response is therefore to correct the error in one or both of the statements offered by the individual (or institutions).

These idealist views of language have been slowly displaced by more materialist views of language over the past few decades. Surprisingly, perhaps, materialist views that are distinctive but consonant in key points come from both brain science and post-structural theories of the social level circulation of discourse.

Although it is not possible to offer a comprehensive summary here, materialist theories of language view language not as meanings (Derrida, 1976), but as physical entities that have effects (McGee, 1982; Johnson, 2007). Different discourse sets—sometimes described as "ideologies"—are presumed to have different social and individual effects. An often overlooked, but previously observed (e.g. Lanoue, 1992), facet of laypeople's relationship to such ideologies (as opposed to discourse of politicians or schooled experts), is that laypeople may have absorbed all or part of multiple discourses. Social theories easily explain how this happens on the social level—individuals are exposed to competing discourses from multiple sources. How such competing discourses can be maintained and managed within the individual is usefully explained by Friedemann Pulvermüller's account of human neural networks and their relationship to language.

Neural network theories posit that human behavior is selected by the activation of specific networks of neurons in the individual human brain by specific cues, including streams of words. Early theories were limited because they conceptualized each "node" in the brain as equivalent to a "concept," and, as Roskos-Ewoldsen et al. (2004) have noted, those conceptualizations did not adequately capture the dynamic nature of mental processing. These early, idealist-influenced theories are giving way to conceptualizations based on distributed networks, most notably that of Pulvermüller (2002). His theory conceptualizes "language" not as if one neuron were equivalent to one word or "concept," but as though the immediate "meaning" (i.e. effectivity) of each word/concept is constituted by the activation of specific portions of a complex network of overlapping neuronal connections distributed throughout many parts of the brain. Networks of neurons associated with any given word are built up through time by linkages of the word with varying stimuli. Thus "dog" is associated with the range of dogs seen and a range of experiences associated with those dogs, e.g. petting, getting bit, getting barked at, etc., but also with how "dog" sounds as a word and with the other words with which it is used (such as a verbally framed opposition to "cat") (for a precisely parallel critical theory see Kenneth Burke's account of language, as developed by Kuseki, 1988).

A key breakthrough in Pulvermüller's account is the discovery that the verbal network associated with a word (e.g. "dog") is not confined to the language areas of the brain. Rather, his research has demonstrated that the neuronal networks associated with words link up multiple parts of the brain. Thus, being cued with the word "wave" activates not only the language reception and production portions of

the brain (where presumably the audio/visual traces of the "word" itself might reside), but also the portion of the brain associated with the sensorimotor control of the arms. Words are thus instantiated in brains not as single nodes, but as neural networks constituted both of other verbal nodes and also of many other parts of the brain. An important implication of these linkages is that neuronal connections can be built by continuous verbal activation as well as by other kinds of experience in the world.

On this distributed network theory of language action, the streams of symbols that constitute communicative interaction, as well as all other kinds of interaction with the world, activate constantly shifting portions of a dense neuronal network. The portions of the network that are activated are based not only on the presence of a particular word, but also 1) on the particular set of words in the immediate word stream ("you dog" activates a different portion of the network than "good dog"), 2) on the intersection of these activated networks with strong, quasi-stable "emotions" and goals (such as self protection or love), and 3) on what have previously been called contextual factors, especially neural priming effects.

A key strength of the distributed network theory is the way in which it accounts for both "short-term" and "long-term" priming (Roskos-Ewoldsen et al., 2002). What Roskos-Ewoldsen et al. have called short-term priming is what cognitive theories and neural specialists have long referred to as "priming." This is the widely observed phenomenon that once a given word (or neural circuit) is activated, it is more likely to fire again (Pulvermüller, 2002). This is why once one has used a word in writing a paragraph, it seems to volunteer itself repeatedly thereafter. Because, as Pulvermüller notes "neurons firing together wire together" and vice versa (p. 20), getting neurons connected with any part of the model to fire at a threshold level can prompt a person to bring the mental model as a template to bear on immediately following phenomena. Both message production and interpretation may immediately thereafter be shaped by any of the components of the model.

The distributed neural network account also explains "long-term" priming, especially attitude accessibility. As a given set of components of a model are used repeatedly, the neural network associated with them is both strengthened and extended throughout the brain. The greater strength means it is more likely to sustain activation upon cueing. The extension throughout the brain means that it is more likely to be activated by more external cues.

Integrating social level discourse theories with Pulvermüller's individual level theory indicates that individuals selectively articulate the discourses to which they have been previously exposed within the society. An individual hears accounts, say of "family likeness" or "family history of disease" in family discussions, on the morning news, or in a crime drama. In high school, the individual learns that genes are deterministic entities—a single gene variation "causes" blue eyes or tongue rolling, for example. At the same time, however, the individual hears multiple accounts in an overlapping set of venues that indicate "smoking causes cancer,"

"exercise and good diet reduce heart disease," "too many sweets can trigger diabetes," etc. Hearing these statements frequently enough leads the individual to develop neuronal circuits that encode each of these two discourses. In Western society, however, the individual is unlikely to encounter discourses that explicitly link these two discourses (e.g. Cheng et al., in press) so that these two sets of neuronal networks are not cross-linked (they do not tend to "fire together"). Consequently, the individual carries around the ability to respond to and articulate these two discourses, but does not have a ready-to-hand discourse for articulating their relationship and does not have a predisposition to activate the two neural networks simultaneously. Which "track" is activated depends on a variety of factors, some of which we are elaborating here. The system is not a closed one, however, because when circumstances are right, the individual can invent new options or integrations, creating new articulations and beginning to "wire together" the two neural networks (and some of our participants did this).

This materialist account does not judge the rationality/irrationality of such responses, especially taken as a whole. It presumes that no language can fully and adequately "describe" the world, and therefore that all descriptions are descriptions for a purpose, with limited scope of applications and effects. The discourses about behavioral influences on health outcomes suffice for particular aspects of particular contexts, even if they do not capture the totality of the situation. The same is true for the statements about genes or family history. Given the impossibility of access to a totally comprehensive vocabulary, people make do by activating the discourse sets embedded in their brains that seem to provide as close a match to their motive states as they can. Focusing on incompatibilities in the statements across contexts is therefore well beside the point.

There are also methodological implications to this theory. Some methodologies ask questions only one way and at one time. Such a methodology may produce a single response that is taken as the "belief" of a participant. Because particular responses have higher attitude accessibility, and because similar cues may be used in repeat testing, these responses may even appear to be "reliable" in the technical sense of the term. However, if one asks similar questions with different cues, one may be able to elicit a variety of different answers reflecting the activation of different distributed networks (i.e. different discourse sets). These different responses might appear inconsistent, because there is no reason for different parts of a neuronal network to exhibit coherence with one another unless they routinely activate together. Different discourse "tracks" have been laid down in different contexts, and are elicited by different questions. Because our interview methodology addressed the same general topic matter through a variety of different discursive contexts, we unintentionally, but fruitfully, elicited the existence of two strong, but separated discourse sets for accounting for health outcomes.

## 4. Method of gathering lay discourse

To gain understanding of the concepts laypeople employ for articulating the

relationships among "genes," "behavior," and "health," we conducted a series of 50 interviews with low income White and African Americans. As members of the organization Southern Center for Communication, Health and Poverty (SCCHP), it is our priority to centralize the discourses and health needs of low income individuals. In conventional studies, a "general" population survey is sought, but using "general" methods usually produces samples that are deficient in representing low income individuals. Most health care research thus ends up further marginalizing those in poverty. To contribute to redressing this imbalance, we use methods that centralize low income participation.

SSCHP defines "low income" using the Centers for Disease Control's categories for "poor" (below the federal poverty level) and "near poor" (100–199% of the federal poverty level), here operationalized as a household income of less than $US35,000. We focused on White Americans (13 women, 10 men) and African Americans (12 women, 13 men; also 1 "other" woman and 1 "other" man) because these are the two largest demographic groups in our region. We divided our recruitment between Atlanta, GA, USA which provides an urban environment and rural counties in the health district surrounding Augusta, GA, USA to gain diversity in population density and lifestyles.

Forty participants were recruited by ORCMacro, a research firm with extensive experience in low income and minority research. They recruited participants by going to locations where low income individuals were present and inviting them to interview through a one-onone approach (i.e. the Salvation Army and low income apartment complexes). Subsequently, members of our research team recruited an additional ten persons from the rural area to increase the number of younger individuals participating in the study.We recruited these individuals by going to barber and beauty shops, asking the management if they would be willing to allow on-site recruiting, and then coordinating with on-site management to approach staff and patrons in off-peak times. Participants were provided an honorarium of $US75 for a one-hour interview. Over 90% of participants in all venues approached agreed to participate. In all cases but six, interviewees were matched by ethnicity with the interviewer.

Interviewers received between two and four hours of training on the interview guide (depending on prior experience). The guide asked the same set of questions on three separate conditions, "heart disease," lung cancer, and "adult" diabetes. These were chosen both because of their severity and familiarity to lay individuals, and because previous research suggested that there might be differences in the way laypeople understood these three conditions. By focusing on a range of common diseases, we could reduce the chances of mistaking the understandings of one disease for a general model of disease causation. Heart disease and diabetes are key targets of current initiatives by medical genetics research to expand the genetic paradigm from single gene disorders to "complex common diseases." The complexity is presumed to lie in the separate contributions of multiple genes and multiple non-genetic causal factors and their interactions. Some contributions of

gene–environment/behavior interactions have been identified for lung cancer, though previous research suggested that laypeople might not have used such an understanding in their accounts.

The interview guide therefore first asked participants to identify someone they knew with heart disease. Participants were asked what they thought caused the disease, in order to elicit unprompted responses with regard to behavior, environment, genetic or other causation. They were then asked for other cases (to ensure that their response was not specific to a particular person, and that all potential individual models manifesting attitudes were included). After elaboration of those ideas, they were prompted to consider the relationship of genes and environment through a scenario (quoted below). The reviewer then turned to the next disease. After all diseases were discussed, the participants were asked to describe their sense of their own health risks. Audiotapes of interviews were transcribed and then were corrected by a second auditor from the team.

While order effects are of concern given this design, the lack of substantial size order effects is supported by the fact that exposure to the genetic account in the heart disease case is followed by the lowest levels of genetically based accounting in the lung cancer case, with a subsequent increase in attribution to genetics in the third case of diabetes. If there are order effects, they appear not to be of a magnitude relevant to the conclusions made.

## 5. Data analysis procedures

These interviews have been analyzed using individual qualitative readings checked by quantitative content analytic follow-up and counter-case examination. The team began with the goal of understanding lay discourse sets regarding the relationship of "genes and environment." In most cases, lay participants referred much more frequently to "behavior" than environment, so our analysis has become focused on "behavior" rather than what the literature generally perceives as the broader term "environment" (which somewhat awkwardly tends to include "behavior").

On the initial reading pass, the Principal Investigator (PI) tentatively identified the most striking feature of the transcripts as rampant contradictory statements by participants, specifically adherence to both genetically deterministic and behaviorally deterministic or behaviorally contributory accounts for health outcomes. The research team then developed a content analytic coding scheme to assess the generality and accuracy of the existence of the perceived elements of this "contradiction" within the transcripts. This content analytic scheme had multiple parts, but reported here are the appearance of the categories "Gene Dominated," "Behavior Dominated," "Gene–Environment Separated," "Other Dominated" (e.g. religion, chance, social structure), and "Gene–Environment NOT Separated" (hereafter we use the term "behavior" instead of "environment," except where we refer explicitly to these coding categories). Coding was done at two levels. The first

used the talk turn as the unit of analysis. Each talk turn was assigned a code, and all specific uses of gene or behavior are captured in this level of analysis (which is not reported here, but which is relevant with regard to the relationship between the detailed analysis of the transcript and the global coding). The second level used the participant in a particular section of the interview guide as a unit, with six units per participant, one for each disease before the interviewer introduced the scenario which cued explicit consideration of gene–behavior relationship and another for each disease after the introduction of the cueing scenario. Units were separated in this way to allow detection of variation by disease and related to explicit cueing of consideration of gene–behavior relationships. Inter-coder reliability was deemed acceptable for any category after a trial coding resulted in a percent matching greater than .70. The maximum number of training runs before achieving this level of agreement was three.

This article centers on a detailed qualitative analysis of one transcript, with reference to the content analytic results that indicate that this interviewee illustrates the majority understanding among our sample. This transcript is not typical of our interviewees in all respects, merely illustrative with regard to the dynamics of the specific features identified, i.e. the shifting from a behavioral to a genetic account based on cueing, disease discussed, and motivations often dealing with a self or other focus. This transcript was selected primarily because it tends to have the clearest and shortest examples for presentation (other participants were wordier or excessively terse in one or more sections). The transcript was selected after a "challenge" or "counter-case" reading in which the PI sought to negate the newly formulated case-theory by challenging it against the transcripts one by one. Only three transcripts so consistently emphasized behavior-based health causation that they were deemed to not fit the type described here. Further checking of the appropriateness of the account written here was provided by team discussion of the PI's reading and interpretation as represented in multiple drafts of this paper.

The strategy of presenting a single transcript was selected for representing the data because it was through the process of working through interview transcripts one by one and trying to explain and understand the discourse produced by each participant at each stage of the interview that the interpretation based on the distributed network theory emerged. The reading of the transcripts congealed the theoretical formulation even as the diffuse elements of the theory began to make the texts explicable.We believe reproducing as much of this reading experience as possible is the best way to indicate the accuracy of the interpretation and the potential utility of the theory.

## 6. Results and interpretation

Both qualitative and content analytic procedures indicated that most participants do not have a mental model of a relationship between genes and health behaviors in producing health outcomes (which we had been looking for). Instead, individuals

tend to have separate mental tracks for linking health to behavior and for linking health to genes. As Figure 1 indicates, prior to cueing by the gene–behavior relationship scenario, the majority (63%) of participants used a behavior-dominated causation model. A minority (21%) employed interspersed accounts describing genes and behaviors as causal factors without linking them. Smaller minorities employed gene-dominated (6%) and other dominated (3%) accounts, and no participants employed accounts predominantly guided by a model articulating a relationship between genes and behaviors (6% of transcripts did not have answers that were elaborated enough or clear enough to be codable). After cueing by the scenario, the accounts of causation articulated changed markedly, with the majority now adopting a gene–behavior separate model as their mode of accounting (51%). Gene-dominated accounts also increased somewhat (to 15%), but in spite of the explicit attempt to cue a gene–behavior relationship model through the scenario, only 1 participant adopted this as a dominant mode of accounting for disease causation in each disease (11% of the transcripts were not codable). We suggest that the shift from behavioral toward gene–behavior separated or gene-dominated accounts indicates that although the strongest discourse (i.e. dense and broad neural network) for most participants is the behavioral one, they are familiar with and accept the genetic account as well. When the genetic account is cued through the scenario, it is activated and they alternate between it and the behavioral account. However, participants either do not have or do not agree with a gene–behavior relationship account and so cueing cannot activate such an account.

To account for the on-its-surface contradictory appearances of gene and behavioral accounts, in the content analytic assessment we also explored whether the gene-based as opposed to behavior-based tracks were used more frequently in association with self than others. That tally shows more attributions both to behavior and to genetics for others. Participants talk more about others than about themselves (in large part due to interview design, because there are three or four sections about others and one about the self, which comes at the end, when fatigue may set in). Additionally, both behavioral and genetics accounts are more commonly assigned to others than to self, because participants express the widely documented optimistic bias toward their own health (discussed in more detail below).

These general trends—the predominance of a two-track model, the variation by disease, and the tendency to avoid describing anything as predictive of negative future health for self—are instructive about the patterns of understanding that people bring to bear. They risk, however, obscuring a key element of lay understandings of the relationship of genes and behavior to health—its instability and variability—which is heavily responsive both to neural priming effects and to the interaction of these effects with a range of personal goals. Reading through a single participant's many answers to questions about genes, behavior, and health will illustrate the way in which different responses are produced by the interactions among particular contents (e.g. disease and self vs. other), non-articulated goals, and immediate priming.

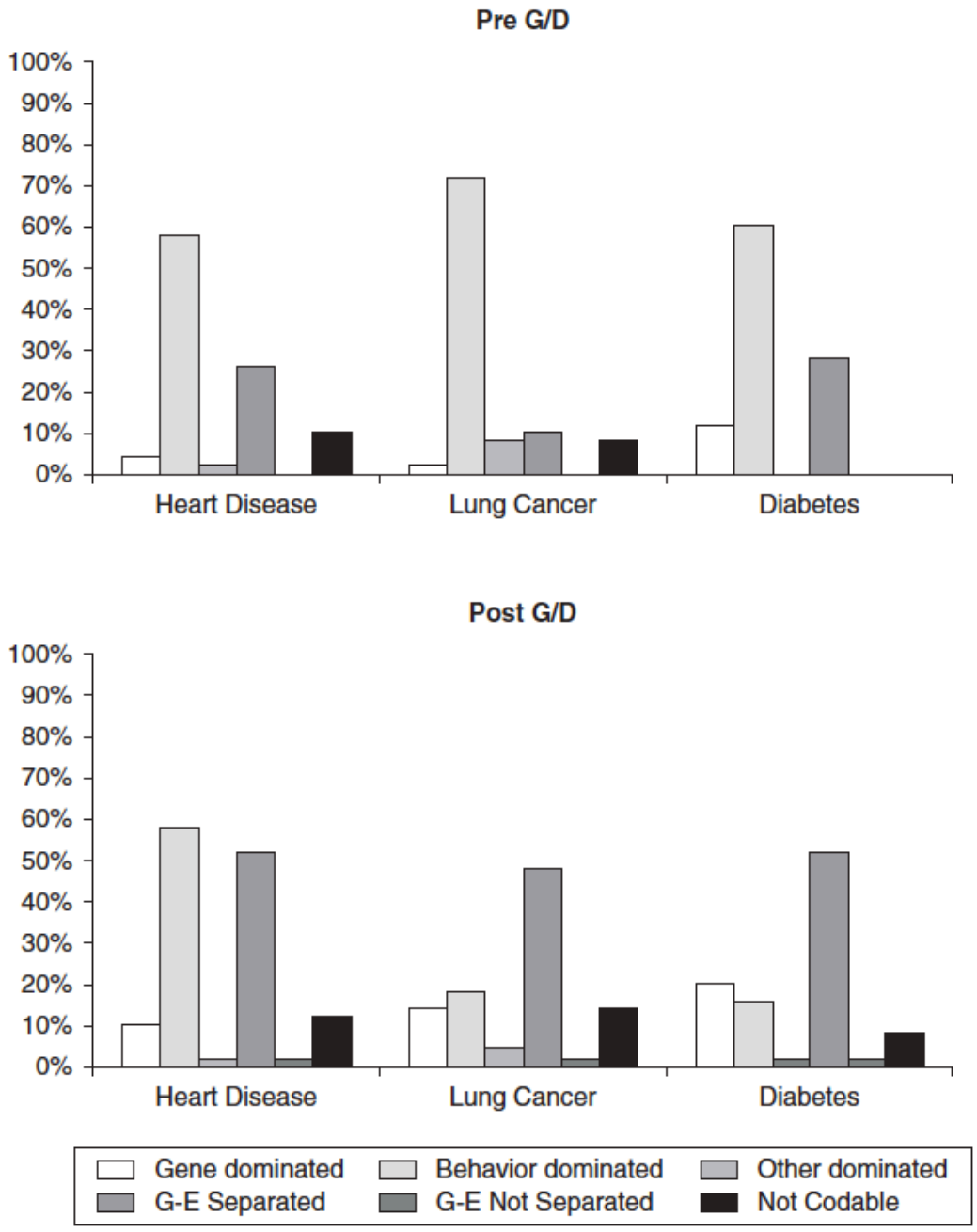


Figure 1. Global models

*A detailed example: participant #56*

*P56 on heart disease*

Participant 56 is a low income African American woman with a high school education who lived in our more rural recruiting area. She responds to the first

question of the interview by describing her grandfather's heart disease. She first attributes the cause to "You know, older people, like, eat the wrong food, like fat back, and all that kind of food, they eat the wrong foods and it's bad for their health." When asked for other causes, she also cites stress, then drinking, and then smoking. This is a highly common way of answering the heart disease causation question among our interview participants—to list behavioral causes, especially food. Nonetheless, when the moderator then asks about other family members who might have had heart disease, the participant responds, "I guess hereditary because there's a lot of people in my family got heart disease …"

This participant's spontaneous, unprompted account of heart disease is behavior based, but when genetics is raised as a causative factor, she endorses that factor as well. This is typical of our participants. A neural network based approach would say that she has developed verbal networks for both accountings based on behaviors and based on genes, but that the behavior-based network is broader and/or stronger. Like most of our participants, she therefore gives that account first with regard to heart disease. She is, however, familiar with the genetic account and not opposed to it, so that she is capable of moving back and forth between these accounts with relative facility. For example, when asked if there are other causes to a particular person's heart disease, whose disease she has defined as "hereditary," P56 responds with "Eating the wrong food, cholesterol, and stress, because he gambled a lot."

When the participant turns to discuss herself, she manifests substantially greater uncertainty. When asked how she could personally avoid getting heart disease, she says, "I really don't know, because a lot of sickness in our family, it just goes—seems like it go on and on with the generation." She does not spontaneously offer behavioral modification as likely to result in changes in her own health outcomes. Nonetheless, when the moderator asks again, "What could you do not to have heart trouble," she responds, "Just keep up on my doctor appointments, and just take care of myself." For herself, she is neither willing to assert a genetically deterministic position, nor willing to assert a strong efficacy of behavioral interventions. She temporizes with a weak acceptance of behavioral interventions. Arguably the stronger emotional involvement (based on desire to preserve herself) activates a broader neural network and she also envisions recruiting medical professionals to her case. Throughout this reading, we will suggest potential motivations such as this one.We may not be correct about what drives the shift in any particular case. Our point, however, is merely to draw attention to the shifts and to suggest that there are plausible motivations for such shifts, rather than mere randomness.

Another twist occurs when the moderator then poses the "Gene and Doug" scenario, which reads as follows:

> Okay. Imagine two people fromdifferent families. Gene has the gene for heart trouble, Doug does not have the gene. If they both stopped smoking, increased their exercise and had a better diet, which one would decrease

their chances of getting heart disease the most?…

She says that Gene will decrease her risk more, because "Doug might not, he may think, you know, that he don't have trouble with his heart, he probably don't think he would ever have trouble with his heart, and Gene, she just got to be cautious with hers." In the context of having just talked about her own perceived hereditary risk, P56 now reads hereditary risk as information that allows one to take preventive action—information Doug lacks, but Gene has access to. The bleed-over from the immediately prior discussion of herself is also supported by the fact that she interprets "Gene" as a female name, which is atypical of our participants and which further suggests she is analogizing "Gene" to herself, as a result of the short-term priming effects.

This trail of responses indicates the way in which the specific responses that the individual gives are contingent on what has previously been discussed (primed) and on the way that activation interacts with specific motivations (e.g. whether one is moved to protect a positive sense of one's future or protect valued habits).When conflicts arise, one may not simply give the answer from the primed neural network. Instead, one "invents," in this case a novel interpretation of the value of the genetic condition that focuses on its value as information that enables action rather than as a deterministic cause (hence the open nature of the neural/discursive circuits).

P56's response to the Gene-Doug scenario also manifests uncertainty. When the moderator repeats the question, she says, "You never know" and laughs.

*P56 on lung cancer*

The interview next moves to the discussion of lung cancer. In contrast to her responses to heart disease, the participant accounts for lung cancer exclusively through behavior. When asked why the model individual got cancer, she says "smoking." When asked whether there might be any other cause, she says, "That I'd say hereditary, because my aunt had it, they said my grandfather had it, but I don't think so." When the moderator asks why she doesn't think it was hereditary, she continues to resist the description of this cancer as hereditary, even while reproducing that articulation, "I don't think so.You can't just judge a person just looking at them because your sister had it or whatever, and they had the same symptoms or whatever, and my aunt … three of my aunts had cancer."

P56 has firmly assigned the primary, apparently the only, cause of lung cancer to the behavior of smoking, even in the face of her awareness that others think the disease has a hereditary cause and in the face of the fact that the people she is talking about manifest a hereditary pattern. She also explicitly says that you can avoid getting cancer by not smoking (some other participants deny that by emphasizing that you can't avoid second hand smoke or environmental toxins). However, this does not mean that P56 is reasoning from a fundamentally non-deterministic frame, because

she also insists that someone who has smoked cannot avoid getting cancer by quitting. When asked about the value of quitting, she says, “No, because it’s probably settled in his lungs already.” This answer not only manifests a deterministic future, but it also comes close to fusing the cause of a disease with the disease itself, as participants appear to do when they attribute genes as causation. The smoke/disease has “already settled” in. Participants are more ambiguous about this fusion in the case of behavioral causation as opposed to genetic causation, but the shared tendency toward fusion may reveal an underlying causal grammar.

*P56 on diabetes*

When discussing the third disease, diabetes, P56 identifies several of her family members who have the disease. When asked about the cause, she says, “I think it was hereditary.” On probing, she repeats this answer. She is much less certain, however, about whether or not this genetic causation means that they could have avoided getting the disease. The first time she is asked, she says “Yeah, because all of them eat, like, that fattening food, like, fat back and collard greens with the meat cooked in them, and everything, they put the meat in it, and they eat a lot of bread and a lot of potatoes, and they eat lots of sugar.” However, when asked again, in follow-up, she says, “No, you never know, you know, because people develop, they’ll get—get diseases and stuff, and you never know, they just get it.” After further discussion, she is asked a third time and she says, “I don’t know. I don’t know.”

The trajectory of these responses seems to exhibit declining confidence in the value of behavioral inputs, induced perhaps by a leading interviewer or simply by additional consideration. However, when she is next asked about her son’s risk, she returns confidently to the behavioral framework: “Because they eat lots of bread. He eats food that’s bad, he doesn’t like vegetables and stuff, you have to make him like taste it.” She says that she could help him avoid the disease, “By making him exercise more and eat more healthier foods and don’t eat a lot of sugar.” When asked how long that would “put off” getting the disease, she says, “I think that would keep it elevated, keep it okay, with the food and stuff.” Thus, when addressing her son, her motivation to envision a positive future may override the immediate neural priming and she employs a more confident stance about the value of behavioral inputs. Recall that this is the opposite of her response on heart disease, where she became less confident about behavioral prevention when discussing herself versus others. This may reflect a stronger motivation to protect the health of one’s loved ones as opposed to protecting the valued habits of one’s self.

In a final variation, when the interviewer turns to the “Gene and Doug” scenario with regard to diabetes, P56 interprets the person with a “gene for diabetes” as someone who already has the disease, saying “No, they can’t do nothing about it if they already got the disease.” This deterministic equation of having the gene with being equivalent to already having the disease was surprising to us and relatively common.

Participant 56 thus offers an extraordinarily wide variety of articulations of the relationship of genes, environment, and behaviors to disease. These can be accounted for by postulating that she has two different neural networks, one of which consists of a set of assumptions and linkages with regard to genes (or "family history") and how these relate to health. The other neural network deals with health behaviors. Both tracks are based on a grammatical relationship between a cause (e.g. "smoking" or "genes") and an effect (disease or disease prevention). However, each track is linked more strongly with particular diseases, but even within these tendencies, movement among tracks occurs with regard to both neural priming effects and personal motivations (e.g. to avoid fear of the uncontrollable, to protect one's child, to avoid undertaking undesired health behaviors for oneself).

## 7. Potential impacts of the dual network system on understandings

It is quite clear from our data that laypeople know both the genetics account of health causation and also the behavioral account. They spontaneously articulate the behavioral account in most cases. At least in part, this may be due to the fact that the heritability component of these three diseases is relatively low, and it would be useful to repeat a similar study incorporating diseases such as sickle cell anemia that are understood as having a high heritability or breast cancer which may be misunderstood as having a high heritability. Nonetheless, in spite of that potential biasing toward behavior, participants tend to assent to the influence of genes in many contexts. When they are employing the genetics track, they tend to see it as highly deterministic. However, most rarely stick with that track for very long, switching instead to behavioral accounts. Behavioral accounts show a range of levels of determinism from highly uncertain to very deterministic (the latter especially when a behavior has been practiced for a long time).

Our examination of these features of the data using a materialist framework helps us to understand these not as merely contradictory statements demonstrating random irrationality, but rather as the deployment of different resources in different contexts. It also explains how both streams of research have presented apt characterizations as far as they go. For most people, genetic perspectives are tantamount to genetic determinism. The strong determinism of the cause–effect grammar may arise because of the way they have been taught about genes, because genes are invisible, because they mis-equate the gene with the disease itself (sometimes calling genes a "virus"), or because they know they can't change a gene to a more favorable configuration. This determinism is what is identified by critics and what manifests itself in particular contexts where priming effects or motivations favor it. However, this determinism dominates only when the genetic account is activated and isolated from other explanatory frameworks. Most (but not all) people are quite adept at switching away from the genetic account and adopting an account based in behavior. Consequently, other experimental designs elicit the behavioral track, and when people are polled for their opinions about health

causation, they do not give an exclusively genetic account, but rather provide an account that in some way totes up the contribution of genes and of behavior.

We can only speculate about the neurological mechanisms by which people get from the separate genetic and behavioral discourses to poll responses such as mental health is "somewhat" caused by genes and behaviors. We suggest that many people have a non-discursively articulated understanding or belief (call it an "intuition") that genes and behavior interact with each other. However, in these interviews (and in message-response focus groups we have conducted, data not reported here), most people do not appear to have a discourse set that enables them to articulate that relationship. By including poll responses such as "somewhat" or "both equally," polls enable people to "say" something about genes and behavior that they are not able to articulate in ordinary conversation.

A materialist analysis asks about the assessment of effects, rather than the consistency of meanings. In message-response studies we are exploring the question of effects more fully, and it may be that one positive effect of this two-track model is that it enables people to focus on what they can change when they are in positions to consider change. For now, however, it seems to us that there are also potential negative effects of the two-track model, arising not solely from the determinism of the genetic track, but also from the relationship between the two tracks.

*The incomprehensibility of the appearance of disease*

One potential problem with the combination of a deterministic articulation of genes operating with a second "track" devoted to behavior is that it appears to inhibit participants' ability to account for why some people get sick and others don't. Instead, health outcomes appear random. As P56 says, "Um, you know, because you never know what happens to a person, they probably can be sick this minute, and the next minutes they'll be well, and you don't know why." She and other participants cite examples of people who smoked and did not get cancer, or of people who have a family history of cancer but do not get sick, or who do get sick and do not smoke. No participants account for such variation by noting the interaction of genes and behavior or environmental factors. No one says, for example, "she smoked and didn't get cancer, but that is probably because she did not have a genetic susceptibility." Indeed, P56's use of this discourse illustrates active resistance to talk that places genes and environment in interaction, as the following dialogue illustrates.

> M: Okay. If someone has the gene for lung cancer and they also smoke, does it make it more likely they'll get lung cancer?
> I 100: No, I can't say.
> M: Why?
> I 101: Because some people smoke their whole life and don't have lung

cancer.

French et al. (2000) have reported a similar lack of what they call "synergy" in the understandings of genes and behavior. Separated two-causation models do not provide comprehensibility to such outcomes. When any of multiple causes might be called in as an account for any given outcome (whether or not each is independently deterministic), then such "causes" cease to seem informative. Random chance or divine intervention seem to determine which "cause" provides an account. Uncertainty and shifting among causal accounts based on other motivations or in response to immediate neural priming is a predictable result.

*Determinism with (unrealistic) optimism*

Simultaneously, however, a two-track discursive net may also facilitate undue health optimism. The predominance of health optimism among the young and middle aged has been widely documented (Weinstein, 1987). Our participants show this same tendency. Recall that P56 identified herself as belonging to a family with a lot of heart disease, and, due to that, expressed uncertainty about whether or not modifying her diet would be effective in preventing the disease for her. Nonetheless, when asked about whether she would get heart disease, lung cancer, or diabetes, she says, "I don't think I'll have heart trouble."When asked, "why don't you think you'll have heart trouble," she replies "You know, I'm not gonna say that, because I was born with a heart murmur, so …" (answer trails off there and ends). In a world of randomly based uncertainty, where neither a genetic account (which spells likely illness) nor a behavioral account (which prescribes behavioral changes that might not be desired and may also spell likely illness due to past behaviors) offers desirable outcomes, why not simply hope that random chance will deliver you a good future? A foreordained future might well be conceived as a favorable one.

*Message implications*

The existence of the two-track configuration raises important issues for how one might design messages about genetics for the public or in contexts such as personalized genetic testing. For example, sources that favor a behavioral (or social or religious or other) account of human causation may not be able to promote their view successfully merely by repetitive broadcasting of their favored account. If, as this materialistic account suggests, learning new discourses does not displace old networks, but merely adds strength to existing discourse sets, then the result of message campaigns that add or intensify specific discursive networks may be the enhancement of confusion and uncertainty. Successful message strategies may thus require discourses that link existing networks together. Developing a gene–behavior or gene–environment interaction account thus might be more successful at eliminating genetic determinism than mere repetition of other causal sources. We would describe such a model as a "gene–behavior interaction" (G*B) model when it

presumes that the behaviors enhance the effect of genes, rather than merely working alongside them.We would describe an "additive" model as one in which individuals see the impact of genes as being added to behaviors.

At the social level, the network approach strengthens the suggestions made by those who insist that individually directed messages cannot effectively change behaviors on a large scale. If specific verbal neural networks are highly responsive to short-term priming, then people's behavioral choices are governed not primarily by a priori networks, but by local activation of networks in response to local stimuli (including messages). Thus, for example, the built environment that surrounds one, the schedule of routine activities, the food choices that are immediately available, and commercial messages encouraging specific patterns of consumption are going to be more determinative of health-related behavioral choices than the neural networks instantiated in people's brains, because the latter are inevitably multiple.

The suggestions offered in this article are, of course, highly tentative, but they do offer testable predictions. At the most demanding level, one might generate messages that provide linkages across dual tracks and assess their differential effectiveness as compared to messages that selectively reinforce either one track. Another route for controlled investigation would be to test the prediction that uncertain, highly fatalistic and highly optimistic articulations cooccur with two-track networks as opposed to single-track or linked networks (either in naturally occurring discourse or with prompted discourse).Whatever the results of future research, it is impressive and daunting to see how complex are the processing mechanisms by which laypeople make sense of the causative forces lying behind human characteristics. It is not surprising, therefore, that multiple research streams have been developed that highlight different aspects of these processes.

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