

Biological Explanations of Generalized Anxiety Disorder: Effects on Beliefs About Prognosis and Responsibility

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Objective: Biological explanations of psychopathology can reduce the extent to which people with mental disorders are blamed for their symptoms but can also yield prognostic pessimism—the belief that psychiatric conditions are relatively immutable. However, few studies have examined whether these effects occur among persons who actually have psychiatric symptoms. This study sought to address this question. **Methods:** Adults living in the United States (N=351) were recruited online in January and February 2012 and assessed for symptoms of generalized anxiety disorder. The participants were randomly assigned to two groups: a biological condition, in which participants (N=176) were provided a description of generalized anxiety disorder and a biological explanation of the etiology of the disorder, and a control condition, in which participants (N=175) were provided the same description without any explanation of etiology. Dependent measures of treatability, duration of symptoms, and responsibility for symptoms were used to gauge beliefs regarding the prognosis and personal responsibility of a typical person with generalized anxiety disorder. **Results:** Among participants with and without symptoms of generalized anxiety disorder, the biological condition was associated with decreased ascriptions of personal responsibility for anxiety ($p=.02$) and expectations of increased duration of symptoms of generalized anxiety disorder ($p=.01$). **Conclusions:** This finding has important social and clinical implications, especially because biological conceptualizations of psychopathology are increasingly prevalent. By causing prognostic pessimism about generalized anxiety disorder, including among those with symptoms of the disorder, biological explanations could negatively affect treatment seeking and outcomes. Efforts to dispel the link between biological explanations and prognostic pessimism are needed. (*Psychiatric Services* 65:498–503, 2014; doi: 10.1176/appi.ps.201300011)

Generalized anxiety disorder, a common mental disorder (1), is increasingly explained in terms of neurobiology and genetics (2–5). Members of the public have also become more likely to believe in biological explanations for mental disorders, including neurochemical imbalances and genetic abnormalities (6).

This conceptual shift was once seen as a potential destigmatizing force, dispelling perceptions that individuals with mental disorders are responsible for their own problems (7–9). Indeed, empirical evidence links biological conceptualizations of psychopathology with reduced blame (10–12).

However, some research has linked biological construals of mental illness to more, not less, negative attitudes toward people with psychiatric illnesses (7–9), including pessimism about the prognoses of mental disorders (13–16). This prognostic pessimism reflects “neuroessentialism” and “genetic essentialism”—the inaccurate beliefs that mental disorders have fundamental, immutable essences (in the brain and genes, respectively) (8,9). This study attempted to address several unanswered questions concerning this phenomenon.

First, most research has looked at prognostic beliefs among the general public rather than among persons with symptoms of mental disorders. However, information about prognostic pessimism among symptomatic individuals would be clinically important, given that outcome expectancies are a key determinant of actual prognosis and responsiveness to treatment (17,18). In fact, patients who expect positive outcomes are more likely to seek treatment or engage with it fully (19,20). Furthermore, people with psychopathology may be especially likely to hold biological beliefs about their disorders (21), given that they may be biologically related to persons with the same disorder or prefer explanations that deflect personal responsibility. Thus it is important to understand how individuals are affected by biological explanations of disorders with which they have personal experience.

A few studies examined the effects of biological explanations of mental disorders among individuals with psychiatric

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symptoms, but they had limitations. In one study, endorsement of biological causation of depression was linked to prognostic pessimism among symptomatic individuals (22), but this research was correlational, precluding conclusions about whether the biological beliefs actually caused the prognostic pessimism. In one experimental study (16), patients showed more prognostic pessimism when told that an individual's panic disorder was caused by biological rather than psychological factors, but panic disorder was not the diagnosis of anyone in the sample. Thus it remains unknown whether biological explanations might affect people's prognostic expectations regarding their own mental health. Perhaps direct experience leads individuals to form strong, concrete beliefs about the causes or prognoses of their own mental disorders, which could be difficult to change, for example, by using biological explanations. Thus biological explanations of mental illness might be less likely to engender prognostic pessimism or otherwise alter beliefs among individuals with personal experience of mental disorders compared with persons without such experience.

In addition, despite their high prevalence among adults (1), anxiety disorders have received relatively little attention in studies of etiological beliefs and their consequences. Most research about beliefs about mental illness have examined mental illnesses generally or depression, schizophrenia, or substance dependence (6,7,23). However, not all mental disorders are subject to the same attitudes and beliefs. For example, although most research has found that biological explanations do not reduce negative attitudes toward psychopathology, some research suggests that they may reduce stigmatization of some disorders (24). To our knowledge, this study represents the first of its type to focus on generalized anxiety disorder. Because this disorder is characterized by excessive worry about the future (1), its sufferers may be particularly vulnerable to concerns about their own health, including mental health. Thus, by contributing to prognostic pessimism, biological explanations of generalized anxiety disorder could cause an exac-

erbation of the symptoms of the disorder. This possibility highlights the importance of the questions asked by this study.

We examined the impact of a biological explanation of generalized anxiety disorder on prognostic pessimism and attributions of personal responsibility among persons with and without symptoms of the disorder. The study involved systematically manipulating whether participants received a biological explanation of the disorder. The explanation was written in such a way as to facilitate the clear conclusion that the disorder had a biological cause. To our knowledge, this is the first study that has examined the consequences of experimentally varying the presence of a biological explanation of a specific mental disorder among individuals who report symptoms of the disorder. In light of existing research (8,9), we predicted that the biological explanation would decrease attributions of personal responsibility but increase prognostic pessimism.

Methods

Participants

Data were collected in January and February of 2012. Adults in the United States were recruited online through Amazon.com's Mechanical Turk service (mTurk), which allows individuals to sign up for short tasks in exchange for small monetary payments (25–27). [Information about the use of mTurk for recruitment is available online as a data supplement to this article.]

Procedures

All procedures were approved by the Institutional Review Board at Yale University and were administered online by using Qualtrics.com software. After providing informed consent, participants completed the Generalized Anxiety Disorder Questionnaire for DSM-IV (GADQ-IV), a reliable and validated measure of the diagnostic criteria for generalized anxiety disorder (28). During data analysis, we used the dichotomous diagnostic approach for scoring the GADQ-IV (29) and then grouped participants according to whether their GADQ-IV responses suggested the presence of generalized anxiety disorder.

Participants were not told about this classification.

Participants were randomly assigned to either the control (N=175) or the biological (N=176) condition. All participants first read a paragraph about the symptoms of generalized anxiety disorder, which was taken from the National Institute of Mental Health's online publication titled *Anxiety Disorders* (30). Those in the biological condition then read an empirically based biological explanation of generalized anxiety disorder. [Both descriptions are available in the online data supplement to this article.]

No information beyond the symptom description was presented to those in the control condition, in order to isolate the effects of adding a biological explanation. We chose not to include an alternative etiological explanation for the control condition because the main goal of the study was to examine potential effects of the current ascendancy of biological explanations of psychopathology. Thus we sought to compare attitudes among individuals whose causal understanding of generalized anxiety disorder was not manipulated with those of individuals who received information about the biology of the disorder. This comparison mirrored how people's reactions to a disorder might change after assimilating new information about biological factors into their preexisting conceptualizations of the condition—an occurrence that is likely to become ever more frequent. Because nonbiological explanations of psychopathology do not appear to have enjoyed a rise in popularity similar to that of biological explanations, this study did not examine their effects.

After reading the text corresponding to their assigned condition, participants were asked to imagine a typical person with generalized anxiety disorder and answer questions about that person. Even participants whose GADQ-IV responses suggested the possible presence of generalized anxiety disorder were asked to rate a typical person with generalized anxiety disorder rather than themselves. We chose this approach because, for ethical reasons, we could not manipulate beliefs about the causes of participants' own symptoms. Therefore, because the biological

explanation applied to generalized anxiety disorder generally, it was more appropriate to measure their beliefs about a typical person with the disorder.

Dependent measures were presented in two counterbalanced blocks. Within each block, the order of questions was randomized. As participants made their responses, the information about generalized anxiety disorder that they had read earlier was displayed at the bottom of the screen for use as a reference.

One block concerned the prognosis of a typical person with generalized anxiety disorder. One item, "To what extent do you believe these symptoms are treatable?" was rated on a scale from 1, very treatable, to 7, very untreatable. The other two items assessed participants' expectations regarding the duration of the person's symptoms: "How long do you think this person would continue to experience these symptoms?" and "How long do you think it would take for these symptoms to go away completely?" Both items were rated on an 8-point scale, with 1 indicating less than a week; 2, one to two weeks; 3, two to four weeks; 4, one to six months; 5, six months to one year; 6, more than one year but not indefinitely; 7, more than five years but not indefinitely; and 8, indefinitely.

The other block measured the extent to which participants considered a typical person with generalized anxiety disorder to be personally responsible for his or her symptoms. The two items were "To what extent do you believe this person is personally responsible for having these symptoms?" and "If this person tried really hard, to what extent do you believe this person would get better?" Both items were rated on a scale from 1, not at all, to 7, completely.

Finally, participants provided basic demographic information and were fully debriefed that anxiety likely results from a combination of genetic, biochemical, environmental, and psychological factors. They also received resources for finding help for psychological difficulties.

Results

The sample consisted of 351 adults (N=181 males; 52%) living in the

United States and ranging in age from 18 to 73 years (mean±SD=31.3±10.9). [More information about the demographic characteristics of the participants is available in the online data supplement.] Ninety-three participants (26%) met the GADQ-IV diagnostic cutoff for generalized anxiety disorder (N=47, control condition; N=46, biological condition). Although this rate was considerably higher than the disorder's estimated prevalence (1), it is consistent with other research that has found that rates of symptoms of anxiety disorder among mTurk users greatly exceed prevalence rates for the general population (31). Of the 258 participants who did not meet the diagnostic cutoff, exactly 50% (N=129) were assigned to each condition.

Among all participants, responses to the two items gauging personal responsibility were significantly correlated ($r=.44$, $p<.001$), so they were averaged to compute a responsibility score for each participant. The scoring range was the same as for the individual items, so that higher scores indicated stronger endorsement of the notion that a person with generalized anxiety disorder is responsible for his or her own symptoms. In addition, responses on the two items measuring expectations of symptom duration were significantly correlated (Spearman's $\rho=.76$, $p<.001$), so they were averaged to compute duration scores. The scoring range was the same as for the individual items, so that higher scores indicated longer expected duration of symptoms—an indicator of more prognostic pessimism—for a typical person with generalized anxiety disorder. The correlation between duration scores and treatability ratings, while significant, was small ($\rho=.13$, $p=.02$), so treatability ratings were analyzed separately.

We then conducted 2×2 (biological versus control condition × met or did not meet the GADQ-IV diagnostic cutoff) analyses of variance (ANOVAs) using responsibility scores and treatability ratings as the dependent variables. Because of the ordinal nature of our duration variable, we analyzed it separately using independent-samples Mann-Whitney U tests.

Participants who met the GADQ-IV diagnostic cutoff had significantly lower mean±SE responsibility scores than participants who did not meet the cutoff (2.81±1.15 versus 3.40±1.30; $F=15.07$, $df=1$ and 347, $p<.001$). There was no significant difference between these two groups for treatability ratings. Participants who met the GADQ-IV diagnostic cutoff also had significantly higher duration scores (mean±SE=6.73±1.47, median=7) than those who did not (mean±SE=6.03±1.73, median=6) ($p=.001$).

Our hypotheses pertained principally to the effects of our experimental manipulations. Indeed, a comparison of duration scores indicated that participants in the biological condition expected symptoms of generalized anxiety disorder (mean=6.44±1.63, median=7) to last longer than did those in the control condition (mean=6.00±1.71, median=6) ($p=.01$). In addition, comparison of responsibility scores indicated that participants in the biological condition attributed less personal responsibility for symptoms of generalized anxiety disorder than did those in the control condition (3.09±1.24 versus 3.39±1.31; $F=5.12$, $df=1$ and 347, $p=.02$).

The ANOVAs revealed no significant condition × diagnostic cutoff interactions, indicating that the effect of our experimental manipulation on responsibility and treatability ratings was the same regardless of whether participants met the GADQ-IV diagnostic cutoff. Nonetheless, we specifically examined the effects of our manipulations among people who met criteria for generalized anxiety disorder, given the potential clinical implications of these results. This approach also allowed us to examine the effects of our experimental manipulation on duration scores of the subset of the sample who met the cutoff. Indeed, an independent-samples Mann-Whitney U test of this subset revealed that those in the biological condition had significantly higher duration scores (mean±SE=7.10±1.20, median=7.75) than those in the control condition (mean±SE=6.36±1.61, median=6.5) ($p=.02$) (Figure 1). Notably, half of participants in the biological condition who met the GADQ-IV cutoff had the maximum duration score.

In addition, an independent-samples *t* test comparing mean responsibility scores among participants who met the cutoff found that those in the biological condition had significantly lower scores than those in the control condition, indicating that they attributed marginally less individual responsibility to a typical person with generalized anxiety disorder (2.60 ± 1.09 versus 3.02 ± 1.19 , $t=1.79$, $df=91$, $p=.08$ (Figure 2).

Discussion

To our knowledge, this study is the first to compare the effects of experimental manipulation of exposure to biological explanations of a mental disorder among people with and without symptoms of the disorder. As predicted, we found that among both people with and people without symptoms of generalized anxiety disorder, the biological explanation decreased ascriptions of personal responsibility and blame but increased prognostic pessimism.

These findings suggest that caution is warranted in disseminating biological explanations of generalized anxiety disorder, for example, in public health campaigns or in the clinical delivery of psychoeducation. An increase in biological conceptualizations of anxiety could encourage the belief that conditions like generalized anxiety disorder are relatively immutable. Among people with generalized anxiety disorder—who already have a tendency toward worrying and anticipating adversity—this belief could have negative implications for their chances of recovery (17,18).

Even our finding that biological explanations decreased ascriptions of personal responsibility for generalized anxiety disorder could be seen as a double-edged sword, reducing blame but also suggesting that persons with anxiety disorders lack control over their psyches. Indeed, one of the items assessing responsibility asked about the extent to which a typical person with generalized anxiety disorder could overcome the disorder “if this person tried really hard.” If biological explanations create or exacerbate the perception that effort to overcome one’s anxiety is likely to be futile, this could potentially decrease motivation to engage with treatment (an effortful process), which

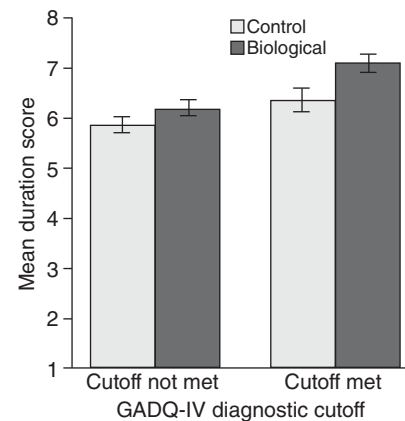
could in turn have negative clinical consequences (20).

Treatability was the only dependent variable for which we did not find a significant effect for the biological explanation. One explanation may be that biological explanations of psychopathology tend to increase the perception that medication is effective but tend to decrease the perception that psychotherapy is effective (32). If our biological explanation of generalized anxiety disorder made some potential treatments seem more efficacious while making others seem less so, these effects could have canceled each other out, leading to an overall null effect. The small correlation between treatability ratings and duration scores, and the fact that our experimental manipulation significantly affected the latter but not the former, could be explained by differences in the wording of the questions. Perhaps the participants’ duration scores reflected their expectations regarding the prognosis of generalized anxiety disorder in the absence of any treatment, given that the items assessing expectations of duration did not mention treatment. Treatability ratings, on the other hand, may pertain more to participants’ beliefs about whether treatment, once employed, would be likely to be effective.

One limitation of this study was that all participants, including those whose GADQ-IV responses indicated the potential presence of generalized anxiety disorder, were asked to answer items related to the dependent measures while imagining a typical person with generalized anxiety disorder, so we cannot conclude definitively that the biological explanation influenced any participants’ beliefs about their own symptoms of anxiety. Nonetheless, the biological explanation affected their general perceptions of the disorder, which would likely influence how they would react if given a diagnosis of generalized anxiety disorder and biological explanations for it. In addition, all participants read the information about generalized anxiety disorder and answered questions about it immediately after completing the GADQ-IV, so they were likely to still have had their own anxiety in mind while completing the

Figure 1

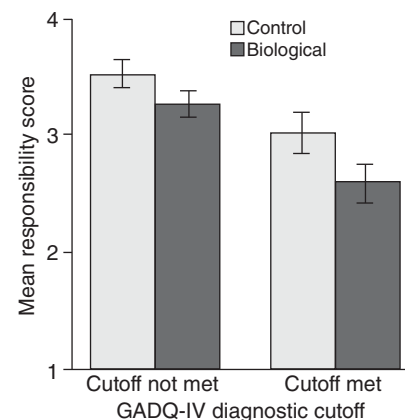
Mean duration scores among participants in the biological and control conditions who did or did not meet the diagnostic cutoff on the Generalized Anxiety Disorder Questionnaire for DSM-IV (GADQ-IV)^a



^a Duration scores indicate expected symptom duration. Possible scores range from 1 to 8, with higher scores indicating expectations of longer duration. Error bars represent plus or minus 1 standard error.

Figure 2

Mean responsibility scores among participants in the biological and control conditions who did or did not meet the diagnostic cutoff on the Generalized Anxiety Disorder Questionnaire for DSM-IV (GADQ-IV)^a



^a Responsibility scores indicate the extent to which a person with generalized anxiety disorder is considered personally responsible for his or her symptoms. Possible scores range from 1, not at all, to 7, completely. Error bars represent plus or minus 1 standard error.

items pertaining to the dependent measures.

In this study, we compared attitudes toward generalized anxiety disorder among participants who received a biological explanation of the disorder versus those who received only a description of the disorder's symptoms. We used this contrast to isolate the impact of learning new biological information. Nonetheless, our data can be used to draw conclusions only about the effects of the biological explanation compared with no causal explanation. It is conceivable that other causal accounts that place the symptoms of psychopathology outside the control of affected individuals could have effects similar to those of a biological explanation. However, this would likely depend greatly on which nonbiological causal factors were being considered, given that they might vary significantly in the extent to which they are perceived to be under individual control. The comparison used in this study avoided any potential confounding effects that could result from the idiosyncrasies of choosing a particular causal explanation as an alternative to the biological one. However, future research could specifically examine the extent to which biological explanations have unique consequences.

Conclusions

These findings highlight the potential negative consequences of biological explanations of mental disorders on prognostic expectations, both among members of the general public and individuals with symptoms of the disorder. An important direction for future research will be to explore ways of presenting biological explanations of mental disorders without yielding prognostic pessimism. Recent trends (6) suggest that public endorsement of biological explanations of psychopathology is likely to continue to increase. However, contemporary research has shown that the influence of biology on mental health is anything but deterministic (33–35). Recent evidence has suggested that some of the negative effects of biological explanations of psychopathology can be eliminated by pairing such explanations with information about how mental disorders can be successfully treated (36).

Perhaps helping the public to understand the malleable nature of biology can help to break the psychological link between biological explanations and prognostic pessimism (22). If so, current trends toward neural and genetic conceptions of psychopathology need not lead to detrimental beliefs among people with and without mental problems.

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The authors report no competing interests.

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