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Impacts of Learning One's Own Genetic Susceptibility to Mental Disorders

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Abstract

Genomic research is rapidly advancing, and personalized genetic risk information about various mental disorders is likely to become readily available for many individuals. Although genetic profiling is intended to improve individually tailored treatments, knowing one's genetic risks or lack thereof can have unintended consequences. Upon learning that they have elevated genetic risks for a mental disorder (e.g., depression), people may become more pessimistic about their prognosis and misremember their symptoms as being more serious because they misconceive genes as immutable and defining of their identity. Upon learning that they are not genetically predisposed to a mental disorder (e.g., alcohol use disorder), people may underplay the downstream ramifications of the symptoms even when they are currently experiencing those symptoms. Possible interventions to counteract these problems and suggestions for future research are also discussed.

Keywords

genetic feedback, mental disorders, psychological essentialism, false reassurance, genetic testing

Suppose David enjoys a couple of glasses of wine with dinner every day, and more beer than he wishes during football games. One day he sends his saliva sample to a direct-to-consumer (DTC) genetic-testing company and learns that he is genetically susceptible to late-onset Alzheimer's disease. Subsequently, David may cut down on drinking and start jogging. Alternatively, he might not change his behavior, thinking that he cannot do anything about his genes. Suppose David instead learns that he has no genetic predisposition for alcohol use disorder (AUD). Could this welcome news now liberate him to drink more wine and beer? This article reviews recent studies examining the psychological impacts of learning about one's own genetic risks for mental disorders. (For reviews of implications for nonpsychiatric health conditions, see Bloss et al., 2011, and Hollands et al., 2016, and for reviews of implications for racism, see Donovan et al., 2021, and Roth et al., 2020.)

Although the extent of hereditary influences varies across mental disorders, there is little controversy over the fact that genes do play a role (Flint et al., 2020). Currently, the U.S. Food and Drug Administration (FDA) has approved DTC genetic testing for only a few diseases related to psychiatric conditions, including Alzheimer's and Parkinson's diseases. Yet, as stated by Morosoli et al. (2021), "Anyone who has access to their genome-wide data can access their individual polygenic risk scores for many mental health disorders, including alcohol dependence, depression, and schizophrenia" (pp. 341–342).

In this article, we first review the psychological impacts of learning that one has elevated genetic risks for a mental disorder and then review the impacts of learning that one does *not* have increased genetic risks for a mental disorder. We also discuss potential interventions to rectify misconceptions concerning genetic risk information about mental disorders.

Effects of Learning About Elevated Genetic Risks for a Mental Disorder

People may want to learn about their own genetic risks for psychiatric conditions, not just out of curiosity, but also to improve their quality of life by gaining this insight. But the benefits of learning that one has elevated genetic risks for mental disorders are not clear-cut.

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Some studies have found that genetic risk information may lead to beneficial changes in people's attitudes. For instance, college smokers who received testing for genetic susceptibility to nicotine dependency were randomly assigned to receive either no feedback (control) or feedback saying that they had either "above average" or "not above average" genetic risk (Lipkus et al., 2015). Those who were told that they had above-average genetic risk perceived their risk of becoming nicotine addicted to be highest and reported the strongest desire to quit smoking. However, these attitude changes did not result in behavioral changes; the quit rate after 7 days did not differ among the three groups, and the quit rate 1 month later was the lowest for students in the control condition and did not differ between those who learned they were at above-average risk and those who learned they were not at above-average risk.

Other studies also raise doubts as to whether learning that one has genetic risks for a mental disorder induces behavioral changes. In Chao et al. (2008), participants who learned that they were ε4-positive (which indicates genetic susceptibility to Alzheimer's disease) reported, 1 year later, a significant improvement in their diet, exercise, and use of medications (including vitamins), compared with those who learned that they did not have this genetic risk, but not compared with those who did not receive any genetic feedback.

One reason why learning about elevated genetic risks for a psychiatric condition fails to lead people to adopt healthier behaviors is likely that they have faulty lay theories of genetics. Specifically, many people appear to believe that genetic processes are more or less immutable (Dar-Nimrod & Heine, 2011), and relatively unaffected by environmental factors or individual behavior.

This misconception likely derives from *psychological essentialism* (Medin & Ortony, 1989), a belief that categories are based on an underlying, fundamental essence (e.g., Fido is a dog because he has "dog essence"). Essences also cause various characteristics of entities (e.g., Fido acts and looks like a dog because of "dog essence"). Essences for biological kinds (e.g., dogs, humans), in particular, are believed to be innate and immutable, largely unaffected by the environment (e.g., a tiger retains its "tiger essence" even if it is brought up by a man in Florida or even if it is surgically changed to look like a bear; see also Keil, 1989).

In reality, however, there are no immutable essences that define either social categories (e.g., gender and racial categories) or biological kinds (e.g., tigers, humans), given evolutionary biology. But humans' tendency to essentialize biological kinds is strong, emerging early in development (e.g., Gelman, 2003) across different cultures (e.g., Atran, 1998). Even for scientists, "it took more than two thousand years of biology, under the influence of Darwin, to escape the paralyzing grip of essentialism" (Mayr, 1982, p. 87). People's tendency to act as if categories have essences is called psychological essentialism, to distinguish it from metaphysical essentialism.

In contemporary society, genes could serve as essences for biological kinds (Dar-Nimrod & Heine, 2011; Nelkin & Lindee, 2010). People are taught that genes are innate and determine the characteristics of the kind. Once genes occupy the essence placeholder, this genetic essentialism can trigger misconceptions about genes. Outcomes of genes or genetic processes could be believed to be relatively immutable, and difficult to be changed by environmental factors. For instance, most people believe that one's height and eye color are genetically determined, and consequently also believe that there is not much one can do to change one's height or eye color.

Evidence for genetic essentialism in people's thinking about mental disorders comes from prognostic pessimism—the belief that mental disorders are resistant to treatment. The extent to which one believes that one's mental disorder has a genetic origin is positively associated with the extent to which one believes that mental disorders are untreatable or inevitable (e.g., Alper & Beckwith, 1993; Dar-Nimrod & Heine, 2011). For instance, the more individuals with depression attribute their symptoms to genetic factors, the more pessimistic they are about their own prognoses (Lebowitz et al., 2013).

These correlational studies allow researchers to examine people's existing beliefs about the symptoms that they are experiencing, but they fall short of providing causal conclusions. For instance, it could be that prognostic pessimism causes people to make more genetic attributions, rather than vice versa.

In other studies, researchers have used hypothetical vignettes to experimentally manipulate participants' thinking about the genetic bases of mental disorders. In one pioneering work, Phelan (2005) showed that when a character with a mental disorder was described as having genetic factors associated with the disorder, rather than as not having those genetic factors, participants judged the character's problems to be more likely to persist throughout the person's whole life.

Similar results were found in a study of participants' views on their own mental disorders (Lebowitz et al., 2014). Participants with symptoms of generalized anxiety disorder (GAD) received either no explanations or biological explanations (including genetic ones) for GAD in general. That is, the experimental manipulation was rather mild in that participants were simply told

that GAD can be genetically caused rather than that the participants themselves had genetic risks for GAD. Nonetheless, participants who received biological explanations judged that their GAD symptoms would last longer compared with participants who received no explanations.

Other researchers have experimentally manipulated participants' beliefs about their own genetic risk for a mental disorder. In these studies, participants' saliva samples were purportedly tested for the presence of target genes, and they received randomly determined genetic feedback (i.e., presence or absence of elevated risk). Although such studies involve deception, the methods closely mimic the experience of receiving results from DTC genetic testing, and because participants are randomly assigned to receive sham feedback, these methods also avoid confounds resulting from differences in real genetic status.

One of the first studies using this method found that participants who were led to believe that they had genetic risks for AUD reported feeling less control over their drinking than did those who were told that they did not have such genetic risks (Dar-Nimrod et al., 2013). In a study using a similar method (Lebowitz & Ahn, 2018), participants who had symptoms of depression and learned that they allegedly had elevated genetic risks for depression were less confident about their ability to deal with depression than were those who were told that they did not have elevated genetic risks.

Prognostic pessimism can have disastrous clinical consequences, as outcome expectancies affect actual prognosis (Krell et al., 2004; Rutherford et al., 2010). Thus, it is imperative to find ways to prevent prognostic pessimism, by correcting the underlying misconception about genetic essentialism. Lebowitz et al. (2013) found that directly teaching participants about the malleability of genes involved in depression (e.g., via interactions with environmental factors) reduced prognostic pessimism, including among participants with depression. Similarly, Farrell et al. (2015) found that compared with participants who received information only about the biological bases of eating disorders, those who learned about the malleability of biological factors associated with eating disorders showed more prognostic optimism and greater confidence in their ability to recover from eating disorder symptoms. In the study involving participants who had depressive symptoms and were told that they were genetically susceptible to depression (Lebowitz & Ahn, 2018), watching a short video explaining how the environment always interacts with gene expression successfully restored participants' feelings of agency in managing their depression in the future.

Although there are promising ways to counteract prognostic pessimism, recent studies have uncovered a more challenging consequence of learning that one is genetically predisposed to a disorder: distorted memory for one's symptoms. In one of these studies (Ahn et al., 2020), participants read two vignettes, each describing a depressed person. The first vignette varied across participants in terms of whether the protagonist had a genetic basis for depression. The second vignette was identical for all participants and described a different person's similar but more severe depression, subjecting the participants to memory confusion. Those who read that the protagonist in the first vignette had genetic risks for depression were more likely to confuse this first protagonist's symptoms with the more severe symptoms from the second vignette. That is, people were prone to interpreting genetically caused symptoms as being more severe than they actually were.

Memory distortion could also occur when participants think about their own symptoms. In the study concerning purported genetic risk for depression (Lebowitz & Ahn, 2018), after participants were given randomized feedback about their risk, they reported their levels of depression-related symptoms (e.g., sad mood, sleep difficulty) in the past 2 weeks using the Beck Depression Inventory-II, a well-validated measure. Although the true levels of their depression in the preceding 2 weeks were unknown, there was no reason to expect any systematic differences between the groups in their levels of past depression, given that participants were randomly assigned to the groups. Nonetheless, those who learned that they allegedly had elevated genetic risk reported significantly higher levels of depression—even exceeding the clinical cutoff used to diagnose major depression—compared with those who learned that they did not have elevated genetic risk for depression.

Prior to responding to the Beck inventory, some participants who had learned that they were genetically predisposed to depression watched the intervention video explaining the malleability of genes. Unfortunately, this video was not effective in preventing the false memory (Lebowitz & Ahn, 2018). One possible explanation is that this intervention is about what people can do going forward (e.g., exercising, socializing to reduce illness risk), and therefore, it might be effective only when people consider future behaviors, but not when they consider the past. Given that almost all mental disorders are diagnosed on the basis of retrospective recall of symptoms, the finding that genetic information can distort memory of symptoms is particularly disturbing, as it may lead to overdiagnosing mental disorders as individuals' gain increasing access to their own genetic information.

Effects of Learning That One Does Not Have an Increased Genetic Risk for a Mental Disorder

Less studied are the potential harms of learning that one does not have an increased genetic risk for developing a certain mental disorder. Consider the earlier example of David, who enjoys daily wine with dinner. Suppose the DTC results indicate that David has no genetic risks of AUD for the variants that were tested. Could David now feel invulnerable to the adverse consequences of drinking?

When 23andMe was seeking FDA approval for their genetic testing for health conditions, they considered the possibility that people may experience false reassurance when informed that they lack a certain genetic variant. Thus, their FDA-approved procedure for informing consumers that they do not have the specific cancer-associated variants in the BRCA1 and BRCA2 genes, for example, involves explaining that the results do not mean that they have no risk of developing breast and ovarian cancers, as there are nongenetic causes and genetic variants not examined in a specific test. However, whether people actually experience false reassurance upon receiving genetic results indicating a lack of genetic variants associated with a disorder and whether such debriefing is sufficient had not been empirically established before FDA approval was granted.

In a recent study (Ahn & Perricone, 2022), we empirically examined false reassurance using AUD as a target condition. The type of reassurance we tested was not just the type that can be considered justifiable. For example, it is justifiable for people who have learned that they lack genetic risk factors for AUD to adjust their belief about their likelihood of developing this disorder to some extent. Instead, however, we examined false reassurance about one's existing symptoms of AUD that occurs when one learns that these symptoms are not caused by genes. Participants were first instructed to imagine that they had AUD symptoms (e.g., spending a lot of time drinking and drinking more than intended) and to rate the seriousness and downstream risks of these symptoms (e.g., urgency of seeking treatment, interference with work and social functioning). Then, they self-administered a sham genetic test of their saliva, which was described as being sensitive to aldehyde dehydrogenase, an enzyme involved in alcohol metabolism. Next, all participants were told that the test results indicated that they did not have genetic risks for AUD. Then they rated the seriousness and downstream risks of their hypothetical AUD symptoms again, having learned that these symptoms would not be genetically caused. As these AUD symptoms were the same as those described before the provision of the sham genetic-test results, their perceived ramifications should not have changed. That is, it is not warranted to interpret a given set of AUD symptoms differently simply because one learns that they do not have a genetic etiology (at least, not according to current knowledge about AUD).[1] Nonetheless, after this feedback, participants discounted the seriousness of the AUD symptoms and were falsely reassured about the risks of those symptoms. Thus, the experience of learning that one does not have genetic risks for AUD could ironically become a risk factor for the disorder, as one may feel it is safe to continue or even increase one's drinking.

We also tested the effectiveness of the debriefing process currently used by 23andMe, which was designed to prevent false reassurance by emphasizing that environmental factors and genetic factors not examined in the test could still put someone at risk. Results were concerning, as we found that these materials were ineffective for those individuals who reported already engaging in problematic drinking behaviors (Ahn & Perricone, 2022). To address this problem, we developed additional educational materials emphasizing that once the symptoms of AUD are present, their ramifications should be taken as equally risky whether or not these symptoms were caused by genes (as formalized as the causal Markov condition; see Pearl, 1988, and Fig. 1). These materials successfully mitigated the false reassurance and the unwarranted discounting of the threats of AUD, including for those participants who were already engaging in harmful drinking.

Summary and Future Directions

Knowledge about one's own DNA is invaluable for precision medicine, but can be detrimental to one's mental health because of misconceptions about genes. We have reviewed two such misconceptions, genetic essentialism and false reassurance. Learning that one has an elevated level of genetic risks for a mental disorder can make one more pessimistic about one's prognosis and inflate one's memory for symptoms of the disorder because of genetic essentialism. Learning that one lacks a genetic variant responsible for a certain disorder can engender false reassurance about symptoms that one already has. Given these initial findings, there are several important areas for future research.

One is to examine whether attributing one's mental disorder to genetic origins elicits greater prognostic pessimism than attributing it to growing up in an abusive or impoverished environment. Although one's past cannot change, if present circumstances do change for

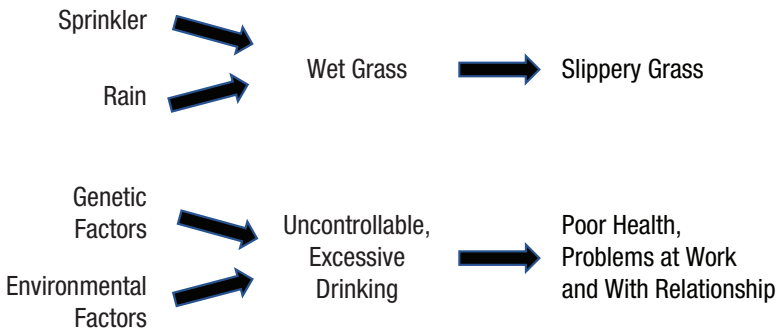

Fig. 1. Illustrations of the causal Markov condition as presented to participants in Ahn and Perricone (2022). According to this condition, when *X* causes *Y*, which causes *Z*, once the value of *Y* is known, the value of *X* has no bearing on the value of *Z*. For instance, as illustrated in the top panel, once the grass is wet, one can predict that the grass is slippery regardless of whether the grass became wet because of rain or a sprinkler. Likewise, as illustrated in the bottom panel, once a person develops symptoms of alcohol use disorder, the ramifications of these symptoms are the same regardless of whether the symptoms are caused by genes or environmental factors.

the better, the effects of a stressful childhood may be perceived as lessened. On the other hand, risk arising from genes may be seen as especially serious if the genetic influences are thought to be ever present.

Another question for future research concerns the durability of the effects discussed above. Because of ethical concerns, genetic feedback for mental disorders cannot be manipulated experimentally to observe subsequent changes in symptoms. Nonetheless, a possible long-term consequence of receiving information about one's genetic makeup could be engaging in behavior like that of someone who has the disorder, which could exacerbate otherwise subclinical symptoms (e.g., Tamir et al., 2007; Turnwald et al., 2019). If genetic testing for mental disorders becomes more available to the public, future studies can employ correlational methods to observe the long-term consequences of real-life genetic feedback.

Additionally, given that most people who undergo genetic testing for a certain mental disorder will learn that they do not have elevated risk, more attention needs to be paid to examining the potential perils of this feedback. For instance, future studies can test the generalizability of false reassurance as well as the efficacy of interventions based on the causal Markov condition (as in Ahn & Perricone, 2022).

Finally, going beyond studying ways in which genetic feedback can be misunderstood, further research should be devoted to redressing genetic misconceptions. First, it is still an open question as to whether the empirically validated interventions discussed above (e.g., teaching people about the malleability of genes or the causal Markov condition) would lead to actual behaviors that promote mental health. Second, additional intervention methods should be developed and tested. In particular, although it has been found that participants who learn they have elevated genetic risk for depression report more severe depressive symptoms compared with those who receive no such feedback (Lebowitz & Ahn, 2018), ways to counteract or prevent this memory distortion are yet unknown. Future intervention methods should also build from those already used by psychiatric genetic counselors (see Austin, 2020, for a review). Third, efforts should be made to disseminate empirically validated educational materials, especially through the DTC genetic-testing companies, because access to genetic counselors may still be somewhat limited (see Boothe et al., 2021).

Recommended Reading

Driver, M. N., Kuo, S. I-C., & Dick, D. M. (2020). Genetic feedback for psychiatric conditions: Where are we now and where are we going. *American Journal of Medical Genetics B: Neuropsychiatric Genetics*, *183*(7), 423–432. https://doi.org/10.1002/ajmg.b.32815. Provides a different kind of review on how genetic feedback for psychiatric disorders affects people.

Haslam, N., & Kvaale, E. P. (2015). Biogenetic explanations of mental disorder: The mixed-blessings model. *Current Directions in Psychological Science*, *24*(5), 399–404. https://doi.org/10.1177/0963721415588082. Provides a broader review of impacts of construing mental disorders in terms of biology, including both genes and brain abnormalities.

Lebowitz, M. S., & Appelbaum, P. S. (2019). Biomedical explanations of psychopathology and their implications

for attitudes and beliefs about mental disorders. *Annual Review of Clinical Psychology*, *15*, 555–577. https://doi.org/10.1146/annurev-clinpsy-050718-095416. Provides a thorough review of effects of biological construal of mental disorders on not only laypeople but also clinicians and individuals with mental disorders.

Transparency

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Note

1. It is possible that future genetic research will show that clinical outcomes are worse for individuals who have elevated genetic risks than for those who do not, and so there would be a rationale for treating symptoms not related to a genetic predisposition as less serious.

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