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THE CHEMICAL IMBALANCE EXPLANATION OF DEPRESSION: REDUCING BLAME AT WHAT COST?

BRETT J. DEACON AND GRAYSON L. BAIRD
University of Wyoming

Brain disease models of psychopathology, such as the popular "chemical imbalance" explanation of depression, have been widely disseminated in an attempt to reduce the stigma of mental illness. Ironically, such models appear to increase prejudicial attitudes among the general public toward persons with mental disorders. However, little is known about how biochemical causal explanations affect the perceptions of individuals seeking mental health treatment. Ninety undergraduate students participated in a thought experiment in which they were asked to imagine feeling depressed, seeking help from a doctor who diagnosed them with major depressive disorder, and receiving, in counterbalanced order, a chemical imbalance and biopsychosocial explanation for their symptoms. Ratings of each explanation's credibility and perceptions of self-stigma (e.g., blame), prognosis, and treatment expectancies were obtained. Compared to the biopsychosocial model, the chemical imbalance model was associated with significantly less self-stigma but also significantly lower credibility, a worse expected prognosis, and the perception that psychosocial interventions would be ineffective. The chemical imbalance explanation appears to reduce blame at the cost of fostering pessimism about recovery and the efficacy of nonbiological treatments. Research is needed on how the chemical imbalance model affects the clinical response of patients receiving mental health treatment.

With a lifetime prevalence of 16.6% (Kessler, Berglund, Demler, Jin, & Walters, 2005), major depression is the most common mental disorder in the United States. The rate of outpatient treatment for depression has dramatically increased in recent years, with the

Correspondence concerning this article should be addressed to Brett Deacon, PhD, University of Wyoming, Department of Psychology, Dept. 3415, 1000 E. University Ave., Laramie, WY 82071. E-mail: bdeacon@uwyo.edu.

vast majority of patients receiving pharmacotherapy (Olfson et al., 2002). At the same time, selective serotonin reuptake inhibitors (SSRIs) have become among the best-selling drugs in medical practice (International Marketing Services Health, 2004). The popularity of SSRIs in the treatment of depression and a variety of other conditions is, in part, the product of a massive and enormously successful advertising campaign by the pharmaceutical industry (Lacasse & Leo, 2005). A cornerstone of this campaign is promoting the notion that mental disorders like depression are caused by a "chemical imbalance" in the brain which is "corrected" with SSRI medications.

Pervasive exposure to pharmaceutical advertisements advancing the chemical imbalance model of depression is common in the United States and affects the treatment-seeking behaviors of consumers and prescribing practices of physicians (Kravitz et al., 2005; Mintzes et al., 2003). Preliminary research (Schreiber & Hartrick, 2002) and clinical experience suggest that physicians and other healthcare providers often inform their patients that depression is a medical disease caused by faulty brain chemistry which requires medication to correct. Such an explanation is at odds with research indicating that depression may be caused by a combination of biological, environmental, and psychosocial factors (National Institute of Mental Health, 2001), as well as evidence that psychological treatments like cognitive-behavior therapy are as effective as SSRIs in the short-term and somewhat more effective in the long-term (DeRubeis et al., 2005; Dimidjian et al., 2006; Hollon et al., 2005). Nevertheless, the brain disease model of depression continues to be heavily promoted, in part because of commercial influence but also owing to the widespread belief that biological explanations of mental illness reduce stigma.

Many healthcare professionals, patient advocacy organizations like the National Alliance on Mental Illness (NAMI, 2006), and governmental entities like the Surgeon General of the United States (U.S. Department of Health and Human Services, 1999) explicitly promote a brain disease model of mental illness based on the assumption that it reduces the shame experienced by patients and fosters more compassionate attitudes from the general public. A representative example appears on the "About Mental Illness" section of NAMI's website: "Mental illnesses are biologically based brain disorders. They cannot be overcome through 'will power' and are not related to a person's 'character' or intelligence." This model re-

flects the notion that the best (and perhaps only) method of reducing stigma is to ascribe psychological problems entirely to factors that are beyond an individual's control. While the brain disease model may be advanced in a genuine desire to reduce the shame and prejudice associated with mental illness, a growing body of research suggests that, ironically, biological explanations may actually increase stigma.

Biological explanations of mental illness reliably elicit more prejudice toward persons with mental disorders than psychological or social explanations. Research by Read and colleagues (Read & Harré, 2001; Read & Law, 1999; Walker & Read, 2002) demonstrates that endorsement of a biological explanation is associated with more negative attitudes towards individuals with mental disorders (e.g., that they are more unpredictable, antisocial, and dangerous) than psychological explanations among college students. Lam and Salkovskis (2006) reported similar findings among depressed or anxious patients who watched a videotape of a female patient with panic disorder whose symptoms were described as caused by either biological factors, psychological factors, or unknown factors. Participants rated the patient as most likely to harm herself and others when her symptoms were ascribed to "genetic factors, involving biochemical imbalances." Mehta and Farina (1997) found that college students delivered electric shocks of higher intensity and duration to an allegedly mentally ill confederate when the confederate's symptoms were described as caused by biological rather than psychosocial factors.

On the other hand, biological explanations fare somewhat better in reducing the extent to which individuals with mental disorders are blamed for their symptoms. Research shows that participants express less blame toward individuals with schizophrenia (Phelan, Cruz-Rojas, & Reiff, 2002) and depression (Goldstein & Rosselli, 2003) when their symptoms are attributed to a biological cause. Similarly, Mehta and Farina (1997) found that allegedly mentally ill confederates were held less responsible for their performance on a learning task when their disorder was attributed to biological as opposed to psychosocial factors. However, Mehta and Farina also reported that a biological explanation did not improve the likeability, perceived adjustment, perceived dangerousness, or willingness to engage in a social relationship with the confederate. While the aforementioned studies provide some support for the beneficial

effects of biological explanations on blame, they also suggest that biological explanations increase other forms of stigma (e.g., desired social distance).

Stigma is a complex concept that encompasses the reactions of the general public toward a stigmatized group (public stigma) and the reactions of members of a stigmatized group toward themselves (self-stigma; Rüsch, Angermeyer, & Corrigan, 2005). The vast majority of stigma research has examined aspects of public stigma, such as how the general public views individuals with mental disorders in terms of culpability for their problem, dangerousness, etc. This research suggests that the brain disease explanation may reduce blame but does so at the cost of fostering generally negative perceptions of those with mental disorders. Unfortunately, little is known about how etiological models affect the way individuals with mental disorders view themselves. For healthcare professionals who wish to convey a causal explanation to their patients that reduces self-stigma, such knowledge is essential. Do patients told they are suffering from a chemical imbalance feel less blame for having developed their symptoms? Do they expect others to feel less negatively toward them? At present, these questions are largely unanswered in the scientific literature.

The brain disease model of mental illness has consistently been shown to deleteriously affect perceptions of prognosis. Researchers have documented this effect with respect to beliefs about the curability, controllability, and chronicity of mental disorders. In studies comparing the effects of biological and psychological explanations of mental illness on expected prognosis, biologically-caused mental health problems are rated as less curable, less likely to respond to treatment, more likely to require intensive and long-term professional help, and more likely to persist over time (Farina, Fisher, Getter, & Fischer, 1978; Lam & Salkovskis, 2006; Lam, Salkovskis, & Warwick, 2005; Phelan et al., 2002; Phelan, Yang, & Cruz-Rojas, 2006). Compared to the somewhat inconsistent literature on the brain disease model and stigma, research reliably demonstrates that biological explanations of mental illness foster pessimism about treatment and recovery. However, few studies have investigated perceptions of prognosis among individuals with mental disorders who are told *their own* symptoms are caused by a brain disease. Given that positive treatment expectancies are essential to the success of mental health treatments (Kirsch, 1999), whether or not the brain

disease model fosters a poor expected prognosis in clinical patients is a matter of substantial importance.

A final issue concerns the influence of the brain disease model on perceptions of mental health treatments. Research shows that both the lay public and most clinical patients prefer psychosocial approaches to pharmacotherapy for mental health problems (Deacon & Abramowitz, 2005; Jorm et al., 1997). It is possible that the brain disease model makes pharmacotherapy more credible and acceptable to patients. Indeed, a chemical imbalance explanation of mental illness is clearly more compatible with pharmacological treatment than psychotherapy. Not surprisingly, research suggests that acceptance of a biological model of mental illness is associated with more favorable perceptions of pharmacotherapy (Phelan et al., 2006). However, it also seems possible that the brain disease model fosters pessimism about nonpharmacological treatments. If one's symptoms are caused solely by defective brain chemistry, what is the value of approaches like psychotherapy, physical exercise, or looking more deeply into the (nonbiological) problems in one's life? Research is needed on how the brain disease model affects treatment expectancies and responses among individuals with mental disorders.

The purpose of the present study was to examine the effects of a chemical imbalance causal explanation of depression on perceptions of credibility, stigma, prognosis, and the effectiveness of different treatments. In contrast to most previous studies that have compared this explanation to a psychosocial model, we elected to adopt a biopsychosocial account. The biopsychosocial model is consistent with current scientific theory and evidence about the cause of depression (National Institute of Mental Health, 2001) and avoids the ideological polarization of comparing extreme prototypical biological and psychosocial models without including the more valid biopsychosocial model. Further, research by Walker and Read (2002) indicates that participants' responses to the biopsychosocial model are highly comparable to those elicited by the psychosocial model, suggesting that the more inclusive and accurate biopsychosocial model is a reasonable alternative to a psychosocial-only account. We elected to study depression, rather than mental illness in general, for two reasons: (a) depression is a relatively common and well-known mental disorder, and (b) based on the findings of France, Lysaker, and Robinson (2007) nearly all participants were expected to have consider-

able familiarity with the chemical imbalance explanation of depression via exposure to drug advertisements. We asked participants to imagine they were depressed and sought treatment from a doctor who diagnosed them with major depressive disorder and provided a specific causal explanation for their symptoms; participants then rated the credibility of the causal explanation, the stigma associated with depression, their expected prognosis, and the perceived efficacy of different treatments for depression. Each participant provided ratings for both the chemical explanation and the biopsychosocial explanation, the order of which was counterbalanced across participants. We hypothesized that compared to a biopsychosocial explanation, the chemical imbalance explanation would be associated with less self-stigma, less credibility, a worse prognosis, and the perception that only treatments involving medication would be effective.

METHOD

PARTICIPANTS

Ninety undergraduate students were recruited from a sophomore-level abnormal psychology course at the University of Wyoming. The mean age was 20.9 (*SD* = 2.4; range = 18-29), 58 participants (64.4%) were women, and 80 (88.9%) described themselves as Caucasian. Twenty-six individuals (28.9%) reported a mental health treatment history, including three with medication only, nine with psychotherapy only, and 14 who had received both treatments.

MEASURES

Participants completed a three-part questionnaire constructed for the present study. One section assessed demographic data, history of participation in psychotherapy and pharmacotherapy for mental health problems, and whether participants believed that depression is primarily caused by "biological factors like brain chemistry" or "psychological or environmental factors like negative thoughts and behaviors or stressful life events." The remaining two sections

asked participants to imagine they had been feeling depressed for several weeks, sought help from a doctor who diagnosed them with "major depression," and were provided with a specific causal explanation. In one section the following chemical imbalance causal explanation, adopted from the National Alliance on Mental Illness online document titled "About Mental Illness" as well as Lacasse and Leo's (2005) review of SSRI advertisements, was provided:

> Depression is a common medical illness that is no different from any other disease. It is caused by a chemical imbalance in the brain involving the neurotransmitter serotonin. It is not the result of a personal weakness or character flaw and is not something that can be overcome through willpower or by pulling yourself together.

In the other section, a biopsychosocial causal explanation adopted from the National Institute of Mental Health (2001) document "The Invisible Disease: Depression" was presented:

> Depression is a common mental disorder. There are a number of reasons why a person might become depressed, including having a family history of depression, stressful life experiences like the death of a loved one, and having low self-esteem and a pessimistic attitude. It is probably a combination of biological, environmental, and psychological factors that causes depression in most individuals.

After reading the first causal explanation, respondents indicated their agreement with 13 questions (presented in Table 1) on a 5-point Likert-type scale ranging from 0 (not at all) to 4 (extremely). These questions assessed the credibility of the explanation (one item), stigma (four items), prognosis (four items), and the perceived efficacy of different treatment approaches (four items). Participants then read the second section which presented the alternative causal explanation of depression and rated the same 13 questions. The study measures may be obtained by contacting the first author.

The items assessing credibility and the perceived efficacy of different treatments were examined individually because each was assumed to assess a distinct outcome. We used principal components analysis (PCA) to determine the extent to which the four stigma and four prognosis items formed separate factors for the chemical imbalance and biopsychosocial explanations, respectively. Each PCA employed an oblique (Oblimin) rotation and specified a two-factor

TABLE 1. Questionnaire Items Assessing Perceptions of Depression in the Context of Different Causal Explanations

Domain and Item
Credibility
1. How believable does this explanation seem to you?
Stigma
2. To what extent would you feel personally responsible for having developed depression?
3. To what extent would you believe personal weakness or a character defect might have contributed to your depression?
4. To what extent should others hold you personally responsible for having developed depression?
5. If other people accepted this explanation for your depression, would you expect them to think negatively of you?
Prognosis
6. To what extent would you believe you could eventually recover from your depression?
7. To what extent would you feel able to effectively control the depression on your own?
8. To what extent would you believe long-term treatment is necessary to overcome your depression?
9. To what extent would you expect your depression to be a chronic problem that persists for years?
Treatment
10. How effective would you expect medication to be in treating your depression?
11. How effective would you expect psychotherapy to be in treating your depression?
12. How effective would you expect a combination of psychotherapy and medication to be in treating your depression?
13. To what extent would you believe that making changes in your attitudes and lifestyle would improve your depression?

solution. PCA of the eight relevant chemical imbalance items yielded a clear two-factor structure (first three eigenvalues = 2.86, 2.15, 0.92) that explained 62.6% of the item variance. The first factor consisted of the four stigma items, each with a factor loading greater than .70 (M = .81). The second factor contained the four prognosis items, all of which had factor loadings above .60 (M = .74). Similar results were obtained in the PCA of the biopsychosocial items. A two-factor solution was supported (first three eigenvalues = 2.65, 1.99, 1.04) that explained 58.0% of the item variance. All four stigma items had salient (>.70) loadings on the first factor (M = .77), and all four prognosis items had salient (>.40) loadings on the second factor (M = .71). Accordingly, the four stigma items were summed

to form a stigma composite score for each causal explanation. Likewise, the four prognosis items were summed into a prognosis composite score for each causal explanation. Internal consistency for the stigma items was (α = .83) for the chemical imbalance explanation and (α = .79) for the biopsychosocial explanation. Alpha coefficients were somewhat lower for the prognosis items for the chemical imbalance (α = .73) and biopsychosocial explanations (α = .68).

As a result of combining the stigma and prognosis items into composite scores, the original pool of 13 items yielded seven distinct scores corresponding to ratings of credibility, stigma, prognosis, and the perceived efficacy of medication, psychotherapy, their combination, and changing one's attitude and lifestyle. For purposes of clarity these seven items will be collectively referred to as *Perceptions of Depression* in the sections that follow.

PROCEDURE

Two versions of the study measures were prepared, one with the chemical imbalance explanation preceding the biopsychosocial explanation, and one with the opposite order. The cover sheet of both packets was identical. Prior to their distribution to study participants, these two questionnaire versions were sorted and arranged in alternating order. The questionnaires were handed out to students on a row-by-row basis in the classroom during a single class period. Exactly 50% of the participants completed each version of the questionnaire. Data were collected prior to any class discussion of the nature and treatment of depression. Students were given the option of signing a consent form to allow their confidential responses to be used for research purposes; all 90 respondents consented. This study was approved by the University of Wyoming institutional review board.

It should be noted that our alternate-person method of distributing the questionnaire packets was a technical error of nonrandom assignment, well worth correcting in future work, but not fatal to the present study because it appears quite unlikely to have affected the results. Every participant rated both the brain chemical explanation and the biopsychosocial explanation, so the random assignment issue pertains only to the order in which they did so. The alternate-person assignment method did not create any obvious sys-

tematic bias in questionnaire order according to the row or area of the classroom participants sat in, which class they were attending, or other such factors as participants freely chose their own seating and were unaware of the experiment when they chose their seats the day it took place. Further, analyses of participant background variables including age, sex, and history of participation in pharmacotherapy and psychotherapy found no significant differences between the group that got the chemical-first packet and the group that got the biopsychosocial-first packet, all p's > .40. Lack of bias in packet assignment is also consistent with the findings reported in detail in the results section below that no significant intergroup effects or interactions were found in any dependent variable as a function of questionnaire order. Therefore we treated the data as if the assignment to questionnaire order had been random, although we acknowledge that randomness can be truly ensured only by procedures such as randomizing the order of packets before distributing them.

RESULTS

EFFECTS OF DIFFERENT CAUSAL EXPLANATIONS ON PERCEPTIONS OF DEPRESSION

A series of 2 (order of questionnaire administration) x 2 (type of causal explanation) mixed ANOVAs were conducted to examine whether the order in which the causal explanations were presented (chemical imbalance vs. biopsychosocial explanation first) affected ratings of the credibility, stigma, prognosis, and perceived treatment efficacy associated with the two causal explanations. None of the interaction terms were statistically significant (all p's > .20), indicating that the within-subjects comparisons reported below are independent of the order in which the measures were administered. Therefore, the remaining data analyses were performed with the data of the two questionnaire orders pooled. Table 2 presents descriptive statistics, paired-samples t-tests, and effect size estimates (Cohen's d) for scores on each questionnaire item or composite score in the two conditions. As can be seen, statistically significant differences

TABLE 2. Perceptions of Depression in the Context of a Chemical Imbalance versus Biopsychosocial Causal Explanation: Descriptive Statistics, Paired-Samples t-tests, and Effect Size Estimates

Domain and Item	Chemical Imbalance Explanation *M* (*SD*)		Biopsychosocial Explanation *M* (*SD*)		*t*[1]	*d*
Credibility						
1. Believability	1.98	(1.03)	3.14	(0.77)	-8.09**	1.28
Stigma						
2. Personally responsible	1.13	(1.05)	2.14	(0.95)	-7.57**	1.01
3. Due to weakness/defect	1.40	(1.14)	2.08	(0.95)	-4.58**	0.65
4. Others should hold me responsible	0.66	(0.81)	1.69	(1.03)	8.00**	1.10
5. Others would think negatively of me	0.92	(1.06)	1.54	(1.10)	4.63**	0.58
Prognosis						
6. Can eventually recover	2.02	(1.19)	2.56	(0.89)	3.68**	0.51
7. Able to control myself	0.94	(0.95)	1.97	(1.03)	7.51**	1.04
±8. Need long-term treatment	2.57	(1.01)	2.22	(0.89)	2.48*	0.37
±9. Chronic problem	2.53	(1.13)	1.97	(1.03)	3.42**	0.52
Treatment						
10. Medication effectiveness	3.06	(1.02)	1.80	(1.07)	9.42**	1.20
11. Psychotherapy effectiveness	1.43	(1.05)	2.52	(0.88)	7.80**	1.12
12. Combined treatment effectiveness	2.61	(0.84)	2.72	(0.80)	-1.10	0.13
13. Effectiveness of changing attitude and lifestyle	1.22	(1.08)	2.97	(0.88)	12.38**	1.76

Note. [1]Degrees of freedom for paired samples *t*-tests range from 86 to 89 due to missing data. ± = items were reverse scored when calculating composite scores. Scale: 0 = "not at all"; 1 = "slightly"; 2 = "moderately"; 3 = "very"; 4 = "extremely." *$p < .05$, **$p < .001$.

and moderate-to-large effect sizes between the two explanations were evident on 12 of the 13 items.

Credibility. As shown in Table 2, participants rated the biopsychosocial explanation as significantly more credible than the chemical imbalance explanation ($p < .001$).

Stigma. The chemical imbalance explanation was rated as significantly less stigmatizing than the biopsychosocial explanation on all four stigma items (all p's $< .001$; see Table 2). Stigma composite scores for the chemical imbalance condition ($M = 4.06$, $SD = 3.30$) were substantially lower than those for the biopsychosocial condition ($M = 7.46$, $SD = 3.14$), t (87) = -8.17, $p < .001$, $d = 1.06$.

Prognosis. As demonstrated in Table 2, the biopsychosocial explanation received significantly more favorable ratings than the chemical imbalance explanation on each of the four prognosis items (all p's $< .05$). Prognosis composite scores were significantly higher in the biopsychosocial condition ($M = 8.38$, $SD = 2.70$) than in the chemical imbalance condition ($M = 5.92$, $SD = 3.18$), t (85) = -5.48, $p < .001$, $d = .83$.

Treatment. The chemical imbalance explanation was associated with more positive expectancies for the efficacy of medication ($p < .001$), whereas the biopsychosocial explanation was associated with higher perceived efficacy of psychotherapy ($p < .001$; see Table 2). There was no difference between causal explanations in the perceived efficacy of combined treatment. Lastly, participants indicated that making changes in their attitudes and lifestyle would be much more helpful ($p < .001$) in the context of a biopsychosocial explanation than a chemical imbalance explanation.

PERCEIVED EFFICACY OF DIFFERENT TREATMENTS WITHIN EACH CAUSAL EXPLANATION

To determine which interventions were perceived as most efficacious in the context of each causal explanation of depression, we conducted separate one-way repeated measures ANOVAs for the ratings of the chemical imbalance and biopsychosocial explanations reported in Table 2. In each analysis, perceived efficacy ratings for the four different treatments were entered as within-subject vari-

ables. For the chemical imbalance explanation, a significant within-subjects effect of treatment type was found, F (3, 85) = 48.64, p < .001. Follow-up Fisher LSD post-hoc tests indicated that medication was perceived as the most efficacious treatment and was rated as significantly more likely to be helpful than changing one's attitudes and lifestyle (p < .001; d = 1.75), psychotherapy (p < .001; d = 1.57), and combined treatment (p < .001; d = .48). Combined treatment was also rated as significantly more effective than changing one's attitude and lifestyle (p < .001; d = 1.44) and psychotherapy (p < .001; d = 1.24).

A significant effect of treatment type was also obtained for the biopsychosocial condition, F (3, 84) = 30.81, p < .001. Follow-up Fisher LSD post-hoc tests indicated that changing one's attitude and lifestyle was rated as the most efficacious intervention and was perceived as significantly more effective than medication (p < .001; d = 1.19), psychotherapy (p < .001; d = .51), and combined treatment (p < .05, d = .30). Medication was perceived as the least efficacious treatment and was rated as significantly less likely to be effective than combined treatment (p < .001; d = 0.97) and psychotherapy (p < .001; d = .76), in addition to changing one's attitude and lifestyle.

PERCEPTIONS OF CAUSAL EXPLANATIONS BY PARTICIPANTS' TREATMENT HISTORY

Participants who have received mental health treatment may have different views of chemical imbalance and biopsychosocial explanations of depression than those without a history of such treatment. To examine this possibility, we conducted a series of 2 (causal explanation) x 2 (treatment history) mixed ANOVAs on each of the seven perceptions of depression. Because most (53.8%) of the 26 participants with a mental health treatment history had received both psychotherapy and medication, we combined these participants into a single group. Given the exploratory nature of these analyses we elected to use a Bonferroni-corrected alpha level of .007 to control for Type I error. Among the seven interaction terms of interest, only the interaction for prognosis was statistically significant, F (1, 84) = 8.33, p < .005. Follow-up paired samples t-tests indicated that par-

ticipants without a treatment history reported a worse prognosis for the chemical imbalance explanation (M = 5.50, SD = 2.86) than the biopsychosocial explanation (M = 8.68, SD = 2.85), t (60) = 7.00, p < .001, d = 1.11. In contrast, those with a treatment history reported a comparable prognosis for the chemical imbalance explanation (M's = 6.76, SD = 3.84) and biopsychosocial explanation (M = 7.20, SD = 2.20), t (24) = 0.54, p > .50, d = .14.

PERCEPTIONS OF CAUSAL EXPLANATIONS BY PRE-EXISTING BELIEFS ABOUT DEPRESSION

Participants' perceptions of the chemical imbalance and biopsychosocial explanations of depression may have been influenced by their pre-existing beliefs about the actual cause of this disorder. For example, individuals who endorse a chemical imbalance explanation might have more favorable perceptions of depression and its treatment (e.g., less stigma, better prognosis) in the context of a chemical imbalance rather than biopsychosocial rationale. To examine this possibility, we examined whether participants who endorsed a primarily biochemical (n = 17) or psychological/environmental (n = 73) cause of depression responded differently to the two causal explanations of depression in this study. Specifically, we conducted 2 (causal explanation) x 2 (pre-existing belief in a chemical imbalance vs. psychological/environmental cause of depression) mixed ANOVAs on each of the seven perceptions of depression using a Bonferroni-corrected alpha level of .007.

A significant causal explanation x pre-existing causal belief interaction was found for credibility, F (1, 88) = 18.20, p < .001. Participants endorsing a chemical imbalance cause of depression rated the two causal explanations as equally credible (both M's = 2.76), whereas those endorsing a psychological/environmental explanation rated the biopsychosocial explanation (M = 3.23, SD = 0.70) as substantially more credible than the chemical imbalance explanation (M = 1.79, SD = 0.99), t (72) = 9.60, p < .001, d = 1.68. Pre-existing causal beliefs about depression did not interact with perceptions of stigma (p > .10) or prognosis (p > .10), and none of the interactions involving the four treatment variables reached the Bonferroni-corrected level of statistical significance.

EFFECTS OF CREDIBILITY OF CAUSAL EXPLANATIONS ON RATINGS OF OTHER PERCEPTIONS OF DEPRESSION

It is possible that differences in the credibility of the two causal explanations might have influenced subsequent ratings of stigma, prognosis, and treatment perceptions. To illustrate, individuals who rated the biopsychosocial explanation as more credible than the chemical imbalance explanation might have reported more favorable perceptions of stigma, prognosis, and treatment effectiveness when depression was attributed to biopsychosocial vs. biochemical factors. To address this possibility, we conducted separate repeated measures ANOVAs for stigma composite scores, prognosis composite scores, and the perceived effectiveness of each depression intervention, with credibility (calculated as the difference in credibility ratings between the two causal explanations) entered as a covariate. Despite the fact that participants rated the biopsychosocial explanation as much more credible than the chemical imbalance rationale, the pattern of significant differences between the two explanations reported in Table 2 remained unchanged. Specifically, the chemical imbalance explanation was associated with significantly less stigma ($p < .001$) and higher perceived effectiveness of medication ($p < .001$), whereas the biopsychosocial explanation was associated with a better prognosis ($p < .05$) and greater perceived efficacy of psychotherapy ($p < .001$) and changing one's attitude and lifestyle ($p < .001$).

DISCUSSION

The purpose of this study was to examine how a chemical imbalance explanation of depression affects individuals' perceptions of themselves, their prospects for recovery, and the perceived effectiveness of different treatment approaches. Compared to a biopsychosocial explanation, the chemical imbalance explanation was less credible and led to decreased personal and moral responsibility for depression, a worse expected prognosis, and the perception that psychosocial interventions are largely ineffective. A history of mental health treatment and pre-existing etiological beliefs about depression had little effect on these findings. Our results are consistent with pre-

vious research in demonstrating the potentially negative effects of attributing psychological problems to purely biological causes and highlight the need for research on whether these effects might extend to treatment-seeking individuals with depression.

As hypothesized, participants perceived themselves as less responsible and morally culpable for being depressed when their symptoms were attributed to faulty brain chemistry as opposed to biopsychosocial factors. Although the reduction of self-blame is an important outcome, accepting some level of responsibility for one's symptoms may not necessarily be harmful. Some authors (e.g., Read, 2007) have expressed concern that the apparent benefit of reduced self-blame comes at the expense of a failure to acknowledge nonbiological factors that contribute to mental health symptoms. Indeed, research suggests that biological explanations of mental illness discourage attention toward situational factors such as occupational and marital problems (Schreiber & Hartrick, 2002) and foster the perception that thinking about the cause and solution to mental problems is of little value (Fisher & Farina, 1979). In the present study, making changes in one's attitude and lifestyle was deemed, on average, only slightly helpful in the context of a chemical imbalance explanation but very helpful when depression was described as biopsychosocial in nature. Thus, it appears that in reducing personal responsibility for developing a mental disorder the biological model may encourage individuals to disregard potentially important psychosocial causal factors and coping strategies. As a result, depressed individuals exposed to the brain disease model might be inclined to dismiss lifestyle interventions with demonstrated efficacy such as behavioral activation (Dimidjian et al., 2006) and physical exercise (Stathopoulou, Powers, Berry, & Smits, 2006).

Participants in the present study reported that the biochemical explanation would likely improve others' attitudes toward them (i.e., reduce public stigma). This finding is at odds with consistent findings that persons with mental disorders are reliably judged most negatively when their symptoms are ascribed to biological factors (Read, 2007; Read, Haslam, Sayce, & Davies, 2006). Why might this be the case? Because biological models foster the perception that the mentally ill lack control over their behavior, such individuals may be viewed by the public as unpredictable, dangerous, unable to care for themselves, requiring harsher treatment, and fundamentally different from those without mental disorders (Angermeyer

& Matschinger, 2005; Hill & Bale, 1981; Mehta & Farina, 1997). Unfortunately, the most popular and widely disseminated biological models of psychopathology (e.g., NAMI's assertion that "mental illnesses are biologically based brain disorders") appear especially likely to worsen the very public stigma they seek to reduce.

Consistent with our hypothesis, the chemical imbalance explanation was associated with a worse expected prognosis than a biopsychosocial explanation. This was evident with respect to the perceived curability and chronicity of depression, self-efficacy in controlling depression oneself, and the need for long-term treatment. These results extend previous research conducted with the general public (e.g., Phelan et al., 2006) and suggest that biological models of mental illness might elicit pessimism about the chances of recovery among treatment-seeking patients. However, our results also indicate that treatment expectancies are highly dependent on the congruency between specific etiological models and interventions. Consistent with the findings of Iselin and Addis (2003), participants in the present study rated treatments involving medication as very likely to be effective in the context of a chemical imbalance explanation of depression. Psychosocial approaches were rated as highly effective for depression caused by biopsychosocial factors. It is important to note that congruency between causal beliefs and treatment modality may not necessarily produce a superior treatment response. To illustrate, Sullivan et al. (2003) reported that belief in a biological cause of depression was associated with a worse response to Paxil among depressed primary care patients. Thus, even when one's etiological beliefs and treatment modality appear congruent, the potentially deleterious effects of adopting a biological model (e.g., a passive or fatalistic attitude toward depression; Sullivan et al., 2003) may impair one's ability to benefit from treatment.

Our findings should be interpreted within the context of several limitations. First, because many undergraduate participants were psychology majors, they may have been predisposed toward more favorable views of a biopsychosocial than a chemical imbalance explanation of depression. The fact that approximately 80% of the sample believed depression was caused largely by psychological/environmental factors supports this conclusion. On the other hand, beliefs about the cause of depression as well as the greater credibility of the biopsychosocial rationale had little effect on perceptions of the explanations, suggesting that the observed differences

between the chemical imbalance and biopsychosocial models were minimally confounded by pre-existing biases regarding etiology. Second, owing to our use of undergraduate student participants, most of whom were young, Caucasian, and of rural origin, the extent to which our results extend to the general population is unclear. This concern may be mitigated somewhat by the observation that causal attributions for depression are generally similar across different cultures (Dietrich et al., 2004). Finally, it is unclear to what extent results of the present thought experiment might generalize to the actual experience of clinical patients in healthcare settings. The present methodology avoids the practical difficulties inherent in randomly assigning treatment-seeking depressed patients to receive different causal explanations from their healthcare providers, as well as the potential ethical concerns involved with assigning depressed individuals to receive a potentially iatrogenic explanation for their symptoms. While this study may be considered a useful first step in investigating this topic, future research should employ designs that more authentically simulate actual clinical practice.

In summary, the present study suggests that the chemical imbalance explanation of depression reduces self-stigma but negatively affects perceptions of prognosis and the effectiveness of nonbiological treatments. Our findings also demonstrate the beneficial effects of a *biopsychosocial* explanation relative to the biochemical explanation (see also Walker & Read, 2002). The biopsychosocial model is particularly appealing owing to its scientific validity, credibility, and association with the perception that depression is more curable, controllable, expediently treated, and responsive to a variety of treatment approaches. The biopsychosocial model also acknowledges the importance of biological contributions to mental health problems but places them within the context of relevant psychological and environmental influences. As others have noted (e.g., Read, 2005), there exists ample scientific evidence to dispel the practice of ascribing mental illness entirely to biological factors in order to reduce stigma. However, given the powerful commercial and institutional forces aligned with the medicalization of mental health problems, the brain disease model will likely persist despite the convincing evidence from experimental treatment studies and etiology studies that has long shown the model to be deficient (Leo & Lacasse, 2008). Exposure to the brain disease model of depression is pervasive in the United States (France et al., 2007) and appears

to dramatically affect the self-perceptions and treatment expectancies of depressed patients (e.g., Schreiber & Hartrick, 2002). Future research should investigate the frequency with which patients receive biochemical explanations from their treatment providers, and how such explanations affect patients' responses to mental health interventions. The widespread dissemination of this model has potentially far-reaching public health implications, and additional research on the effects of biological models on real-world clinical outcomes is needed.

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