Effects of biological explanations for mental disorders on clinicians' empathy

Matthew S. Lebowitz[1] and Woo-kyoung Ahn

Department of Psychology, Yale University, New Haven, CT 06520

Edited by Nick Haslam, University of Melbourne, Parkville, VIC, Australia, and accepted by the Editorial Board October 31, 2014 (received for review July 23, 2014)

Mental disorders are increasingly understood in terms of biological mechanisms. We examined how such biological explanations of patients' symptoms would affect mental health clinicians' empathy—a crucial component of the relationship between treatment-providers and patients—as well as their clinical judgments and recommendations. In a series of studies, US clinicians read descriptions of potential patients whose symptoms were explained using either biological or psychosocial information. Biological explanations have been thought to make patients appear less accountable for their disorders, which could increase clinicians' empathy. To the contrary, biological explanations evoked significantly less empathy. These results are consistent with other research and theory that has suggested that biological accounts of psychopathology can exacerbate perceptions of patients as abnormal, distinct from the rest of the population, meriting social exclusion, and even less than fully human. Although the ongoing shift toward biomedical conceptualizations has many benefits, our results reveal unintended negative consequences.

empathy | mental disorders | biological explanations | dehumanization | essentialism

Mental disorders are common; lifetime prevalence rates are near 50% in the United States (1) and reach double digits in every country examined by the WHO (2). Mental disorders are also the leading cause of disability worldwide (1), with a global cost exceeding US$2 trillion (2).

In the fight against these widespread and disabling conditions, biological approaches to understanding psychopathology (e.g., through genetics and neuroscience) have been increasingly seen as a promising tool (3). This trend has been fueled by changes in funding priorities for mental health research, including President Obama's Brain Research through Advancing Innovative Neurotechnologies initiative (4, 5). The public's understanding of mental disorders has also become more biological (6).

How do biological conceptualizations of psychopathology affect mental health clinicians? Tens of millions of Americans receive mental health treatment annually (7), but little is known about how ascendant biological conceptualizations might impact clinicians. The present study examines how biological explanations of psychopathology affect clinicians' empathy toward patients, an important factor in clinical care (8).

One possibility is that when symptoms are construed biologically, clinicians' empathy for patients could increase due to reduced blame. A pervasive argument is that biological explanations are beneficial because they reduce the blame ascribed to patients (9)—indeed, trial judges gave shorter prison sentences given biological explanations of defendants' psychopathy (10). Reduced blame, in turn, would increase empathy (11).

However, there is extensive evidence linking biological conceptions of psychopathology with negative and stigmatizing social attitudes toward people with psychiatric illnesses—such as a perception that such individuals are dangerous and a desire to limit social interaction with them (12–15)—and negative attitudes are inversely associated with empathy (11, 16–18). There are several related explanations for biological explanations' deleterious effects on social attitudes. For example, biological explanations appear to lead to certain forms of so-called psychological essentialism (specifically, genetic essentialism and neuroessentialism) in which mental disorders are seen as having unique, immutable essences—located in the brain or DNA—that produce the symptoms and behavior of patients (13, 19). This view, in turn, can yield the belief that people with mental disorders are categorically dissimilar from so-called normal people, and the perception of such strict social boundaries between groups of people can lead to more negative intergroup attitudes (13, 20–22). It can also exacerbate the perception that mental disorders are relatively permanent and difficult to overcome or treat effectively, which is known as prognostic pessimism (9, 13, 23). Additionally, if the behavior of people with psychiatric disorders is seen as deterministically governed by biological abnormalities outside of their control, they may be seen as unpredictable, contributing to perceptions of them as dangerous, fearsome, and meriting avoidance (22, 24, 25). Moreover, viewing the actions of people with psychiatric illnesses as caused by neural or genetic aberrations—rather than by their own agency (23)—might trigger mechanistic dehumanization, which occurs when people are equated to automata or systems of interacting parts and is strongly linked to negative attitudes (20).

Given the link between negative attitudes and decreased empathy, it is unsurprising that many of the factors undergirding the link between biological conceptions of psychopathology and negative attitudes toward their sufferers—e.g., strict or essentialized boundaries between assumedly dissimilar social categories, skepticism about others' capacity for agency and other human mental states, mechanistic dehumanization—are also associated with diminished

Significance

Mental disorders are increasingly understood biologically. We tested the effects of biological explanations among mental health clinicians, specifically examining their empathy toward patients. Conventional wisdom suggests that biological explanations reduce perceived blameworthiness against those with mental disorders, which could increase empathy. Yet, conceptualizing mental disorders biologically can cast patients as physiologically different from "normal" people and as governed by genetic or neurochemical abnormalities instead of their own human agency, which can engender negative social attitudes and dehumanization. This suggests that biological explanations might actually decrease empathy. Indeed, we find that biological explanations significantly reduce clinicians' empathy. This is alarming because clinicians' empathy is important for the therapeutic alliance between mental health providers and patients and significantly predicts positive clinical outcomes.

Author contributions: M.S.L. and W.A. designed research; M.S.L. and W.A. performed research; M.S.L. analyzed data; and M.S.L. and W.A. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission. N.H. is a guest editor invited by the Editorial Board.

[1]To whom correspondence should be addressed. Email: matthew.lebowitz@yale.edu.

This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.1414058111/-/DCSupplemental.

empathy (18, 20, 26, 27). Thus, biological conceptualizations of psychopathology could actually decrease clinicians' empathy for patients with mental disorders. Such a finding would be of particular importance in the field of mental health because empathy is a bedrock of the therapeutic alliance that underlies clinician–patient relationships. Indeed, therapists' empathy positively predicts clinical outcomes (8). Thus, if biological explanations of mental illness decrease clinicians' empathy, patients' mental health could suffer as a result, counteracting benefits gained through ascendant biomedical approaches. However, research concerning the consequences of biological conceptualizations of psychopathology has tended to neglect empathy as an outcome measure, relative to other reactions toward people with mental disorders (e.g., prognostic pessimism, perceptions of dangerousness, desire for social distance). Furthermore, to our knowledge, no existing study has examined how biological conceptions affect the empathy of treatment providers.

Studies 1 and 2

For the first two studies, we recruited national samples of practicing mental health clinicians as participants (n = 132 in study 1; n = 105 in study 2). The samples consisted of mental health professionals, as opposed to general health professionals, and included both medically trained mental health clinicians (i.e., psychiatrists) and those with less biomedical training (e.g., psychologists and social workers).

In both studies, clinicians read vignettes describing patients with mental disorders. Study 1 used schizophrenia and social phobia, and study 2 used major depression and obsessive–compulsive disorder (OCD). For each disorder, we created two explanatory passages: One described biological (i.e., genetic and neurobiological) factors, and the other described psychosocial factors (i.e., aspects of the individual's life history). See *Supporting Information* for examples and details. Each of these explanations was paired with one of two fictitious patient case vignettes describing symptoms of a given disorder; the pairings were counterbalanced across participants. Each participant viewed all four vignettes (two disorders × two explanations).

To measure how clinicians felt about the fictitious patients, we used a well-validated method that has been extensively used in empathy research (11). For each case vignette, clinicians read a list of 18 adjectives and rated how much each one described their feelings toward the patient described. Six adjectives (e.g., warm, compassionate, sympathetic) measured empathy (i.e., other-oriented feelings of warmth and compassion important for the therapeutic alliance) (17, 28). In addition, we included six adjectives (e.g., upset, troubled, disturbed) measuring personal distress (i.e., self-oriented feelings of unease) (17, 28). The latter were included because if empathy given biological explanations was found to be lower than empathy given psychosocial explanations, we wished to verify that this was not merely because psychosocial explanations happened to be more upsetting or disturbing, resulting in increased empathy. The remaining six adjectives (angry, frustrated, happy, joyful, pleased, and resentful) were included as fillers and are not included in our analyses. For both the six empathy adjectives and the six distress adjectives, Cronbach alpha was greater than 0.87 for all disorder–explanation pairs in both studies 1 and 2, indicating a high degree of internal reliability (see *Supporting Information* for further information).

Participants also rated the extent to which they believed each patient's symptoms could improve with medication and with psychotherapy (see *Supporting Information* for details) and the clinical utility (e.g., usefulness for devising a treatment plan) of each biological and psychosocial explanatory passage (29, 30). See *Supporting Information* for discussion of additional measures.

We analyzed the data from studies 1 and 2 using a series of two (explanation: biological versus psychosocial) × two (disorder) repeated-measures ANOVAs. Our main interest was in elucidating the effects of biological explanations for mental disorders, so main effects of disorder are not reported. See Tables S1–S3 for a comprehensive breakdown of descriptive statistics.

Across disorders, the biological explanation yielded significantly less empathy than the psychosocial explanation, both in study 1 [$F(1,122) = 32.66$, $P < 0.001$] and study 2 [$F(1,103) = 18.16$, $P < 0.001$] (Fig. 1). To examine whether clinicians' biomedical training moderated these effects, we reran these ANOVAs with an additional variable distinguishing medical doctors (MDs) from non-MDs, and found that MDs reported significantly less empathy overall than non-MDs. More importantly, in both studies, the biological explanations yielded less empathy than the psychosocial explanations among both MDs and non-MDs (see *Supporting Information* for further details).

Personal distress ratings indicated that the aforementioned effects on empathy were unlikely to have resulted because the psychosocial explanations were simply more upsetting or disturbing than the biological explanations. The biological explanations did not induce higher personal distress than the psychosocial explanations for any disorder [all $t < 0.86$, $P > 0.39$]; except for schizophrenia [$t(123) = 2.99$, $P < 0.01$].

Biological explanations were also perceived to have less clinical utility than psychosocial explanations for all disorders except schizophrenia, which clinicians consider to be highly biological (31). That is, in study 1, clinical utility scores showed a two-way interaction [$F(1,122) = 10.11$, $P < 0.01$]; no significant difference emerged for schizophrenia, but the biological explanation was rated as less useful in the case of social phobia [$t(123) = -3.15$, $P < 0.01$]. In study 2, the biological explanations yielded lower clinical utility scores across disorders [$F(1,104) = 19.98$, $P < 0.001$] (Fig. 2). In both studies, there were no significant differences in clinical utility ratings between MDs and non-MDs or significant training (MD vs. non-MD) by explanation interactions.

The different explanations also led to differences in perceived effectiveness of treatment methods (see Fig. S1). Across disorders in both studies, clinicians perceived psychotherapy to be significantly less effective when symptoms were explained biologically rather than psychologically (all $t > 2.00$, all $P < 0.05$) (see *Supporting Information* for additional details). For all disorders except schizophrenia, biological explanations yielded significantly higher medication effectiveness ratings than did psychosocial explanations (all $t > 3.87$, all $P < 0.001$) (see *Supporting Information* for further details).

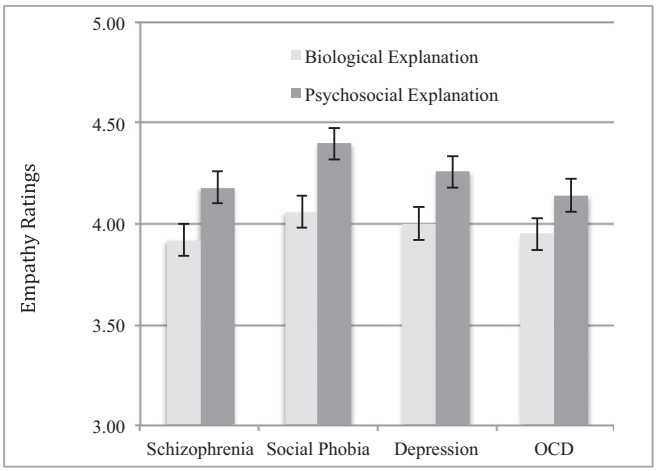

Fig. 1. Mean empathy scores, by disorder, in studies 1 (schizophrenia and social phobia) and 2 (depression and OCD). Scores range from 1 to 7, with higher numbers indicating more empathy. Error bars represent 1 $SEM^{pairedDiff}$ (36).

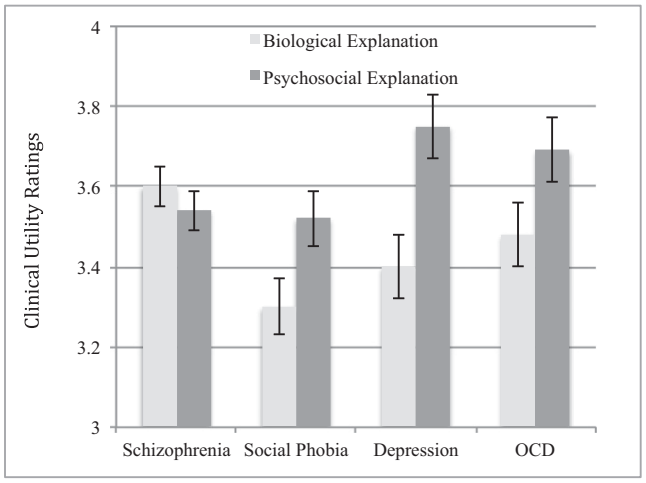

Fig. 2. Mean clinical utility scores, by disorder, in studies 1 (schizophrenia and social phobia) and 2 (depression and OCD). Scores range from 1 to 7, with higher numbers corresponding to greater perceived clinical utility. Error bars represent 1 $SEM^{pairedDiff}$ (36).

Study 3

Overall, the results of studies 1 and 2 suggested that biological explanations of psychopathology can adversely affect mental health clinicians' empathy for potential patients, as well as their ratings of psychotherapy effectiveness and perceived clinical utility. However, the explanatory passages used in these studies consisted of purely biological or purely psychosocial explanations, never combining the two. Thus, we conducted study 3 to examine the effects of explanations that included both biological and psychosocial background details, but differed in which type of information predominated. By contrasting a combined explanation dominated by psychosocial information against one dominated by biological information, we sought to more closely mirror the real world, in which biological conceptualizations of psychopathology may gradually come to predominate over psychosocial explanations without totally eliminating them.

For study 3 (n = 106), we adapted the social phobia vignettes from study 1 and the depression vignettes from study 2. Each vignette was paired with a background explanation containing both biological and psychosocial information, but for each disorder one of the vignettes was paired with an explanation in which biological information predominated and the other was paired with an explanation in which psychosocial information predominated.

The predominantly biological explanations yielded significantly lower empathy than the predominantly psychosocial explanations across disorders [$F(1,104) = 9.43$, $P < 0.01$] (see *Supporting Information* for more details). As before, there were no significant effects for personal distress ($P > 0.71$). Across disorders, the predominantly biological explanations yielded significantly higher ratings of medication effectiveness [$F(1,104) = 31.71$, $P < 0.001$] and significantly lower ratings of psychotherapy effectiveness [$F(1,104) = 25.33$, $P < 0.001$].

Discussion

Our findings across the three studies indicate that biological explanations of mental illness can have clinically relevant negative consequences among mental health clinicians. Most strikingly, clinicians' feelings of empathy for potential clients were consistently decreased when symptoms were explained using biological information (versus psychosocial information). Even when all of the hypothetical patients' symptoms were explained with both biological and psychosocial information, empathy was significantly lower when biological information predominated in the explanation versus when psychosocial information predominated.

One possible explanation for this finding is that construing psychiatric symptoms biologically portrays their sufferers as systems of interacting mechanisms rather than as human beings (27). This would amount to a form of mechanistic dehumanization, which can diminish empathy (20). Also, biological explanations can trigger genetic essentialism and neuroessentialism (13, 19), in which genes and neurobiology are assumed to represent the permanent, immutable bases of psychopathology. Such beliefs can make people with mental disorders appear categorically different from normal people.

Although further studies would be needed to substantiate the above mechanistic account, the present research ruled out one alternative possibility, as the different explanations generally did not differ in the amount of personal distress they evoked. This suggests that the observed differences in empathy did not emerge simply because the psychosocial information was upsetting whereas the biological information was emotionally neutral; instead, differences in emotional responses to the two explanatory passages appeared to be specific to empathy.

Notably, biological explanations can reduce blame (9), which would normally increase empathy (11). For instance, as mentioned earlier, trial judges imposed less severe sentences on a convict whose psychopathy was explained biologically (10). However, in the present research biological explanations reduced clinicians' empathy. One possible explanation is that clinicians—unlike judges (10)—do not see their role as one of assigning responsibility and blame for undesirable circumstances. Another is that our vignettes described patients' symptoms rather than acts of wrongdoing. However, another possibility is that our biological explanations did reduce blame, but the empathy-reducing effects of dehumanization were so strong that they overrode any such effects (32). The current research is limited in that it did not directly measure blame.

In addition, data from the present research can only show that mental health clinicians' empathy for patients is reduced when symptoms are explained through biological accounts relative to when they are explained with psychosocial information. Thus, it is not possible to determine from the present data whether biological explanations reduce empathy in an absolute sense, as opposed to psychosocial information increasing empathy in an absolute sense whereas biological information fails to do so (at least to the same extent). However, for methodological reasons, we found it necessary to compare the effects of biological explanations to those of other types of explanations rather than comparing them to those of a symptom description with no explanation. Specifically, by including the psychosocial explanations when biological explanations were absent, we attempted to control (to the extent possible) for the total amount of information about each patient that clinicians read. If we had simply compared reactions to vignettes accompanied by biological explanations against reactions to vignettes accompanied by no explanation, it would have been impossible to determine whether any observed effects were due to the type of explanation presented or simply due to the presentation of any explanation (i.e., the presence of any extra information about the patient). Moreover, in the current system of mental healthcare, psychiatric diagnoses are generally based on a clinical interview in which the clinician learns about the patient's symptomatology and life history. The differences between the two types of explanations used in the present study were designed to reflect a hypothetical but plausible future shift away from this system of diagnosis in favor of one informed by biological data rather than psychosocial history. Seen in this light, our findings suggest that such a shift could reduce mental health clinicians' empathy for patients.

The current study also found that biological explanations of patients' symptoms tend to lead clinicians to more strongly endorse medication and believe less strongly in the potential for psychotherapy to be effective (31). Given the ascendancy of

biomedical conceptions of psychopathology, clinicians' pessimistic expectancies could actually decrease patients' chances of benefiting from psychotherapy (33). Furthermore, if biological conceptualizations lead clinicians to increasingly favor pharmacotherapy over psychotherapy, this could potentially exacerbate the kind of decreases in empathy found in the current study. That is, knowing patients as individuals is important for avoiding dehumanization and fostering empathy (27), but such individuation may suffer if biological conceptualizations decrease the importance that clinicians place on personal interactions, such as those inherent in psychotherapy.

Additionally, clinicians—even including MDs—generally viewed psychosocial information as significantly more clinically utile than biological information. This finding suggests that despite the ever-increasing prominence of biological explanations for mental disorders, clinicians may be reluctant to use such explanations. This reluctance may stem from the absence to date of definitive discoveries identifying specific biological causes of mental disorders or particular biomarkers that can reliably inform psychiatric diagnosis (3).

The present research dealt only with mental disorders, which lend themselves particularly well to comparisons between the effects of biological explanations and psychosocial explanations. For many physical disorders, by contrast, such comparisons are relatively implausible, as only biological causes are traditionally conceptualized as etiologically relevant. Indeed, it may be a type of mind/body dualism that allows clinicians to make a conceptual distinction between biologically and psychosocially caused mental disorders (31)—a distinction that would not apply to many physical disorders. It remains an open empirical question whether the kinds of differences in clinicians' reactions observed in the present research would generalize to cases of physical disorders for which both psychosocial and biological explanations are plausible. For example, it is possible that a clinician presented with a patient with chronic back pain would indeed experience greater empathy—and be more likely to recommend psychotherapy—if provided with psychosocial reasons, rather than a biological explanation, for why the patient is seeking treatment. However, because the symptoms of physical disorders may be seen as affecting aspects of an individual that are less fundamental to a person's identity as a human being than those of mental disorders (34), biological explanations for physical symptoms may be less likely to dehumanize and emphasize difference. Thus, biological explanations for physical disorders may not reduce empathy relative to physical disorders, and could even increase it by deflecting blame from the affected individual. Future research could be designed to specifically test how broadly generalizable the current findings are, and what their boundary conditions may be.

Finally, the explanations for patients' symptoms provided to clinicians in the present research—even in study 3, wherein biological and psychosocial explanatory information was somewhat intermixed—were greatly oversimplified, falling far short of capturing the true complexity of mental disorders' etiology. Contemporary causal understandings of psychopathology incorporate the notion that biological, environmental, social, developmental, and psychological factors interact in a highly complex manner along the causal pathways that lead to psychiatric illness. This necessitates pluralistic conceptions of the etiology of mental disorders (35). Indeed, keeping this knowledge in mind may be important for counteracting some of the negative consequences that our findings suggest could result from the field's current trend toward biomedicalization. That is, our research suggests that embracing explanations of psychopathology that overemphasize biological explanations so strongly as to discount or minimize the importance of other factors may be damaging to clinicians' empathy for their patients. However, such negative consequences may be less likely to result if clinicians adopt conceptions of patients' symptoms that recognize how even when biology plays an important etiological role, it is constantly interacting with other factors, and biological "abnormalities" do not create strict distinctions between members of society with and without mental disorders (23).

Materials and Methods

Recruitment and Participants. In study 1, contact information for clinicians (e.g., psychiatrists, psychologists, counselors, social workers, etc.) was obtained from PsychologyToday.com clinician advertisements. In studies 2 and 3, the same method was used and was supplemented with a nationwide list of contact information for psychiatric professionals obtained from the American Psychiatric Association's membership directory. Postcards were sent to clinicians inviting them to participate in an online study and directing them to a Web address where they could complete the study if they wished to participate (sample recruitment postcard text is included in *Supporting Information*). An online questionnaire was used to obtain informed consent and administer all study procedures. Demographic information is provided in Table S4. In exchange for their participation, participants were compensated with a US$25 gift certificate to Amazon.com.

In an attempt to increase the representativeness of the sample by ensuring geographic diversity, for each study we sent recruitment postcards to clinicians in more than a dozen different US states. We calculated response rates by computing the percentage of successfully delivered recruitment postcards that resulted in responses for each study. These were 16.88% in study 1, 14.19% in study 2, and 12.83% in study 3; these response rates are comparable to those of other published studies of mental health clinicians (29).

Measures and Procedures. All procedures were approved by Yale University's Human Subjects Committee, and all participants provided informed consent before completing the experiments. Samples of the case vignettes used in the present research, as well as the explanatory passages paired with them, are included in *Supporting Information*. The disorders used were chosen to span the continuum from those that clinicians tend to view as highly biological to those they tend to see as highly nonbiological (31). The vignettes did not include diagnostic labels, but they described fictitious patients whose symptoms met diagnostic criteria for the disorder in question, according to the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (Text Revision)* (37), which was current when studies 1 and 2 were conducted. The newer fifth edition (38) does not include any changes to the diagnostic criteria for the disorders included in the present research that would be expected to affect our results. The explanatory passages described biological and/or psychosocial factors that have been linked to the disorder in question in the empirical literature and/or in existing explanatory descriptions available for public consumption, such as the National Institute of Mental Health's online "Mental Health Information" factsheets (www.nimh.nih.gov/health/topics) and the website WebMD.com.

After reading each vignette and its associated explanation, participants rated the extent to which a list of adjectives characterized their feelings about the person described, on a seven-point scale from 1 = "not at all" to 7 = "very much". Among these adjectives were six (sympathetic, softhearted, warm, compassionate, tender, and moved) that have been used to measure empathic concern, and six (alarmed, troubled, distressed, upset, disturbed, and worried) that have been used to measure personal distress (17, 28). Ratings of each group of adjectives were averaged to compute an empathy score and a distress score for each participant's response to each vignette. Participants in all studies also used seven-point scales to rate the extent to which they believed that the individual's symptoms could improve with medication or with psychotherapy (where 1 = "not at all" and 7 = "completely").

Additionally, in studies 1 and 2 we measured the clinical utility of each explanatory passage by adapting six questions from those used in previous studies (29, 30). The first three questions asked participants to rate how useful each explanatory passage was for determining a prognosis, devising treatment plans for the person described, and understanding the causes of any mental disorder that the person might have. The remaining questions solicited participants' ratings of the information's usefulness for communicating with other mental health professionals and the patient, and for comprehensively describing all of the patient's important problems. Principal components analyses (with varimax rotation) conducted separately for each disorder–explanation pair revealed that all six clinical utility items loaded onto a single factor for both studies 1 and 2. Cronbach alpha was greater than 0.85 for each disorder–explanation pair for both studies 1 and 2, indicating a high degree of reliability, so all clinical utility items were averaged to compute clinical utility scores for each participant (separately for each disorder–explanation pair).

For study 3, we created predominantly biological explanations and predominantly psychological explanations for major depression and social phobia as follows. For each disorder, we first created two sets (B1 and B2) of four biological factors (e.g., abnormal brain activity or genes) and two sets (P1 and P2) of four psychological factors (e.g., childhood trauma, bereavement). We then created four predominantly biological explanatory passages by combining the four factors from set B1 with one factor from set P1, and four predominantly psychosocial explanations by combining the four from set P2 with one factor from set B2. We developed a total of four patient case vignettes (two for social phobia and two for major depression), which were adapted from those used in studies 1 and 2. Participants in study 3 viewed all four vignettes; for each disorder, one vignette was randomly paired with one of the predominantly psychosocial explanations and the other was randomly paired with one of the predominantly biological explanations.

ACKNOWLEDGMENTS. This work was supported by National Institutes of Health Grant R01 HG007653 (to W.A.).

1. Whiteford HA, et al. (2013) Global burden of disease attributable to mental and substance use disorders: Findings from the Global Burden of Disease Study 2010. *Lancet* 382(9904):1575–1586.
2. Bloom DE, et al. (2011) *The Global Economic Burden of Noncommunicable Diseases* (World Economic Forum, Geneva).
3. Deacon BJ (2013) The biomedical model of mental disorder: A critical analysis of its validity, utility, and effects on psychotherapy research. *Clin Psychol Rev* 33(7):846–861.
4. Insel T, et al. (2010) Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *Am J Psychiatry* 167(7):748–751.
5. Insel TR, Landis SC, Collins FS; The NIH BRAIN Initiative (2013) Research priorities. *Science* 340(6133):687–688.
6. Pescosolido BA, et al. (2010) "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *Am J Psychiatry* 167(11):1321–1330.
7. Olfson M, Marcus SC (2010) National trends in outpatient psychotherapy. *Am J Psychiatry* 167(12):1456–1463.
8. Elliott R, Bohart AC, Watson JC, Greenberg LS (2011) Empathy. *Psychotherapy* 48(1): 43–49.
9. Kvaale EP, Haslam N, Gottdiener WH (2013) The 'side effects' of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clin Psychol Rev* 33(6):782–794.
10. Aspinwall LG, Brown TR, Tabery J (2012) The double-edged sword: Does biomechanism increase or decrease judges' sentencing of psychopaths? *Science* 337(6096):846–849.
11. Batson CD (2011) *Altruism in Humans* (Oxford Univ Press, New York).
12. Read J, Haslam N, Sayce L, Davies E (2006) Prejudice and schizophrenia: A review of the 'mental illness is an illness like any other' approach. *Acta Psychiatr Scand* 114(5):303–318.
13. Haslam N (2011) Genetic essentialism, neuroessentialism, and stigma: Commentary on Dar-Nimrod and Heine (2011). *Psychol Bull* 137(5):819–824.
14. Kvaale EP, Gottdiener WH, Haslam N (2013) Biogenetic explanations and stigma: A meta-analytic review of associations among laypeople. *Soc Sci Med* 96:95–103.
15. Angermeyer MC, Holzinger A, Carta MG, Schomerus G (2011) Biogenetic explanations and public acceptance of mental illness: Systematic review of population studies. *Br J Psychiatry* 199(5):367–372.
16. Batson CD, Chang J, Orr R, Rowland J (2002) Empathy, attitudes and action: Can feeling for a member of a stigmatized group motivate one to help the group? *Pers Soc Psychol Bull* 28(12):1656–1666.
17. Batson CD, et al. (1997) Empathy and attitudes: Can feeling for a member of a stigmatized group improve feelings toward the group? *J Pers Soc Psychol* 72(1):105–118.
18. Batson CD, Ahmad NY (2009) Using empathy to improve intergroup attitudes and relations. *Soc Issues Policy Rev* 3(1):141–177.
19. Dar-Nimrod I, Heine SJ (2011) Genetic essentialism: On the deceptive determinism of DNA. *Psychol Bull* 137(5):800–818.
20. Haslam N (2006) Dehumanization: An integrative review. *Pers Soc Psychol Rev* 10(3): 252–264.
21. Mehta S, Farina A (1997) Is being "sick" really better? Effect of the disease view of mental disorder on stigma. *J Soc Clin Psychol* 16(4):405–419.
22. Read J (2007) Why promoting biological ideology increases prejudice against people labelled "schizophrenic". *Aust Psychol* 42(2):118–128.
23. Lebowitz MS, Ahn WK, Nolen-Hoeksema S (2013) Fixable or fate? Perceptions of the biology of depression. *J Consult Clin Psychol* 81(3):518–527.
24. Dietrich S, Matschinger H, Angermeyer MC (2006) The relationship between biogenetic causal explanations and social distance toward people with mental disorders: Results from a population survey in Germany. *Int J Soc Psychiatry* 52(2):166–174.
25. Speerforck S, Schomerus G, Pruess S, Angermeyer MC (2014) Different biogenetic causal explanations and attitudes towards persons with major depression, schizophrenia and alcohol dependence: Is the concept of a chemical imbalance beneficial? *J Affect Disord* 168:224–228.
26. Cikara M, Bruneau EG, Saxe RR (2011) Us and them: Intergroup failures of empathy. *Curr Dir Psychol Sci* 20(3):149–153.
27. Haque OS, Waytz A (2012) Dehumanization in medicine: Causes, solutions, and functions. *Perspect Psychol Sci* 7(2):176–186.
28. Batson CD, Fultz J, Schoenrade PA (1987) Distress and empathy: Two qualitatively distinct vicarious emotions with different motivational consequences. *J Pers* 55(1): 19–39.
29. Rottman BM, Ahn WK, Sanislow CA, Kim NS (2009) Can clinicians recognize DSM-IV personality disorders from five-factor model descriptions of patient cases? *Am J Psychiatry* 166(4):427–433.
30. Rottman BM, Kim NS, Ahn WK, Sanislow CA (2011) Can personality disorder experts recognize DSM-IV personality disorders from five-factor model descriptions of patient cases? *J Clin Psychiatry* 72(5):630–639.
31. Ahn WK, Proctor CC, Flanagan EH (2009) Mental health clinicians' beliefs about the biological, psychological, and environmental bases of mental disorders. *Cogn Sci* 33(2):147–182.
32. Schomerus G, Matschinger H, Angermeyer MC (2014) Causal beliefs of the public and social acceptance of persons with mental illness: A comparative analysis of schizophrenia, depression and alcohol dependence. *Psychol Med* 44(2):303–314.
33. Meyer B, et al. (2002) Treatment expectancies, patient alliance, and outcome: Further analyses from the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *J Consult Clin Psychol* 70(4):1051–1055.
34. Lebowitz MS (2014) Biological conceptualizations of mental disorders among affected individuals: A review of correlates and consequences. *Clin Psychol Sci Pract* 21(1):67–83.
35. Kendler KS (2012) The dappled nature of causes of psychiatric illness: Replacing the organic-functional/hardware-software dichotomy with empirically based pluralism. *Mol Psychiatry* 17(4):377–388.
36. Franz VH, Loftus GR (2012) Standard errors and confidence intervals in within-subjects designs: Generalizing Loftus and Masson (1994) and avoiding the biases of alternative accounts. *Psychon Bull Rev* 19(3):395–404.
37. American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Publishing, Arlington, VA), 4th Ed, Text Rev.
38. American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Publishing, Arlington, VA), 5th Ed.