



Shorter communication

Effects of a chemical imbalance causal explanation on individuals' perceptions of their depressive symptoms

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ARTICLE INFO

Article history:

Received 2 November 2013

Received in revised form

10 January 2014

Accepted 27 February 2014

Available online 6 March 2014

Keywords:

Depression

Chemical imbalance

Stigma

Prognosis

Etymology

ABSTRACT

Although the chemical imbalance theory is the dominant causal explanation of depression in the United States, little is known about the effects of this explanation on depressed individuals. This experiment examined the impact of chemical imbalance test feedback on perceptions of stigma, prognosis, negative mood regulation expectancies, and treatment credibility and expectancy. Participants endorsing a past or current depressive episode received results of a bogus but credible biological test demonstrating their depressive symptoms to be caused, or not caused, by a chemical imbalance in the brain. Results showed that chemical imbalance test feedback failed to reduce self-blame, elicited worse prognostic pessimism and negative mood regulation expectancies, and led participants to view pharmacotherapy as more credible and effective than psychotherapy. The present findings add to a growing literature highlighting the unhelpful and potentially iatrogenic effects of attributing depressive symptoms to a chemical imbalance. Clinical and societal implications of these findings are discussed.

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Biomedical causal explanations of depression, principally the “chemical imbalance” theory, have been vigorously promoted in recent decades to reduce public stigma and facilitate pharmacotherapy (Lacasse & Leo, 2005). As a result, the chemical imbalance theory has become the dominant cultural understanding of depression in the United States (France, Lysaker, & Robinson, 2007). Anti-stigma initiatives by the National Alliance for Mental Illness (NAMI) portray depression as a “chronic medical illness” (NAMI, 2013). Characterizing depression in biomedical terms is assumed to reduce stigma according to attribution theory, which predicts that attributing a mental disorder to an uncontrollable cause reduces blame among observers (Corrigan, 2000). However, increased public endorsement of the chemical imbalance explanation has not resulted in improved attitudes toward depressed individuals (Pescosolido et al., 2010). Indeed, research findings suggest that biomedical causal explanations for depression do not reliably reduce blame and may worsen perceptions of dangerousness and unpredictability (Kvaale, Gottdiener, & Haslem, 2013).

Biomedical explanations for mental disorders may produce essentialist thinking, in which biological causes suggest inherent differences in the nature of sufferers (Boysen & Gabreski, 2012;

Haslam, 2000, 2011; Phelan, 2005). An essentialist perspective views biologically-based mental disorders as deep-seated, immutable defects which make an individual categorically distinct from others. One predicted consequence of this perspective is prognostic pessimism, the belief that the problem is unlikely to respond to remedial action (Dar-Nimrod & Heine, 2011; Haslam, 2011). In studies of public attitudes toward individuals with mental disorders, prognostic pessimism appears to be worsened by biomedical causal explanations (e.g., Bennett, Thirlaway, & Murray, 2008; Phelan, 2005; Phelan, Yang, & Cruz-Rojas, 2006). Although studies of the attitudes of laypersons are necessary to inform efforts to reduce public stigma, such research does not address a question of critical importance to clinicians: how do biomedical causal explanations affect how individuals with mental disorders view *their own* symptoms?

At the time of this writing, only two empirical studies have examined the effects of biomedical causal attributions on individuals' perceptions of their depressive symptoms. In a preliminary investigation using an analog sample and thought experiment methodology, Deacon and Baird (2009) found that a chemical imbalance explanation reduced self-blame in comparison to a biopsychosocial explanation, but also decreased self-efficacy in managing depression, increased prognostic pessimism, and fostered the perception that psychotherapy would be less effective than medication. A web-based correlational study of individuals

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with marked depressive symptoms by [Lebowitz, Ahn, and Nolen-Hoeksema \(2013\)](#) found that endorsement of biochemical and genetic causes of depression was associated with greater prognostic pessimism. The clinical relevance of these findings is underscored by the well-established relationship between prognostic expectancies and actual prognosis ([Rutherford, Wager, & Roose, 2010](#)). Prognostic expectancies are a primary mechanism of the placebo effect and account for the majority of the improvement observed in treatments for depression ([Kirsch, 2010](#)). The finding that a chemical imbalance explanation reduced self-efficacy in controlling depression oneself ([Deacon & Baird, 2009](#)) suggests that this causal attribution may affect depressed individuals' perceived ability to regulate their own negative moods. Negative mood regulation expectancies affect individuals' coping behaviors and directly influence depressed mood ([Kirsch, Mearns, & Catanzaro, 1990](#)). Because negative mood regulation expectancies are based on the perceived ability to change one's mood state, belief in a deterministic biomedical causal explanation may lessen the extent to which depressed individuals view their symptoms as under their own control.

Despite a wealth of speculation and anecdotal reports on the potentially detrimental effects of biomedical causal explanations on individuals with mental health problems (e.g., [Cohen & Hughes, 2011](#); [Deacon & Lickel, 2009](#); [France et al., 2007](#); [Whitaker, 2010](#)), experimental research has yet to examine how biomedical attributions affect depressed individuals' perceptions of themselves and their symptoms. Particular interest surrounds the effects of the ubiquitous chemical imbalance explanation on depressed individuals' self-stigma, perceived prognosis, negative mood regulation expectancies, and treatment expectancies. Given the popularity of the chemical imbalance explanation of depression in both clinical and societal contexts ([Deacon, 2013](#); [France et al., 2007](#)), it is essential to understand the consequences of endorsing this causal explanation of one's own depressive symptoms.

To our knowledge, the present investigation is the first to experimentally examine the effects of the chemical imbalance explanation on perceptions of stigma, prognostic pessimism, and treatment expectancies among individuals with depressive symptoms. In an attempt to approximate the direct, face-to-face causal feedback treatment-seeking individuals might receive from healthcare providers, participants reporting having experienced an episode of depression were provided with the results of a bogus but credible biological test indicating that their symptoms were or were not caused by a chemical imbalance in the brain. It was hypothesized that test results indicating a chemical imbalance cause of depressive symptoms, as opposed to test results indicating no chemical imbalance, would result in: (a) no improvement in self-blame, (b) worse perceived prognosis, (c) lower negative mood regulation expectancies, (d) the perception that pharmacological treatment would be more credible than psychotherapy, and (e) the expectation that pharmacological treatment would be more effective than psychotherapy.

Method

Participants

Participants were recruited from an undergraduate psychology participant pool at the University of Wyoming and were eligible to participate if they endorsed a past or current depressive episode on an online depression screening item. Ninety-one individuals agreed to participate in response to an e-mail invitation and were randomly assigned to either the chemical imbalance condition or the control condition. At the end of the study, a two-question

measure was administered to assess the credibility of the Rapid Depression Test (see below). Only participants who reported the manipulation to be sufficiently credible, according to a-priori criteria, were included in the analyzed sample. The final sample included 73 participants, 37 of whom were randomized to the chemical imbalance condition and 36 of whom were randomized to the control condition.

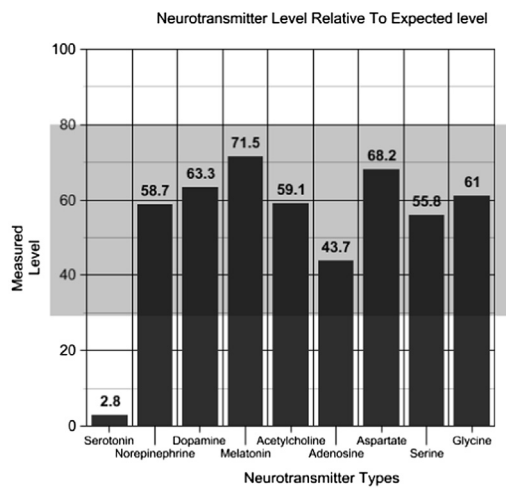
The sample had a mean age of 20.0 ($SD = 4.95$) years, and most participants were women (64.4%) and Caucasian (94.5%). Thirteen participants (17.8%) reported receiving a past or present diagnosis of clinical depression from a treatment provider, and more participants had been prescribed medication ($n = 18$) than had participated in psychotherapy ($n = 8$) for their depression. Baseline characteristics were evaluated to determine the groups' appropriateness for comparison. Only gender differed significantly ($p < .05$) between conditions, with significantly more women randomized to the control condition than the chemical imbalance condition, $\chi(1) = 5.56, p < .05$. Thus, the conditions demonstrated an appropriate level of baseline equivalence to permit direct comparison in subsequent analyses.¹

Procedure

Participants were randomly assigned to the chemical imbalance condition or the control condition. Following informed consent and collection of demographic information, participants were administered the "Rapid Depression Test" (RDT). The RDT was described as a test of neurotransmitter levels whose results would allow participants to determine whether or not their depressive episode(s) were caused by a chemical imbalance in the brain. Participants were led to believe the purpose of the study was to improve understanding of how individuals respond to learning the cause of their depression, before release of the RDT into clinical practice. The test procedure entailed swabbing the inside of the participant's cheek with a sterile cotton swab and placing the cotton swab into a sterile collection container. Next, the experimenter (a male undergraduate research assistant wearing a lab coat) instructed participants that he was leaving the experiment room to take their saliva sample to the lab and run the test. The experimenter returned 10 min later with the condition-specific results of the RDT. In the chemical imbalance condition, participants were informed that test results indicated their current or past depression to be caused by an imbalance in the neurotransmitter serotonin. Participants were presented with a bar graph of their test results (see [Fig. 1](#)) depicting very low serotonin levels relative to levels of other neurotransmitters, all of which were in the normal range. In the control condition, participants were told their past/current depression was not the result of a chemical imbalance, based on purported test results (and a corresponding bar graph) indicating that all neurotransmitter levels were in the normative range.² After receiving the results of the RDT, participants completed the post-manipulation measures packet (CADS, PDS, NMR, CEQ, and DCQ; see below for measure details). Participants were subsequently debriefed and completed the Deception Credibility Questionnaire to assess the credibility of the manipulation. Compensation for participation was provided in the form of course credit. This study was reviewed and approved by the University of Wyoming institutional review board and was conducted in accordance with the provisions of the World Medical Association Declaration of Helsinki.

¹ Entering gender as a covariate yielded a pattern of findings nearly identical to those presented below.

² The test feedback script for the chemical imbalance and no-chemical-imbalance conditions can be obtained from the corresponding author upon request.



CONCLUSION: 99.97% probability of serotonin deficiency.

Fig. 1. Results of the rapid depression test in the chemical imbalance condition.

Measures

Depressed Mood Screener. Participants were asked, “Have you ever experienced a period of at least two weeks during which your mood was depressed most of the day, nearly every day, that was not a normal response to a significant loss in your life (such as the death of a loved one)?” This item was intended to assess criterion A.1 for major depressive disorder in the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (APA, 2000)*. A single depressed mood screening item was used in lieu of a self-report inventory of depressive symptoms in order to recruit participants who endorsed having experienced an episode of depressed mood, thereby increasing the face validity of the study manipulation (see below). Not all individuals who report high levels of depressive symptoms on self-report measures, such as the participants with Beck Depression Inventory – II (Dozois, 2010) scores ≥ 16 recruited by Lebowitz et al. (2013), may identify themselves as depressed or having experienced an episode of depressed mood.

Causal Attributions for Depression Scale. Participants rated the extent to which nine factors (e.g., life stress, negative thinking pattern, genetic predisposition) caused their depressed mood on a 5-point scale ranging from 1 (“definitely not the cause”) to 5 (“definitely the cause”). Among these factors, one item (“chemical imbalance”) assessed the causal attribute relevant to the manipulation. Analysis of between-group differences on this item served as a manipulation check of test feedback purporting to demonstrate a chemical imbalance cause of depression.

Perceptions of Depression Scale (PDS). Two scales from the PDS (Deacon & Baird, 2009) were used to assess self-stigma and prognostic pessimism. The four-item stigma scale measures self-blame and the extent to which blame is expected and deserved from others (sample item: “To what extent would you feel personally responsible for having developed depression?”). The four-item prognosis subscale assesses the extent to which one’s depression is perceived as chronic, uncontrollable, and deserving of long term treatment (sample item: “To what extent would you believe you could eventually recover from your depression?”). Respondents indicate their agreement with each item on a 5-point scale ranging from 0 (“not at all”) to 4 (“extremely”). Total scores on the PDS

stigma and prognosis scales range from 0 to 16, with higher scores denoting increased perceptions of stigma and increased prognostic optimism, respectively. Deacon and Baird (2009) reported that the PDS stigma and prognosis scales loaded onto separate factors in a principal components analysis, and that each scale possessed adequate internal consistency (α s ranged from .79 to .83 for the stigma scale and .68 to .73 for the prognosis scale). In the present study, the PDS prognosis scale demonstrated adequate internal consistency ($\alpha = .78$), whereas the PDS stigma subscale demonstrated minimally acceptable internal consistency ($\alpha = .60$).

Negative Mood Regulation Scale (NMR). The NMR scale (Catanzaro & Mearns, 1990) is a 30-item questionnaire measuring expectancies of the ability to regulate one’s negative mood states. This measure contains three 10-item subscales assessing expectancies for successfully regulating negative moods in general (e.g., “I can usually find a way to cheer myself up”), as well as through specific cognitive (e.g., “I’ll feel okay if I think of more pleasant times”) and behavioral (e.g., “I can feel better by treating myself to something I like”) strategies. The NMR scale has demonstrated high internal consistency as well as adequate construct and discriminant validity (Catanzaro & Mearns, 1990). All items have the same stem: “When I’m upset, I believe that...” and responses are given on a 5-point scale ranging from 1 (“strong disagreement”) to 5 (“strong agreement”). For the present study the stem was changed to: “When I am depressed, I believe that...” in order to assess expectancies specific to depressed mood as opposed to general negative mood. A NMR total score (range = 30–150) was calculated by summing all 30 items; subscale scores were also calculated for the 10-item general, cognitive, and behavioral subscales (range = 10–50). The total, general, cognitive and behavioral subscales demonstrated adequate internal consistency in the present study ($\alpha = .90, .84, .76, .65$, respectively).

Credibility and Expectancy Questionnaire (CEQ). The CEQ is a well-established measure of treatment credibility and expectancy that has demonstrated good test-retest reliability and internal consistency (Deville & Borkovec, 2000). Credibility (e.g., “At this point, how logical does the above treatment seem?”) and expectancy (e.g., “At this point, how much do you really feel that this treatment would help you to reduce your depressive symptoms?”) subscale totals in the current investigation were derived using the scoring procedure established by Nock, Ferriter, and Homberg (2007). Specifically, items 1, 2, 3, and 5 were scored on a nine-point scale ranging from 1 (“not a lot of sense/no improvement”) to 9 (“a lot of sense/very much improvement”), and items 4 and 6 were scored on an 11-point scale (e.g., 0–100%). Items 4 and 6 were recoded to accord with the 1 to 9 scale used with items 1–3 and 5, such that values in the 40–60% range were collapsed into one value (i.e., 5). Thus, each subscale had a range from 3 to 27, with higher scores indicating increased credibility or expectancy associated with a particular treatment approach.

The CEQ was completed following paragraph-length descriptions of both psychotherapy and pharmacological treatment for depression.³ The psychotherapy description noted the efficacy of cognitive-behavioral therapy (CBT), briefly outlined its implementation, and characterized CBT as effective because of its benefits on maladaptive thoughts, participation in enjoyable activities, and problem-solving skills. The pharmacotherapy description noted the efficacy of selective serotonin reuptake inhibitors (SSRIs), briefly described their use, and characterized SSRIs as effective because of their ability to “restore the brain’s chemical balance by increasing the supply of serotonin in the brain.”

³ The descriptions of both the psychotherapy and pharmacological treatments presented to participants while completing the CEQ can be obtained from the corresponding author upon request.

Deception Credibility Questionnaire. To assess the credibility of the manipulation, participants were asked two questions: (a) “did you believe that the Rapid Depression Test was a real tool for determining the cause of depression,” and (b) “how much did you believe that the results of the Rapid Depression Test were correct?” Participants endorsed the first question with a simple “yes” or “no” response, and responded to the second question using a 5-point scale with the following response options: 0 = “Not at all,” 1 = “Very little,” 2 = “Some,” 3 = “Very much,” and 4 = “Extremely.” Determined on an a priori basis, participants were eligible for inclusion in the study if they responded “yes” to question (a) and answered question (b) with a response of ≥ 2 .

Results

Manipulation check

Participants in the chemical imbalance condition rated a chemical imbalance cause of their symptoms as significantly more likely than individuals in the control condition, $t(71) = 4.40$, $p < .001$, $d = 1.03$. Mean ratings of a chemical imbalance cause were 3.14 ($SD = 1.29$) and 1.86 ($SD = 1.18$) in the chemical imbalance and control conditions, respectively. Thus, chemical imbalance test feedback was successful in modifying participants' causal attributions.

Primary analyses

Table 1 presents descriptive statistics and results of between-group comparisons on the study measures. PDS stigma scale scores did not differ significantly between conditions, $t(71) = .06$, $p = .95$, $d = .01$. Given that scores on this measure were nearly identical between conditions, it is unlikely that significant differences would have emerged had this measure been more internally consistent than $\alpha = .60$. In contrast, a significant difference between conditions was obtained on the PDS prognosis scale, with lower scores in the chemical imbalance condition than the control condition, $t(71) = 2.11$, $p = .04$, $d = .50$. Compared to the control condition, the chemical imbalance condition produced significantly lower negative mood regulation expectancies on the NMR general subscale, $t(71) = 2.44$, $p = .02$, $d = .57$, and the NMR cognitive subscale, $t(71) = 2.17$, $p = .03$, $d = .51$, and approached significance

Table 1
Descriptive statistics and between-group comparisons on measures of self-stigma, prognostic pessimism, negative mood regulation expectancies, and treatment credibility and expectancy.

Measure	Chemical imbalance condition (n = 37)		Control condition (n = 36)		t(71)	p	d
	M	SD	M	SD			
PDS Stigma Scale	6.38	2.78	6.42	2.60	-.06	.95	.01
PDS Prognosis Scale	10.73	2.71	12.14	2.98	-2.11	.04	.50
NMR Total	100.73	15.60	107.44	15.62	-1.84	.07	.43
NMR General Subscale	34.46	6.41	37.97	5.90	-2.44	.02	.57
NMR Cognitive Subscale	30.38	5.61	33.42	6.33	-2.17	.03	.51
NMR Behavioral Subscale	35.89	5.37	36.06	5.00	-.14	.89	.03
<i>Pharmacological treatment</i>							
Credibility	20.71	4.77	13.67	6.72	5.17	.000002	1.21
Expectancy	17.95	4.32	13.89	5.97	3.33	.001	.78
<i>Psychosocial treatment</i>							
Credibility	17.22	3.86	15.11	5.79	1.83	.07	.43
Expectancy	14.81	4.29	15.19	4.79	-.36	.72	.08

Note. PDS = Perceptions of Depression Scale; NMR = Negative Mood Regulation Scale.

on the NMR total scale, $t(71) = 1.83$, $p = .07$, $d = .43$. However, conditions did not differ significantly on the behavioral subscale ($p = .89$). Taken together, these findings are consistent with hypotheses that chemical imbalance test feedback would not reduce self-blame, would increase prognostic pessimism, and would lower participants' perceived ability to regulate negative mood states.

A 2 (Condition: chemical imbalance vs. control) \times 2 (Treatment: pharmacotherapy vs. psychotherapy) repeated measures ANOVA was conducted on treatment credibility ratings. Results revealed a significant main effect for Condition, $F(1, 71) = 21.55$, $p < .001$, $\eta_p^2 = .23$, with the chemical imbalance condition scoring higher than the control condition. The main effect for Treatment was not significant, $F(1, 71) = 1.69$, $p = .20$, $\eta_p^2 = .02$. A significant Condition \times Treatment interaction emerged, $F(1, 71) = 9.86$, $p = .002$, $\eta_p^2 = .12$, and was decomposed via follow-up simple effects analyses. Participants in the chemical imbalance condition rated pharmacotherapy as more credible than psychotherapy, $t(36) = 4.00$, $p < .001$, $d = .80$. In contrast, participants in the control condition rated these treatments as equally credible, $t(35) = 1.10$, $p = .28$, $d = .23$. Thus, as hypothesized, chemical imbalance test feedback elicited the perception that pharmacological treatment was more credible than psychotherapy.

A similar analysis was conducted on treatment expectancy ratings. Results revealed a significant main effect for Condition, $F(1, 71) = 4.34$, $p = .04$, $\eta_p^2 = .06$, with higher scores in the chemical imbalance condition compared to the control condition. The main effect for Treatment was not significant, $F(1, 71) = 1.58$, $p = .21$, $\eta_p^2 = .02$. Lastly, the Condition \times Treatment interaction was significant, $F(1, 71) = 9.31$, $p = .003$, $\eta_p^2 = .12$. Follow-up simple effects analyses revealed that participants in the chemical imbalance condition rated pharmacotherapy as more likely to be effective than psychotherapy, $t(36) = 43.53$, $p < .001$, $d = .73$. In contrast, expectancies for pharmacotherapy and psychotherapy did not differ significantly in the control condition, $t(35) = 1.13$, $p = .27$, $d = .24$. Thus, as hypothesized, chemical imbalance test feedback elicited the perception that pharmacological treatment would be more effective than psychotherapy.

Discussion

This experiment was conducted to examine the effects of a chemical imbalance causal explanation on individual's perceptions of their own depressive symptoms. Participants who reported experiencing a depressive episode were given bogus but credible test feedback showing their depressive symptoms to either be caused, or not caused, by a chemical imbalance in the brain. As hypothesized, chemical imbalance test feedback increased prognostic pessimism, lowered negative mood regulation expectancies, and led participants to view pharmacotherapy as more credible and effective than psychotherapy. These effects were not offset by reduced stigma, as chemical imbalance feedback had no effect on self-blame. Overall, the present findings suggest that providing individuals with a chemical imbalance causal explanation for their depressive symptoms does not reduce stigma and activates a host of negative beliefs with the potential to worsen the course of depression and attenuate response to treatment, particularly psychotherapy.

To our knowledge, this is the first experimental study to demonstrate that a chemical imbalance causal attribution causes prognostic pessimism in individuals who experience depressive symptoms. The present findings conceptually replicate previous research (Deacon & Baird, 2009; Lebowitz et al., 2013) and demonstrate that attributing depressive symptoms to a chemical imbalance in the brain causes individuals to view their symptoms as more chronic and intractable. Chemical imbalance feedback was

also found to lower individuals' perceived ability to successfully regulate their depressed moods, particularly via cognitive mood regulation strategies (e.g., trying to understand why one is depressed). The observed detrimental effects of a chemical imbalance causal attribution on prognostic pessimism and negative mood regulation expectancies are consistent with neuro-essentialism theory (e.g., Dar-Nimrod & Heine, 2011; Haslam, 2011), which posits that a mental health problem ascribed to brain abnormalities will be perceived as stable and resistant to corrective action. Notably, emphasizing the malleability of biological influences on depression appears to foster less prognostic pessimism and hopelessness among depressed individuals (Lebowitz et al., 2013), suggesting that neuroessentialist beliefs may be modified with corrective information.

Although biomedical causal explanations are predicted by attribution theory to reduce stigma by invoking uncontrollability attributions (Corrigan, 2000), chemical imbalance test feedback did not reduce self-stigma in the present study. This finding contradicts results from Deacon and Baird's (2009) thought experiment and suggests that the effects of chemical imbalance attributions on blame may differ between the imagined and actual experience of depressive symptoms. The present findings complement the public stigma literature in demonstrating that attributing depression to biomedical causes does not improve perceptions of blame and responsibility (Pescosolido et al., 2010).

As hypothesized, chemical imbalance test feedback resulted in the perception that pharmacotherapy was more credible and more likely to be effective than psychotherapy. Previous research has found that treatment expectancies reflect the congruency between etiological explanations and interventions (e.g., Iselin & Addis, 2003). This reality is well known to the pharmaceutical industry, which promotes the chemical imbalance explanation of depression in order to facilitate sales of medications that purportedly correct chemical imbalances (Lacasse & Leo, 2005). Although our findings suggest that a chemical imbalance causal attribution increases the attractiveness of pharmacotherapy for depression, the opposite appears to be the case for psychotherapy. Consistent with the results of Deacon and Baird (2009), the present study found that psychotherapy is perceived as less likely to be effective when participants believe a chemical imbalance is to blame for their depressive symptoms. Given that a chemical imbalance causal explanation reduces the credibility and expected effectiveness of psychotherapy relative to medication, patients who endorse a chemical imbalance cause of their symptoms may be more likely to seek pharmacotherapy than psychotherapy. Widespread exposure to the chemical imbalance causal explanation in clinical and societal contexts (Deacon, 2013) may direct consumers seeking treatment for depression to pharmacotherapy instead of psychotherapy despite the favorable cost–benefit profile of psychotherapy (Kirsch, 2010).

This study has several limitations. First, participants were a convenience sample of undergraduate student participants, most of whom were young, Caucasian, and of rural origin. As a result, the generalizability of the present findings to the general population is unclear. A second limitation concerns our decision to screen depressed individuals using a single item rather than a validated inventory of depressive symptoms in order to increase the face validity of the experimental manipulation. Ensuring that participants reported both a history of depressive episodes and high scores on a measure of depressive symptoms would have provided even stronger evidence that our participants were a clinically representative sample. Third, providing bogus chemical imbalance test feedback to an analog sample of undergraduate students reporting a history of depressive episodes is an admittedly artificial approximation of typical interactions between patients and

healthcare providers. A clinically representative study might randomly assign treatment-seeking depressed individuals to receive different causal explanations from healthcare providers. However, this study design may not be ethical as it risks interfering with patients' treatment (Lam & Salkovskis, 2007). Although correlational studies are useful in examining the association between biomedical causal attributions and predicted outcomes (e.g., Lebowitz et al., 2013), it is possible that experimental investigations of this association may only feasibly be conducted using analog samples. Under certain circumstances an analog approach provides unique strengths, even when the objective is to better understand a clinical phenomenon (Tull, Bornovalova, Patterson, Hopko, & Lejuez, 2008), and the sample and procedure used in this study are an example of such an exception.

In summary, the present experiment demonstrated that chemical imbalance feedback provided to individuals reporting depressive symptoms failed to reduce stigma, worsened prognostic pessimism and negative mood regulation expectancies, and led pharmacotherapy to be perceived as more credible and effective than psychotherapy. Findings from the present study, taken together with previous research on the effects of biomedical causal attributions on self-stigma (e.g., Lebowitz et al., 2013) and public stigma (e.g., Pescosolido et al., 2010), suggest that biomedical causal explanations of depression, principally the chemical imbalance theory, convey no reliably discernable psychological benefits and foster beliefs that may interfere with recovery and response to treatment, particularly psychotherapy. As an alternative approach, clinicians, scientists, and advocates seeking to promote positive beliefs about depressed individuals and the nature and treatment of depression itself are encouraged to disseminate a biopsychosocial perspective which acknowledges the contribution of biological influences but avoids the potentially iatrogenic effects of attributing depression to purely biomedical causes (Deacon, 2013).

Acknowledgment

The authors are grateful to Karis Rowley, B.A., for his assistance with data collection.

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