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Shorter communication

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

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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.

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.