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Beneficial and Detrimental Effects of Genetic Explanations for Addiction

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Abstract

Background—Addictions are highly stigmatized and increasingly construed as biomedical diseases caused by genes, partly to reduce stigma by deflecting blame. However, genetic explanations may have negative effects, which have been understudied in the context of addiction. How the effects of genetic explanations might differ for substance addiction versus behavioral addictions is also unknown.

Aims—This study examined the impact of genetic explanations for addiction on measures of treatment expectancies, blame, and perceived agency and self-control, as well as whether these varied depending whether the addiction was to a substance or a behavior.

Methods—Participants read about a person (“Charlie”) with either alcohol use disorder or gambling disorder, receiving either a genetic or nongenetic explanation of Charlie’s problem. They rated how much they blamed Charlie for his disorder, his likelihood of benefitting from medication or psychotherapy, and how much agency and self-control they ascribed to him.

Results—Compared to the nongenetic explanation, the genetic explanation reduced blame and increased confidence in the effectiveness of pharmacotherapy. However, it also decreased the expected effectiveness of psychotherapy and reduced ascriptions of agency and self-control.

Conclusions—Genetic explanations for addiction appear to be a “double-edged sword,” with beneficial effects that come at a cost.

Keywords

Addiction; Genetic Etiology; Agency; Blame; Treatment Expectancies

Introduction

The 2014 National Survey on Drug Use and Health in the United States estimated that approximately 8% of Americans (i.e., roughly 1 in 12) had experienced a substance use disorder (SUD) in the past year (Center for Behavioral Health Statistics and Quality, 2015). The price tag of substance abuse in the United States—which contributes to healthcare

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expenses, lost earnings, accidents, and crime—has been estimated at more than \$440 billion annually (Office of the Surgeon General, 2016). Addiction also contributes to various medical problems and a host of social woes (e.g., family dysfunction, unemployment, homelessness, and violence).

In efforts to combat the severe negative consequences of SUDs, conceptions of addiction as a chronic disease rooted in genetics have been increasingly emphasized. The view of addiction as a disease with genetic roots has become a dominant perspective in the United States, favored by many scientists, practicing clinicians, and, increasingly, members of the general public (Deacon, 2013; Pescosolido et al., 2010; Reinarman, 2005; Wiens & Walker, 2015), with studies of addiction genomics becoming a priority for research funding (McGuire, 2016; National Institutes of Health, 2017). Some have argued that conceptualizing addiction as a disease will yield more effective (and less stigma-driven) substance-related social policy and better healthcare outcomes (e.g., Leshner, 1997; Volkow, Koob, & McLellan, 2016).

The notion that genetic explanations could help to temper the kinds of negative attitudes toward addiction that are widespread among members of the public is consistent with the tenets of attribution theory, which states that anger, blame, and punishment intentions are reduced when a person's stigmatized characteristics are attributed to factors that the individual did not cause (Phelan & Link, 2012). Indeed, even some research that has described biomedical explanations as harmful to social attitudes when applied to other mental disorders has nonetheless argued that they are beneficial when applied to SUDs (Schomerus, Matschinger, & Angermeyer, 2014), perhaps because one of the benefits of genetic explanations is their capacity to reduce blame, a prominent component of the stigma attached to addiction.

However, assumptions that genetic explanations for addiction will be universally beneficial may be misguided, as biomedical (e.g., genetic) conceptualizations could actually *exacerbate* certain negative views of people with addictions. For example, these conceptual frameworks may engender *genetic essentialism*—the misguided view that DNA contains the biological, largely immutable “essence” of a disorder, inevitably consigning people with certain genes to the fate of developing a particular health problem (Dar-Nimrod & Heine, 2011; Haslam, 2011). Although the essentialist view that addiction results deterministically from genes may deflect individual blame by countering the notion that people with addictions are simply choosing freely to engage in problematic behaviors, it may also promote the notion that people with genetically caused disorders lack agency or control over their behavior, and are thus helpless against their symptoms (Dar-Nimrod, Zuckerman, & Duberstein, 2013; Kong, Dunn, & Parker, 2017; Meurk et al., 2016).

Genetic conceptions of addiction could also engender pessimistic beliefs about the likely effectiveness of non-biomedical treatment approaches, due to *mind-body dualism*—particularly Cartesian dualism, or the erroneous perception that psychological and physiological phenomena are entirely separate (Kendler, 2005). Because people often tend to view the mind as separate from the physical body, genetic conceptualizations of addiction could lead to the perception that addiction resides in a person's physiology and that

treatments targeting psychological processes are therefore unlikely to be effective. Indeed, there is evidence—from research among mental-health clinicians, patients, and laypeople—that biological explanations for mental disorders are seen as incompatible with psychosocial explanations and can reduce confidence in the effectiveness of non-biological treatments (Ahn, Proctor, & Flanagan, 2009; Iselin & Addis, 2003; Lebowitz & Ahn, 2014; Marsh & Romano, 2016; Miresco & Kirmayer, 2006), although research of this type focused specifically on genetic explanations of addiction is lacking.

If genetic conceptualizations of addiction do lead to reductions in the personal agency or self-control ascribed to people with addictions and doubt about the effectiveness of non-biomedical treatments, this could have potentially significant negative consequences. Importantly, non-biomedical interventions—such as cognitive behavioral therapy and motivational interviewing—are indeed effective treatments for addiction (Hettema, Steele, & Miller, 2005; McHugh, Hearon, & Otto, 2010), including when offered in combination with pharmacotherapy (Loreto et al., 2017). However, patients are most likely to benefit from an addiction treatment if they believe that treatment is likely to be effective and feel confident in their own agency and self-efficacy (i.e., perceive themselves as capable of succeeding in their efforts to overcome their addictions) (Cropsey et al., 2014; DiClemente, Doyle, & Donovan, 2009; Kadden & Litt, 2011; Laudet & Stanick, 2010; Moos, 2007). Similarly, patients are likely to benefit from being treated by clinicians who expect the treatment to be effective and view their patients as possessing agency (Haque & Waytz, 2012; Joyce, Ogrodniczuk, Piper, & McCallum, 2003; Lebowitz & Ahn, 2016; Meyer et al., 2002). Thus, if genetic explanations—which are increasingly ascendant—yield perceptions of patients as lacking agency and as unlikely to benefit from non-biomedical treatments, such effects could actually harm clinical outcomes.

Additionally, although SUDs are perhaps the prototypical examples of addictions, behavioral addictions also increasingly are seen as worthy of clinical attention (Yau & Potenza, 2015). Etiological beliefs, as well as social judgments, can differ substantially for these two categories of addictions (Thege et al., 2015), yet research to date has not examined whether genetic attributions have the same effects across these two classes of addictive disorders.

The present study investigated how a genetic explanation for addiction would affect perceptions of the personal agency and blameworthiness of a person with an addiction, as well as expectations about the person's capacity to benefit from biomedical and non-biomedical forms of treatment. The addictions examined were alcohol use disorder (AUD), the most prevalent addictive disorder (Grant et al., 2015), and gambling disorder (GD), the first behavioral addiction to be formally recognized as a diagnostic category by the American Psychiatric Association (Mann, Fauth-Bühler, Higuchi, Potenza, & Saunders, 2016).

Methods

Participants and recruitment

Participants were 403 U.S. adults recruited using Amazon.com's Mechanical Turk (MTurk) service, which allows individuals to complete short tasks in exchange for payment

(Buhrmester, Kwang, & Gosling, 2011). Sample demographics are summarized in Table 1. Although recruiting participants via MTurk has the potential to introduce sampling bias in that MTurk users are not representative of the general population (Mason & Suri, 2012), the present study used experimental methods to investigate the effect of genetic explanations by randomly assigning individuals to different conditions, rather than survey methods intended to estimate the prevalence of particular opinions or attitudes among the general population. Moreover, the evidence suggests that MTurk respondents tend to be more diverse than typical samples used in psychology research (e.g., college students and non-MTurk internet samples), suggesting that compared to other widely used methods, recruiting via MTurk may actually reduce sampling bias and result in a more broadly representative sample (Buhrmester et al., 2011).

To advertise the study to potential participants, we posted a listing on the MTurk website stating: “Thank you for your interest in participating in this study. This study examines people’s attitudes and beliefs about human behavior. This study is being conducted by researchers at the NY State Psychiatric Institute. The study will help researchers understand how people react to certain kinds of information about the behavior of others. Your participation in this study is completely voluntary. You might find the questions upsetting. You may choose to skip any question in the study that you do not wish to answer. You will not be individually identified and your responses will be used for statistical purposes only.” Participants were provided with an email address to use if they had questions about their rights as a participant or were dissatisfied with any aspect of the study; no emails were received through this channel. The MTurk listing said, “If you consent to participate in this study, please click the ‘Continue’ link below to begin.” Participants who clicked the “Continue” link were directed to a website where the study procedures were administered using Qualtrics.com online data-collection software. Because participants were only instructed to click this link if they were interested in participating, it is not possible to determine how many MTurk users may have viewed the listing but chosen not to participate in the study.

Stimuli

All procedures of the present study were approved by the Institutional Review Board at the New York State Psychiatric Institute. Each participant began by reading a short vignette describing a patient, “Charlie,” who had been diagnosed with an addiction. Participants were randomly assigned to vignettes describing Charlie’s disorder as either AUD ($n=205$) or GD ($n=198$). They were also randomly assigned to a genetic condition ($n=204$; 99 in GD arm) or a nongenetic condition ($n=199$, 99 in GD arm). Those in the genetic condition were told that “Charlie has a type of [drinking/gambling] problem that is caused by his genes,” meaning that “Charlie has his [drinking/gambling] problem because of his DNA,” while those in the nongenetic condition were told that “Charlie has a type of [drinking/gambling] problem that is NOT caused by his genes (DNA),” meaning that “Charlie has his [drinking/gambling] problem because of the environments that he has been exposed to.” Next, participants were asked to “write a few sentences to summarize what you have learned about the causes of Charlie’s [drinking/gambling] problem,” to ensure that they paid attention to the genetic/nongenetic information.

Manipulation check

Participants were asked to rate how much of a role they believed genetics had played in causing Charlie's problem, on a scale from 1 (no role or a very minor role) to 7 (a very major role). Responses suggested that the genetic/nongenetic manipulation had been successful: ratings were significantly higher among participants in the genetic condition ($M=4.94$, $SD=1.79$) than among those in the nongenetic condition ($M=1.77$, $SD=1.24$), $F(1, 399)=423.25$, $p<.001$. There was no significant disorder $\times$ condition interaction, $F(1, 399)=2.40$, $p=.12$, suggesting that the effect of the genetic/nongenetic manipulation was constant across the two disorders, and there was no main effect of disorder, $F(1, 399)=.51$, $p=.48$, suggesting that neither disorder was seen as significantly more genetic overall.

Dependent measures

After participants completed genetic attribution ratings, they rated the likelihood that Charlie's addiction would benefit from psychotherapy and medication (each measured on a scale from 0% to 100%). They indicated how much agency they ascribed to Charlie by rating their agreement—on a scale from 1 (completely disagree) to 7 (completely agree)—with three statements adapted from those used in previous research (Lebowitz, Ahn, & Nolen-Hoeksema, 2013). These statements were as follows: "There are things Charlie can do to overcome his [drinking/gambling] problem," "Charlie has the ability to get better," and "Charlie has control over his [drinking/gambling] problem." Factor analysis with principal axis factoring revealed that the ratings of Charlie's level of control over his addiction did not load onto the same factor (see Table 2) as the other two agency items (which were highly consistent with one another, Cronbach $\alpha=.811$); thus, the self-control item was analyzed separately, while the other two agency ratings were averaged to compute an agency score for each participant. Participants also rated how much they blamed Charlie for his addiction on a scale from 1 (not at all) to 9 (very much).

At the end of the procedures, participants were asked demographic questions (see Table 1) and were compensated for their time.

Results

A 2 (disorder: GD vs. AUD) $\times$ 2 (condition: genetic vs. nongenetic) ANOVA revealed significant main effects of condition on all dependent variables (see Figure 1).

Specifically, participants in the genetic condition (compared to the nongenetic condition) rated Charlie as significantly less likely to benefit from psychotherapy, $F(1, 399)=13.59$, $p<.001$, $d=.37$ and as having significantly less agency, $F(1, 399)=24.44$, $p<.001$, $d=.50$, and control over his addiction, $F(1, 399)=6.42$, $p=.01$, $d=.25$. They also rated Charlie as less blameworthy, $F(1, 399)=49.46$, $p<.001$, $d=.71$ and more likely to benefit from medication, $F(1, 399)=43.17$, $p<.001$, $d=.64$, although medication was not seen as more likely to be effective than psychotherapy even in the genetic condition. There were no significant disorder $\times$ condition interactions (all F s < 2.74 , p s $> .10$), suggesting that the effects of the genetic/nongenetic manipulation were constant across the two disorders. The only significant main effect of disorder was on estimated likelihood of medication

effectiveness: compared to GD ($M=39.15\%$, $SD=28.19$), AUD ($M=53.01\%$, $SD=27.83$) was rated as significantly more likely to benefit from medication, $F(1, 399)=26.39$, $p<.001$, $d=.50$.

Discussion

The results of the present study suggest that while genetic explanations of addiction can increase perceived effectiveness of medication and decrease the extent to which people with addictions are blamed for their own disorders, they can also have negative effects. Specifically, they appear to reduce the personal agency and self-control ascribed to people with addictions and to decrease the perceived effectiveness of non-biomedical treatment (i.e., psychotherapy).

Interestingly, our measure of the extent to which participants perceived Charlie having control over his addiction did not load onto the same factor as the other two items measuring perceptions of the extent to which Charlie possessed agency to surmount his addiction, suggesting that it may have been gauging a separate psychological construct. Perhaps participants considered the question of whether Charlie has control over his addiction in a broader sense (e.g., one that included his ability to control whether or not he developed his addiction in the first place), whereas the other two agency measures focused more narrowly on Charlie's ability to recover from his addiction. Nonetheless, ratings of Charlie's level of control and ratings of his agency in overcoming his addiction were affected similarly by genetic explanations: both were lower on average in the genetic condition than in the nongenetic condition.

One limitation of the present study is that the genetic and nongenetic explanations included in our stimuli were simple and did not approximate the true complexity of the role of genetic and environmental factors in causing addiction. Rather than attempting to provide an accurate and comprehensive overview of the etiology of addiction, these explanations were designed to allow us to examine the effects of shifting people's causal attributions for Charlie's addiction in an experimentally controlled manner. This level of experimental control was a strength of the present study, as randomly assigning participants to either the genetic or nongenetic condition allows us to conclude that all differences we measured between the two groups of participants were caused by our experimental manipulations. By contrast, if we had merely correlated ratings on our dependent measures with participants' naturally-occurring causal attributions for Charlie's symptoms, it would have been impossible to ascertain any causal relationship between the two.

Nonetheless, separating genetic and nongenetic etiology in the manner we did may have introduced a sort of false dichotomy, as both genetic and environmental factors are likely involved in causing most (or all) cases of addiction. Although this fact was not explained to participants in the present study, future studies could provide such an explanation to participants, for example by including it in debriefing materials presented to participants after their participation is complete. Future research could also examine the impact of more sophisticated descriptions of the role of genes in addiction, as well as their interplay with environmental factors. Indeed, educating laypeople about the ways in which environments

and experiences can interact with and moderate the effects of genetic liability to disorders may be a key strategy for dispelling the kinds of essentialist assumptions (e.g., of a link between genetic etiology and reduced agency) that likely underlie the detrimental effects observed in the present study (Lebowitz, Ahn, & Nolen-Hoeksema, 2013; Lebowitz & Ahn, 2015).

Future research should also examine whether these effects are present among people with addictions and clinicians who treat addictions. As noted in the introduction, patients' and clinicians' beliefs about the self-efficacy of people with addictions and their ability to benefit from treatment can significantly impact clinical outcomes. Thus, it will be important to determine whether a pattern of results similar to the one observed in the present study occurs among clinicians and patients, because such individuals may not be affected by genetic explanations for addiction in the same ways as members of the general public. For example, given that clinicians' and patients' familiarity with addiction presumably exceeds that of unaffected laypeople, they may be less easily influenced by genetic information (or, in the case of clinicians, the effects of genetic information on their responses may depend on the extent to which their training background is biomedically oriented). Additionally, while the effects of genetic attributions were constant across the two disorders in the present study, this may not be the case among clinicians. For example, AUD is much more prevalent in the general population than GD (Grant et al., 2015; Yau & Potenza, 2015), so clinicians are likely to have had more experience treating the former than the latter, and exposure to concrete exemplars of a disorder can make clinicians less receptive to biological (e.g., genetic) explanations than they would be if they were conceiving of the disorder in more abstract terms (Kim, Ahn, Johnson, & Knobe, 2016). In general, the preliminary evidence about the effects of genetic explanations for addiction provided by the present study suggests a need to examine their impact among more clinically relevant samples—e.g., of people with addictions and clinicians who treat them—which would have even more substantial public health implications.

Overall, the present results suggest that the benefits of genetic explanations for addiction should be weighed against such explanations' detrimental effects. The ascendancy of biomedical conceptions of addictive disorders may not be as universally advantageous as is often assumed.

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Figure 1. Mean ratings for agency, blame, and self-control (top) and treatment effectiveness (bottom), by nongenetic vs. genetic condition. Error bars: ± 1 SE.

Table 1

Demographic characteristics of participants, by condition.

	Nongenetic Condition (n=199)	Genetic Condition (n=204)
Gender	52.8% female, 44.7% male, 1.5% other, 1% no response	58.3% female, 40.2% male, 0.5% other, 1% no response
Age	Range: 19–75 Mean: 36.29 years (<i>SD</i> =11.57)	Range: 19–70 Mean: 36.50 years (<i>SD</i> =11.26)
Years of Education	Range: 12–24 Mean: 15.51 years (<i>SD</i> =2.28)	Range: 8–25 Mean: 15.25 years (<i>SD</i> =2.39)
Ethnicity	Not Hispanic or Latino: 91.5% Hispanic or Latino: 5.5% No response: 3%	Not Hispanic or Latino: 87.3% Hispanic or Latino: 10.3% No response: 2.5%
Race (It was possible to select more than one response)	American Indian or Alaska Native: 3.5% Asian: 7.5% Black or African American: 7.5% Native Hawaiian or Pacific Islander: 0.5% White: 80.4% More than one race: 3% Prefer not to answer: 3%	American Indian or Alaska Native: 1.5% Asian: 5.9% Black or African American: 11.3% Native Hawaiian or Pacific Islander: 0% White: 81.4% More than one race: 2.5% Prefer not to answer: 1.5%

Table 2

Factor matrix for agency and self-control items. Participants responded by rating their agreement with each statement on a scale from 1 (completely disagree) to 7 (completely agree). Factor loadings are based on Principal Axis Factoring. Given the low factor loading of item 3, only items 1 and 2 (which were highly consistent with one another, Cronbach $\alpha=.811$) were averaged to compute agency scores, while item 3 was analyzed separately.

Item		Factor Loading
1	There are things Charlie can do to overcome his [alcohol/gambling] problem.	.817
2	Charlie has the ability to get better.	.836
3	Charlie has control over his [alcohol/gambling] problem	.446