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Implications of Genetic and Other Biological Explanations for Thinking about Mental Disorders

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In recent decades, there has been a growing emphasis on understanding the role of genes in the etiology of mental disorders. This has been reflected in funding priorities for mental health research, which are apparent from an examination of investments made by funding agencies. It is also reflected in clinical care, as genetic testing has begun to be used by some providers, most often in the form of pharmacogenetic testing to inform the choice of which psychiatric medications to recommend for particular patients.[1] The use of personalized genetic information in clinical care for mental disorders is expected to become increasingly common as precision medicine makes its way into psychiatry.

The emphasis on genetic understandings of mental disorders has also taken hold in the public imagination, as is evident from the frequent appearance of headlines touting developments like the discovery that a “depression gene really exists” or that “schizophrenia is actually eight distinct genetic disorders,” or asking if any number of disorders is “genetic.” Unsurprisingly, in this climate, members of the public have increasingly adopted genetic attributions for mental disorders.[2]

Given the increasing adoption of genetic etiological beliefs regarding psychiatric disorders, a growing body of research has focused on trying to elucidate the effects that these explanatory frameworks might be having on how mental disorders are perceived by patients, clinicians, and the general public.[3, 4] Notably, genetic and other biomedical explanations of mental disorders have long been seen as a potential tool in efforts to destigmatize mental disorders, given the harshness of the negative attitudes about them that are widespread in society and the important negative clinical and social impacts of this stigma.[5] The question of whether genetic explanations actually reduce stigmatizing attitudes is an empirical question that researchers have attempted to address.[3, 4]

What underlies the assumption that seeing mental disorders as genetically caused will lead to a reduction in negative attitudes? The conventional wisdom has appeared to be that because the effects of genes are seen as falling outside of individual control, conceiving of mental disorders as caused by genes casts patients as blameless, thereby reducing stigmatization. Indeed, the results of experimental and correlational research have

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now robustly linked genetic and other biomedical explanations for mental disorders with reductions in the extent to which people are blamed for their own psychiatric symptoms[3, 4]

However, research examining the impact of genetic and other biomedical explanations of mental disorders has also suggested that they can have significant downsides. In particular, the most consistent negative effect of these kinds of explanations appears to be that they can lead to the assumption that mental disorders are unlikely to improve or abate.[4] This effect may even operate through the same psychological mechanism as the corresponding reductions in blame: if genetic and other biomedical explanations of mental disorders place patients' psychiatric symptoms outside of their control, it follows that they would be seen as less blameworthy but also as less empowered to overcome their psychopathology. Genetic and other biomedical explanations of mental disorders can also increase people's confidence in the effectiveness of biomedical treatments (i.e., pharmacotherapy) but decrease their confidence in the effectiveness of "non-biomedical" treatments (e.g., psychotherapy) [6-8].

Research among symptomatic individuals

The notion that biomedical explanations of mental disorders lead people to view *others* as unlikely to overcome their symptoms raises a parallel question with potentially significant implications: do such explanations also produce this kind of "prognostic pessimism" among people reflecting on the causes of their *own* psychiatric conditions? In recent years, a growing body of research has suggested an affirmative answer to this question.[9-12]

One reason why the question itself carries significant weight is that in the case of mental disorders in particular, patients' beliefs about their prognoses can actually affect their clinical outcomes. For example, research in the area of depression has shown that when patients adopt a pessimistic outlook about their chances of getting better, they tend to be less engaged in their treatment and experience less symptom reduction.[13] In other words, their pessimistic beliefs about their own prognoses can became a self-fulfilling prophecy.

Given the aforementioned research concerning the clinical importance of depressed patients' expectations regarding their own prognoses, much of my own research in this area has focused on major depression as a disorder of interest. In the first study of its kind, we recruited samples of U.S. adults with symptoms of depression and examined the relationship between their endorsement of genetic and biochemical causes for their symptoms and their prognostic beliefs about their moods.[12] We asked participants to rate how much of a role they believed each of several different causal factors played in bringing about their symptoms, as well as how long they predicted their symptoms would last. Our analyses revealed a significant relationship between biochemical and genetic attributions and more pessimistic expectations about symptom duration: the more people attributed their depressive symptoms to neurochemical imbalances or genetic abnormalities, the longer they expected to remain depressed.

While this finding was intriguing, one of its principal limitations was its correlational nature. That is, we had found that the people who most strongly attribute their depressive symptoms to genetic and biochemical causes also tend to be those who expect to be depressed the

longest, but this does not necessarily imply that people became pessimistic about their prognoses as a *consequence* of their biogenetic causal attributions for their symptoms. One approach that we have used to attempt to overcome this limitation has been to design studies that can experimentally manipulate whether or not people have a genetic explanation for their own psychiatric symptoms.

In recent work, we developed a technique to examine the consequences of *telling* people that their own symptoms are genetically caused. This approach has taken inspiration from the growing popularity of direct-to-consumer genetic testing services, which members of the public can use purportedly to learn a range of information about their genetic makeup, including their susceptibility to a variety of health problems.

In our work, we created bogus but credible-looking testing kits and mailed them to participants who were recruited online, in an attempt to simulate a direct-to-consumer genetic testing situation in which people would get the kits and then use them—and receive their "results"—in the privacy of their own homes.

When participants received their kits, they were told that they would undergo a biochemical test of their saliva, which would determine whether or not they carried a genetic predisposition to major depression. Each kit contained a small container of mouthwash, as well as a single test strip. In reality, the test strip was sensitive only to glucose, but participants were told it would detect their salivary levels of 5-Hydroxyindoleacetic acid (a metabolite of serotonin), which would reveal the extent of their genetic susceptibility to depression. Participants were told to rinse their mouths with the mouthwash, spit it out, then put their test strip under their tongue for several seconds. Unbeknownst to participants, the mouthwash contained glucose, so following these procedures caused an area of the test strip to change color from blue to brownish green. Based on random assignment, some participants were told that this color change indicated that they did have a genetic predisposition to depression, while others were told that it meant they lacked such a genetic predisposition. Because the content of this feedback was determined completely at random, there was no reason to expect any systematic differences (e.g., in genetic makeup, clinical beliefs, or pre-existing attitudes) among people who were told they had the genetic predisposition and people who were told they did not. Thus, we were able to conclude that any differences observed between the groups were specifically caused by the "genetic" feedback—an important advantage of experimental methodologies that use random assignment to conditions. To adhere to ethical guidelines by minimizing the duration of the deception inherent in this research design, we had to inform participants shortly after the "saliva test" (i.e., immediately after collecting the relevant data in each study using this approach) that the test was bogus and had not actually revealed any information about their genetic makeup. Thus, these studies can tell us only about the immediate effects of "genetic" feedback and do not provide any information about long-term effects.

In an early study employing this saliva test method,[11] we used the Beck Depression Inventory (BDI) to identify participants who reported symptoms consistent with at least mild depression. After they completed our sham saliva test, we measured their prognostic pessimism with a modified version of the Negative Mood Regulation (NMR) scale, on

which higher scores indicate more confidence in one's ability to cope with or respond effectively to depressive symptoms. Our analyses revealed that compared to people who were told that they lacked a genetic predisposition to depression, people who were told that they did have the genetic predisposition scored significantly lower on this measure of negative mood regulation confidence (the effect size was in the small-to-medium range and reflected mean scores that were about 10% lower). In other words, even though the so-called genetic feedback was determined completely at random, merely being told that their genes predisposed them to depression rendered people less confident in their ability to cope with those symptoms—a finding that is particularly disquieting in light of the fact that all participants in the study already reported elevated levels of depressive symptomatology. However, the study also included a group of participants who were also told that they had the genetic predisposition to depression but then watched a short educational video, designed to teach them about the idea that genes play only a non-deterministic role in causing depression by explaining, for example, that genes produce their effects by interacting with other genes and with the environment (including a primer on epigenetics), that treatment can lead to changes in biology, and that some risk factors for depression are under individual control. Our analyses revealed that among these participants, the negative effects of being told that they had a genetic predisposition to depression were successfully mitigated by the educational intervention.

In other recent research,[14] we have examined the question of whether people might see their genetics as so fundamental or essential to their self-concept that telling them they have a genetic predisposition to a disorder might actually lead them to recall having experienced more symptoms of that disorder in the past. In this work, we used the BDI as the dependent, or outcome, variable, as it measures people’s self-reported recall of having experienced depressive symptoms over the most recent two weeks. That is, the higher one scores, the more symptoms of depression one remembers having experienced over the prior two weeks. In the first of these studies, we again randomly assigned participants to one of three conditions using our saliva test method: some participants were told that they had a genetic predisposition to depression without watching the educational video, some were told they had the predisposition but also watched the video, and others were told they lacked the predisposition. When participants then completed the BDI, the participants who had been told that they were genetically predisposed to depression reported having experienced significantly more depressive symptoms over the past two weeks than participants who had been told that they were not genetically predisposed—even though the genetic predisposition feedback was actually determined at random, meaning that the difference resulted merely from receiving the genetic feedback itself. Furthermore, in this study, watching the educational video did not successfully mitigate the effect of the genetic feedback on BDI scores: the intervention produced no significant difference among participants who were told they had a genetic predisposition to depression. Instead, participants who were told they had the genetic predisposition scored significantly higher on the BDI than those who were told that they did not, whether or not they saw the intervention video. Indeed, the average BDI scores of those who were told they had the genetic predisposition were in the range of scores that are generally interpreted to signify the presence of at least mild depression, according to conventional clinical interpretations, while those of participants who were told they lacked

the genetic predisposition were low enough to indicate the absence of anything more than minimal depressive symptoms.

Upon obtaining these results, we wondered whether telling people that they were genetically predisposed to depression merely induced negative moods in them and whether this, rather than any fundamental change in self-concept after learning about their genetic makeup, could have been responsible for the elevated BDI scores in this group. As such, we conducted a follow-up experiment, which included an additional condition wherein participants were told that they were genetically predisposed to hypertension rather than depression. We reasoned that if simply receiving "bad news" from a genetic test was enough to produce elevated BDI scores, we would see such a pattern of results from participants told they had a genetic predisposition to hypertension just as in those who were told they had a genetic predisposition to depression. Instead, however, participants who were told that they were genetically predisposed to hypertension did not score any higher on the BDI than those who were told that they affirmatively *lacked* a genetic predisposition to depression, but once again those who were told they did have such a genetic predisposition to depression scored significantly higher.

These studies, which provide evidence that genetic test results about susceptibility to depression can actually affect people's self-reported recall of depressive symptoms, suggest that this kind of personalized genetic information may have quite profound effects on people, leading them to provide answers on self-report symptom measures that look like those of people who actually are suffering from depression. Indeed, if genetic feedback is powerful enough to cause people to rethink their past experiences and revise their recall to incorporate the assumption that they must have had experiences consistent with a disorder for which they have been told they are genetically susceptible, this may indicate that altering people's genetic self-knowledge may actually alter their self-concept in meaningful ways.

Research among clinicians and laypeople

The research reviewed so far provides converging evidence suggesting that negative consequences may result when individuals are led to understand their own psychiatric symptoms as resulting from their genes, such as by providing them with personalized genetic test results. Other research from recent years has also explored how mental-health professionals respond to genetic and other biological explanations for patients' symptoms. For instance, one set of experiments[7] examined how the empathy experienced by mental-health clinicians when reading descriptions of patients might vary as a function of whether the patients' symptoms were described in biological terms. Although one might expect that such a biomedical framing might facilitate empathy by casting patients as blameless and thus meriting sympathy, our results actually indicated that clinicians consistently felt less empathy when patients' symptoms were explained biologically rather than with psychosocial information about the individuals' life histories. These experiments carefully controlled for the influence of other aspects of the information provided (such as the specific symptoms in question and the identity of the patient), indicating that the difference in empathy truly stemmed from the psychosocial/biomedical distinction. Indeed, this difference emerged across a variety of disorders, ranging from those that are often construed as

highly biological (i.e., schizophrenia) to those that are typically viewed as highly non-biological (i.e., social anxiety disorder). These findings have potentially significant clinical and ethical implications, given that empathy is a crucial ingredient for the therapeutic relationship between patient and treatment provider—the so-called therapeutic alliance. Indeed, therapist empathy has actually been shown to be an important predictor of patients' clinical outcomes.[15] Thus, the notion that ongoing shifts in favor of genetic and other biomedical explanations for mental disorders could actually blunt clinician empathy should be cause for concern.

In addition to their effects on symptomatic individuals and clinicians, genetic and other biomedical explanations for mental disorders may also have a significant impact on how members of the general public perceive people with psychiatric symptoms. Such perceptions are important to examine, in part because widespread stigmatizing attitudes among members of the public toward people with mental disorders are known to impose significant burdens on patients' qualify of life.[5] Indeed, one of the motivations for promoting genetic and other biomedical conceptualizations of mental disorders has been its potential for reducing the extent to which people with mental disorders are blamed for their own conditions, which has been seen as a means of diminishing public stigma.

Addictive disorders have been highlighted as a case in which it could be particularly helpful to emphasize genetic and other biological explanations, as these disorders are often seen as moral failings rather than health problems, suggesting that reducing the blame ascribed to people with addictions could be an important way to reduce the social stigma that they face. However, the push to emphasize the role of genetic and other biological factors in the etiology of addiction has also been controversial, in part due to concerns portraying addictions as being completely determined by genes might seem to deny people of the personal agency that they need in order to overcome their disorders. Indeed, research has indicated that a sense of agency and self-efficacy can play an important role in a person's efforts to overcome an addiction,[16] and the suggestion that people with addictions are governed by their biology and are helpless to control their own behavior could itself be stigmatizing.

One recent study[6] examined how genetic explanations of addiction would affect people's perceptions of patients' blameworthiness, agency, and self-efficacy. Participants read about an adult man named Charlie and were randomly assigned to be told that Charlie had either a substance addiction (alcohol use disorder; AUD) or a behavioral addiction (gambling disorder; GD). Furthermore, participants were randomly assigned to either receive a genetic explanation of Charlie's addiction, in which they were told that it was caused by his DNA, or a "nongenetic" explanation, in which they were told that Charlie's addiction was caused by environmental factors. After reading their assigned description of Charlie, participants completed ratings of the extent to which they believed that Charlie was to blame for his problem, their perceptions of Charlie's agency to overcome his addiction, and their beliefs about how much control Charlie had over his behavior. Our analyses revealed that regardless of whether Charlie was described as having AUD or GD, he was seen as deserving less blame for his addiction when it was explained genetically than when it was explained nongenetically. However, accompanying their lower blame ratings, participants who were

told that Charlie's addiction stemmed from his genes also viewed him as having less agency to overcome it and as having less control over his behavior, compared to participants who received the nongenetic explanation. These results suggest that individualized genetic explanations for addiction may be a double-edged sword, portraying patients as blameless—which could be helpful for reducing stigma—but at the cost of potentially seeming to deny patients the agency to change their behavior for the better, instead casting them as helpless victims of biology. This would be of particular concern if genetic explanations of addiction are found to have similar effects among addiction patients and clinicians who treat them, given how clinically important it is, particularly in the case of addiction, for patients and their treatment providers to embrace the belief that the patient can get better.

Discussion

This paper has reviewed several recent studies examining the consequences of genetic and other biological explanations for mental disorders among symptomatic individuals, mental-health clinicians, and members of the general public. Unfortunately, rather than being straightforwardly beneficial, these biomedical explanations appear to have a number of important negative consequences. When symptomatic people view their own psychological problems through a biogenetic lens, this can lead them to endorse more pessimistic prognostic expectations about their own symptoms, and can even seemingly alter people's memories, leading them to recall having experienced more psychiatric symptoms than they might otherwise report. Moreover, compared to psychosocial explanations, genetic and other biological explanations for a patient's symptoms seem to lead treatment providers to feel less empathy for the patient. Additionally, although genetic explanations can reduce the extent to which patients are blamed for their disorders—an effect that might be particularly helpful in the case of addiction—this effect may be a "double-edged sword," as it can be accompanied by reduced perceptions of patient self-efficacy and agency.

An important conclusion from these findings is that pronouncements about the supposed failure to find negative psychosocial effects of personalized genetic health information seem premature. Importantly, the size and nature of the effects of genetic and other biomedical explanations for mental disorders may in some cases depend on the psychological motivations and pre-existing beliefs of the recipients of such explanatory accounts. However, much of the research reviewed above used experimental methods to render these kinds of factors implausible as potential confounding explanations for the results observed. Thus, although there may be some benefits of learning about one's own genetic health risks, and some measures may fail to register negative effects of this kind of information or suggest that any negative impact is transitory, it is far from clear that personalized genetic information in healthcare—at least in the area of mental health—is entirely benign. Indeed, people with mental disorders like depression may have cognitive biases that make them especially sensitive to negative information.[17] This may mean that they are at elevated risk of pronounced negative reactions to the kinds of personalized genetic susceptibility information that might have less powerful effects among other groups of patients. This highlights the importance of considering how genetic information might have unique effects among specific individuals or groups. Overall, the picture is decidedly not straightforward, as the positive and negative effects of genetic and other biological information may operate

through the same psychological mechanisms or even be “two sides of the same coin,” as when they lead to the perception that patients lack control over their symptoms and are therefore blameless but also powerless to get better. Although the potential negative effects of genetic information may be somewhat subtle or challenging to measure, they nonetheless merit the attention of researchers and clinicians. Indeed, patients and consumers likely stand to benefit most if we can develop an understanding of the range of consequences that personalized genetic feedback can have—including, ideally, how to intervene effectively against any potential harms—*before* this kind of information becomes more widely available as a part of routine healthcare.

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