Behaviour Research and Therapy 71 (2015) 125–130

Contents lists available at ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Shorter communication

Emphasizing Malleability in the biology of depression: Durable effects on perceived agency and prognostic pessimism

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ARTICLE INFO

Article history:
Received 14 March 2015
Received in revised form
4 June 2015
Accepted 9 June 2015
Available online 10 June 2015

Keywords:
Depression
Psychoeducation
Biological explanations
Prognostic pessimism
Agency

ABSTRACT

Biological attributions for depression, which are currently ascendant, can lead to prognostic pessimism—the perception that symptoms are relatively immutable and unlikely to abate (Kvaale, Haslam, & Gottdiener, 2013; Lebowitz, Ahn, & Nolen-Hoeksema, 2013). Among symptomatic individuals, this may have important clinical ramifications, as reduced confidence in one's own ability to overcome depression carries the risk of becoming a self-fulfilling prophecy. Previous research (Lebowitz, Ahn, et al., 2013) has demonstrated that educational interventions teaching symptomatic individuals about how the effects of genetic and neurobiological factors involved in depression are malleable and can be modified by experiences and environmental factors can reduce prognostic pessimism. While previous research demonstrated such effects only in the immediate term, the present research extends these findings by testing whether such benefits persist six weeks after the intervention. Indeed, among individuals who initially considered biological factors to play a major role in influencing their levels of depression, exposure to malleability-focused psychoeducation reduced levels of depression-related prognostic pessimism and stronger belief in their ability to regulate their moods. Critically, this benefit persisted six weeks after the intervention. Clinical implications of the findings are discussed.

Biological attributions and explanations for mental disorders, including depression, are currently in ascendancy. Large majorities of the American public view neurochemical imbalances and genetic abnormalities as causes of depression (Pescosolido et al., 2010). In general, biomedical approaches to understanding, studying and treating psychopathology have become predominant (Deacon, 2013; Kemp, Lickel, & Deacon, 2014).

Although biological conceptualizations of psychopathology have been touted for their well-documented power to reduce the blame ascribed to sufferers for their own symptoms, they also have notable negative consequences among members of the general public; for example, they can increase *prognostic pessimism*—the perception that disorders are unlikely to remit (Kvaale, Haslam, & Gottdiener, 2013). This effect may occur because biological explanations can lead to so-called "essentialist" assumptions, in which abnormalities in individuals' brains or genes come to be seen as deep-seated, fundamental, immutable essences of their symptoms (Dar-Nimrod & Heine, 2011; Haslam, 2011; Medin & Ortony, 1989).

In recent years, several studies have examined the effect of biological attributions specifically among people who display psychiatric symptoms (Lebowitz, 2014). For example, among people with elevated levels of depressive symptomatology, attributing one's symptoms to neurochemical or genetic causes is associated with pessimistic expectations about the duration of one's own depression (Lebowitz, Ahn, & Nolen-Hoeksema, 2013). Relatedly, individuals who were told that they carried a gene associated with alcoholism rated themselves as less able to avoid drinking alcohol, compared to individuals who were told that they did not carry such a gene (Dar-Nimrod, Zuckerman, & Duberstein, 2013). Biological explanations for generalized anxiety disorder also increased prognostic pessimism among people whose self-report suggested the presence of the condition (Lebowitz, Pyun, & Ahn, 2014). More recently, when individuals with depression were given purported biological test results indicating that their symptoms were caused by a neurochemical imbalance, it increased their pessimism about their own prognoses and decreased their belief in their ability to regulate their own moods (Kemp et al., 2014).

The clinical implications of the aforementioned findings represent an important cause for concern. Specifically, the prognostic expectancies of people with depression and other disorders are significant predictors of actual clinical outcomes and

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http://dx.doi.org/10.1016/j.brat.2015.06.005
0005-7967/

responsiveness to treatment (Greenberg, Constantino, & Bruce, 2006; Mondloch, Cole, & Frank, 2001; Rutherford, Wager, & Roose, 2010). That is, when people are pessimistic about their own prospects of overcoming a disorder or benefitting from treatment—which may be especially likely if they attribute their symptoms to biological causes—their negative outlooks can become self-fulfilling prophecies.

However, essentialist beliefs about the biology of depression and other mental disorders are inconsistent with current science. The brain maintains the ability to change and adapt to environments and experiences, known as neuroplasticity, well into adulthood (Lozano, 2011), and psychiatric treatments—including non-biomedical psychotherapies—cause observable neurobiological changes in patients (Linden, 2006). The relationship between genes and risk for psychiatric disorders, including depression, can also be moderated by environments and experiences, and in some cases these factors can lead to epigenetic changes that chemically alter gene expression (Lau & Eley, 2010; Rutter, Moffitt, & Caspi, 2006). Clearly, the assumption that biological understandings of psychopathology imply that symptoms are immutable and outside the control of their sufferers—an assumption upon which the common association of biological attributions with prognostic pessimism appears to be based—is misguided.

Such misunderstandings may be a promising target for intervention. In previous research, we developed a psychoeducation intervention, delivered in the form of a brief audiovisual presentation, which focused on the malleability of biological factors involved in depression, including information about neuroplasticity and how gene effects can be altered through epigenetics and gene-by-environment interactions (Lebowitz et al., 2013). Among people with and without elevated depressive symptomatology, this intervention significantly decreased prognostic pessimism, increased feelings of agency regarding the ability to positively influence one's own mood, and reduced generalized feelings of hopelessness.

However, these effects were observed immediately following administration of the intervention, leaving the question of whether such an intervention might have longer-term effects unresolved. A demonstration of long-term benefits would be critical to determining whether this kind of intervention might have useful real-world clinical applications.

Thus, in the present study, we tested whether the beneficial effects of a similar psychoeducation intervention would remain at follow-up after six weeks. We also measured participants' beliefs about the extent to which biological factors influenced their moods and levels of depression. Because the intervention was targeted at changing their views about the role of biology in depression, we hypothesized that it might be more effective among individuals who more strongly believed that biological factors (e.g., genes, neurochemistry) determine their moods and levels of depression.

1. Method

1.1. Participants and recruitment

Participants were recruited using the online Mechanical Turk system from Amazon.com, which allows users to complete short tasks in exchange for monetary compensation (Buhrmester, Kwang, & Gosling, 2011). The initial sample consisted of 454 US. adults (55.3% female, 43.4% male, 1.3% unknown gender; 83.9% White/Caucasian) ranging in age from 18 to 70 years ($M = 33.27$, $SD = 10.62$).

Six weeks after their initial ("T1") participation, participants were contacted and asked to provide follow-up ("T2") data (see *Procedures*). Mechanical Turk user IDs were used to match T2 responses to the T1 responses of the same participant. The follow-up sample ($n = 255$) was demographically comparable to the full initial sample. Specifically, T2 respondents were 52.5% female, 46.3% male, 1.2% unknown gender, and 86.7% White/Caucasian; they ranged in age from 18 to 70 years ($M = 34.63$, $SD = 11.14$).

1.2. Procedures

Study procedures were administered using Qualtrics.com online survey software. Upon initially providing informed consent at the beginning of T1, participants were randomly assigned to one of two conditions: intervention ($n = 227$) or control ($n = 227$). All participants then completed the Beck Depression Inventory-II (BDI-II), a well-validated and widely used measure of depression symptomatology (Dozois, 2010). (We omitted one BDI-II item, "Suicidal Thoughts or Wishes," because our online procedures precluded appropriate responses to reports of suicidality.) We collected this data to ensure that there was no systematic difference in depressive symptomatology between the two conditions. Indeed, there were no significant differences in BDI-II scores between participants in the intervention ($M = 13.22$, $SD = 11.36$) and control ($M = 13.52$, $SD = 11.35$) conditions, $t(452) = .28$, $p = .78$.

Next, participants were presented with a list of six factors that could plausibly lead to depressive symptoms (e.g., "Stress") and asked to "indicate the extent to which you believe each of the following factors is involved in determining your mood (for example, how much each factor affects whether or not you feel sad or depressed)." These ratings were made on a 7-point scale (1 = "Not at all" 7 = "Very much"). Two of the factors were biological: "Genetics" and "Neurobiology (e.g. brain chemistry)." The remaining items were fillers to disguise the true reason for these ratings (e.g. "Your childhood or the way you were raised," "Your environment or events that take place in your life").

Participants in the intervention condition were then presented with an onscreen audiovisual psychoeducation intervention in the form of a YouTube video, approximately 7 min in length. Based on a similar intervention developed for earlier research (Lebowitz et al., 2013), it focused on the malleability of biological factors involved in depression, including a primer on how genes can be "turned on and off" through epigenetic mechanisms and how brain chemistry and activity can be modulated through experience, including psychotherapy. Participants who watched the video were then instructed, before proceeding, to write a short letter to a depressed individual, using information from the video they watched, to persuade the person to see depression "in a new light." This approach took advantage of the "saying-is-believing" effect, a tendency for people to internalize viewpoints they have advocated (Aronson, Fried, & Good, 2002; Higgins, 1999; Lebowitz et al., 2013; Walton & Cohen, 2011). Participants in the control condition did not view any video or receive any other intervention and did not complete this "saying-is-believing" procedure.

All participants then completed the two T1 dependent measures. The first, which gauged participants' perceptions of their own agency in responding to possible future episodes of depression, was a version of the Negative Mood Regulation (NMR) scale (Catanzaro & Mearns, 1990; Kemp et al., 2014) that was modified for the present research. The original measure consists of a single stem ("When I'm upset, I believe that …") and asks respondents to rate each of 30 possible sentence-completing items (e.g., "… I can do something to feel better") on a five-point scale. To create our modified version, we removed some items (e.g., "Doing something nice for someone else will cheer me up") deemed irrelevant to the contents of our intervention, and altered others to increase their relevance (e.g., from "I can find a way to relax" to "Reducing my stress will help cheer me up"). That is, the intervention video did