Mental disorders are increasingly construed in terms of their biology, revolutionizing the conceptualization of psychopathology. Depression provides a compelling example of this conceptual shift: In 2006, 80% of Americans endorsed “a chemical imbalance in the brain,” as a cause of depression, and 64% endorsed “a genetic or inherited problem”—both figures representing double-digit increases from just 10 years earlier (Pescosolido et al., 2010).

Unfortunately, studies have found that attributing mental disorders (e.g., depression) to such biological causes is associated with prognostic pessimism among symptomatic individuals. Those with significant depressive symptomatology (a Beck Depression Inventory-II score of at least 16) rated their endorsement of biochemical and genetic causal attributions for their symptoms and indicated expected length of symptom duration. An audiovisual intervention emphasizing the malleability of gene effects and neurochemistry was developed, and its effects on symptomatic individuals’ prognostic pessimism, feelings of agency, guilt, and general hopelessness were measured.

**Results:** Biochemical and genetic causal attributions for depression were significantly associated with prognostic pessimism among symptomatic individuals. The malleability intervention significantly reduced prognostic pessimism, increased feelings of agency, and decreased general hopelessness. **Conclusions:** Biochemical and genetic attributions for depression are related to prognostic pessimism among individuals with depressive symptoms, and not just among the general public. However, emphasizing the malleability of gene effects and brain chemistry in depression can foster more optimism about depression-related beliefs.

**Keywords:** depression, prognostic pessimism, etiological explanations, biological attributions

**Supplemental materials:** [http://dx.doi.org/10.1037/a0031730.supp](http://dx.doi.org/10.1037/a0031730.supp)
clinetically important, as depressed individuals’ prognostic expec-
tancies can significantly affect their actual prognoses (Rutherford,
Wager, & Roose, 2010).

The second goal was to examine whether psychoeducation
about neuroplasticity and the malleability of gene effects can
reduce prognostic pessimism among symptomatic individuals.
Given the increasing prevalence of biological explanations for
psychopathology, ways of presenting them without increasing
prognostic pessimism are urgently needed, but research to date
has not focused on this need. For this aim, we drew inspiration from
previous research that has used educational interventions to dispel
perceptions of immutability and promote the concept of mallea-
ility in domains like intelligence and social belonging (Aronson,
Fried, & Good, 2002; Blackwell, Trzesniewski, & Dweck, 2007;

Studies 1a and 1b

Method

All study procedures were approved by the Institutional Review
Board. U.S. adults were recruited through Amazon.com’s Mecha-
nical Turk (mTurk) website in exchange for small payments. Re-
search has shown that mTurk participants tend to participate for
their own enjoyment, provide data whose quality is independent of
compensation rates, and are more demographically diverse than
standard Internet and undergraduate samples (Buhrmester, Kwang,
& Gosling, 2011). By recruiting from the general population rather
than a clinical sample, we precluded possible selection biases (e.g.,
biological imbalance)—were of primary interest. Endorsement
of these two causes were significantly correlated, r = .58, p < .01,
for Study 1a and r = .74, p < .01, for Study 1b, so we averaged
them to form a “biochemical/genetic attribution” score for each
participant. The other eight items, which served as fillers, were
“Day-to-day problems and/or stress,” “Beliefs of style of thinking
(cognitive factors),” “Abnormal brain structure/development,”
“Brain Injury,” “Substance Abuse,” “Weakness of Character,”
“Problems from childhood or the way you were raised,” and
“Recent traumatic events.” Biochemical and genetic attributions
had the strongest correlation of any two causal factors in both
studies, and factor analyses with maximum-likelihood extraction
and varimax rotation revealed that genetic and biochemical attri-
butions loaded onto the same factor (with respective loadings
of .85 and .68 in Study 1a and 1.0 and .74 in Study 1b). No other
attribute loaded onto this biochemical/genetic factor with a load-
ing above .42 in either study.

After rating causal attributions, participants answered the ques-
tion, “How long do you think that you will continue to feel sad,
blue, or depressed?” Study 1a used a 7-point scale comprising
Less than 1 week (coded as 1), 1 to 2 weeks, 2 to 4 weeks, 1 month to
6 months, 6 months to 1 year, More than 1 year, but not indefi-
nitely, and Indefinitely (coded as 7). Study 1b used a 9-point scale
for greater granularity. The first five and final scale points were the

Table 1
BDI-II Data for All Participants Who Completed the BDI-II in Each Study

<table>
<thead>
<tr>
<th>Study group</th>
<th>N</th>
<th>BDI-II range</th>
<th>BDI-II M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study 1a</td>
<td>108</td>
<td>16–50</td>
<td>24.41 (8.00)</td>
</tr>
<tr>
<td>Study 1b</td>
<td>40</td>
<td>16–56</td>
<td>25.42 (9.36)</td>
</tr>
<tr>
<td>Study 2 (BDI-II high scorers)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malleable condition</td>
<td>81</td>
<td>16–59</td>
<td>26.27 (9.70)</td>
</tr>
<tr>
<td>Control condition</td>
<td>65</td>
<td>16–46</td>
<td>24.69 (7.80)</td>
</tr>
<tr>
<td>Biological illness condition</td>
<td>86</td>
<td>16–54</td>
<td>25.24 (8.96)</td>
</tr>
<tr>
<td>Study 2 (BDI-II low scorers)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malleable condition</td>
<td>127</td>
<td>0–15</td>
<td>7.06 (4.35)</td>
</tr>
<tr>
<td>Control condition</td>
<td>127</td>
<td>0–15</td>
<td>7.06 (4.35)</td>
</tr>
<tr>
<td>Biological illness condition</td>
<td>131</td>
<td>0–15</td>
<td>6.65 (4.50)</td>
</tr>
<tr>
<td></td>
<td>127</td>
<td>0–15</td>
<td>6.95 (4.56)</td>
</tr>
</tbody>
</table>

Note. Because of the symptom-duration variable’s ordinal nature, in Study 2 we confirmed the finding of a
significant effect of condition using a rank transformation of the original symptom-duration rating, F(2, 189) =
3.14, p < .05. BDI-II = Beck Depression Inventory-II.
same as in Study 1a, and the 6–8 points were 1 to 2 years, 2 to 5 years, and More than 5 years, but not indefinitely.

Results

We conducted linear regressions with biochemical/genetic attributions and BDI-II scores as independent predictors and symptom-duration ratings as the dependent variable. Biochemical/genetic attributions significantly predicted higher scores on the symptom-duration scale in both Study 1a ($\beta = .23$, $p = .02$) and Study 1b ($\beta = .42$, $p < .01$). (Because of the symptom-duration measure’s ordinal nature, we also confirmed these results using ordinal regressions, $p = .01$, in Study 1a, $p = .02$, in Study 1b.) BDI-II scores were a significant predictor in Study 1a only ($\beta = .28$, $p < .01$) (see Table 2 for $R^2$ values). To our knowledge, these results are the first empirical demonstration that the more people with depressive symptoms attribute those symptoms to genetic and biochemical causes, the longer they tend to expect their symptoms to last.

Study 2

Study 2 examined an approach to countering the prognostic pessimism associated with biochemical and genetic attributions for depression.

Method

All study procedures were approved by the Institutional Review Board. The procedures for participant recruitment; administering the BDI-II; notifying high scorers that they seemed to be feeling “sad, blue, or depressed”; and measuring their causal attributions for their symptoms were identical to those used in Study 1b. In Study 2, we also collected data from participants who scored under 16 on the BDI-II. (Table 2 shows BDI-II descriptive statistics for all participants.) BDI-II low scorers were told, “Based on your answers to the preceding questions, it seems that you are NOT feeling particularly sad, blue, or depressed.” For causal attributions, the low scorers rated the likelihood that each factor “might be causing the average depressed person’s sad, blue, or depressed feelings.”

After rating their causal attributions, all participants were randomly assigned to one of three conditions. In the “malleable” condition, participants watched an audiovisual psychoeducation intervention emphasizing the malleability of gene expression and brain chemistry associated with depression. The video provided a basic primer on epigenetics, explaining that genes can be “turned on or off” by environmental factors, and described how experience affects brain chemistry and activity. Previous research has found that providing environmental explanations for depression (e.g., stressful experiences) along with biological ones can reduce negative effects of biological explanations (Deacon & Baird, 2009). We avoided describing such environmental factors so that any effect of this video could not be attributed to mentioning environmental causes of depression. Instead, the malleability video discussed environmental factors only in terms of their ability to moderate the influence of biology on mood (e.g., “Aerobic exercise and exposure to sunlight have also been shown to change brain chemistry and activity in a way that helps with feelings of depression”). In the “biological illness” condition, participants watched a video focusing on the concept of depression as a biomedical condition. Similar to arguments promoted in scientific literature and popular media, the video explained that depression tends to run in families and that studies have documented differences between the brains of depressed and nondepressed individuals. Both videos were approximately 6 min long and narrated by the same person. Every effort was made to ensure that the two videos were similar in comprehensibility and in the amount of scientific information and the number of treatment options described, such that the explanatory emphasis was the only dimension on which they differed. The audio narrations for both videos are transcribed in the supplemental materials. In the control condition, participants received no intervention.

Participants who watched either video were 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

<table>
<thead>
<tr>
<th>Table 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Linear Regressions Modeling the Effects of Biochemical/Genetic Attributions</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>$n$</th>
<th>Model $R^2$</th>
<th>Biochemical/genetic attributions</th>
<th>BDI-II score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predicted duration of symptoms (Study 1a)</td>
<td>108</td>
<td>.14</td>
<td>$-227^*$, .015</td>
<td>.277, .003</td>
</tr>
<tr>
<td>Predicted duration of symptoms (Study 1b)</td>
<td>40</td>
<td>.23</td>
<td>$-421^*$, .006</td>
<td>.185, .210</td>
</tr>
<tr>
<td>Predicted duration of symptoms (Study 2)</td>
<td>193 (BDI-II ≥ 16)</td>
<td>.16</td>
<td>$-171^*$, .015</td>
<td>.311**, &lt;.001</td>
</tr>
<tr>
<td></td>
<td>328 (BDI-II &lt; 16)</td>
<td>.02</td>
<td>$-124^*$, .024</td>
<td>—</td>
</tr>
<tr>
<td>Predicted duration of symptoms, with treatment (Study 2)</td>
<td>191 (BDI-II ≥ 16)</td>
<td>.09</td>
<td>$-112^*$, .099</td>
<td>.243**, .001</td>
</tr>
<tr>
<td></td>
<td>324 (BDI-II &lt; 16)</td>
<td>.19</td>
<td>$-135^*$, .048</td>
<td>.378**, &lt;.001</td>
</tr>
<tr>
<td>Perceived odds of symptom desistance (Study 2)</td>
<td>193</td>
<td>.01</td>
<td>$-113^*$, .042</td>
<td>—</td>
</tr>
<tr>
<td>Perceived agency regarding depressive symptoms (Study 2)</td>
<td>192</td>
<td>.12</td>
<td>$-089^*$, .213</td>
<td>$-310^*$, &lt;.001</td>
</tr>
<tr>
<td>Guilt concerning depressive symptoms (Study 2)</td>
<td>179</td>
<td>.34</td>
<td>.07</td>
<td>.557, .001</td>
</tr>
<tr>
<td>BHS score (Study 2)</td>
<td>174</td>
<td>.34</td>
<td>.103</td>
<td>.548, &lt;.001</td>
</tr>
</tbody>
</table>

*Note.* Results are for individuals with BDI-II scores of at least 16, except where noted. Regression models used endorsement of biochemical/genetic attributions and/or BDI-II score as independent predictors. The number of participants varies slightly in each of the Study 2 regressions reported above because some participants did not respond to all measures. BDI-II = Beck Depression Inventory-II; BHS = Beck Hopelessness Scale. Dashes indicate that data were not obtained or are not reported.

$p < .05$. $^*$ $p < .01$. 

This document is copyrighted by the American Psychological Association or one of its allied publishers.
This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.
the “saying-is-believing” effect, a tendency for people to internalize viewpoints they have advocated (Aronson et al., 2002; Higgins, 1999; Walton & Cohen, 2011).

The subsequent procedure differed for high scorers and low scorers. High-scoring participants in all three conditions rated their expectations regarding the prognosis of their depression symptoms, perceptions of personal agency regarding their mood, feelings of guilt concerning their depression symptoms, and outlook for the future. Prognostic expectations were measured using the same symptom-duration scale from Study 1b, plus an identical scale asking how long they expected their symptoms would last if they received treatment, and a 0%–100% scale asking the odds that their depressed mood would “go away.” Agency perceptions were gauged using ratings of agreement with the statements “There are things I can do to eliminate my sad, blue, or depressed mood” and “I am able to improve my sad, blue, or depressed mood” (from 1 [Completely Disagree] to 7 [Completely Agree]). These two agency items were significantly correlated ($r = .68$, $p < .01$), so they were averaged to create an agency score for each participant. As a measure of guilt, the same agreement scale was used for the statement “I feel guilty about my sad, blue, or depressed mood.”

We gauged participants’ outlook for the future using the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974), but modified each of the 20 true/false judgments to a 6-point scale (very false, false, somewhat false, somewhat true, true, very true); scores thus ranged from 20 to 120, with higher scores indicating more hopelessness.

BDI-II low scorers responded to the same prognosis and agency items as the high scorers, but with respect to “the average depressed person” (e.g., “What do you think are the odds that the average depressed person’s sad, blue, or depressed mood will go away?”).

Finally, all participants were debriefed that depression “likely results from a combination of genetic, biochemical, environmental, and psychological factors” and received resources to find treatment for depression.

Some participants did not complete the study; most attrition appeared to result from difficulties viewing the videos using their own devices. The number of participants completing each dependent measure, by condition, is displayed in Figures 1–4. We computed demographics for participants who completed at least one dependent measure, regardless of BDI-II score. Those in the malleable condition ranged in age from 18 to 60 ($M = 26.44$) and were 43.5% female, 51.3% male, and 5.2% unknown gender. Those in the biological-illness condition ranged in age from 18 to 62 ($M = 30.26$) and were 47.7% female, 48.9% male, and 3.4% unknown gender. Those in the control condition ranged in age from 18 to 64 ($M = 29.04$) and were 46.6% female, 50.3% male, and 3.1% unknown gender.

Results

Associations between biochemical/genetic attributions and prognostic pessimism. We first examined relationships between BDI-II high scorers’ biochemical/genetic attributions and their ratings on our dependent measures. A moderated multiple regression approach (O’Connor, 1998), conducted to determine whether condition moderated the effects of premanipulation biochemical/genetic attributions, controlling for BDI-II score, revealed no significant attributions × condition interaction for any of our dependent variables. As such, we collapsed participants across conditions and conducted regression analyses for each of our dependent variables using the same predictor variables as in Studies 1a and 1b. Table 1 shows details of the results for all measures. In particular, among BDI-II high scorers, biochemical/genetic attribution scores were a significant predictor of longer expected symptom duration (replicating Studies 1a and 1b) ($\beta = .18$, $p = .01$) and lower perceived odds of recovery ($\beta = .15$, $p = .02$).

Regression models with low BDI-II scorers showed that their biochemical/genetic attributions were also a significant independent predictor of longer expected symptom duration ($\beta = .12$, $p = .012$) and lower perceived odds of recovery ($\beta = -.11$, $p = .04$). Ordinal regressions also showed biochemical/genetic attributions to significantly predict expected symptom duration ($p = .01$ among BDI-II high scorers; $p = .03$ among low scorers).

Effects of experimental manipulations. To examine the experimental manipulations’ effects, we first conducted 2 (symptomatology: high vs. low BDI-II score) × 3 (condition) analyses of variance (ANOVA) for symptom-duration ratings (with and without treatment), predicted odds of recovery, and agency scores. These revealed significant interactions ($p < .05$) for all variables. Thus, subsequent analyses considered high- and low-scoring participants separately.

To examine the effects of our manipulations on individuals with high BDI-II scores, we first conducted a series of ANOVAs with BDI-II scores as a covariate to control for symptom severity. These omnibus ANOVAs revealed significant effects of condition on expected symptom duration, $F(2, 189) = 3.10, p < .05$; perceived odds of recovery, $F(2, 187) = 5.31, p < .01$; agency perceptions, $F(2, 189) = 15.15, p < .01$; and BHS scores, $F(2, 175) = 5.67, p < .01$. Figures 1–4 display each condition’s mean for these variables. There were no significant effects of condition on the measures of guilt or expected symptom duration with treatment.

Then, on the four variables for which omnibus ANOVAs yielded significant effects of condition, we compared the responses of BDI high scorers in the malleable and control conditions using simple weighted contrasts. Overall, the malleability intervention yielded more optimistic views among BDI-II high scorers about their depressive symptoms. Specifically, those in the malleable condition expected shorter symptom durations ($p = .01$; see Figure 1, confirmed with rank-transformation analysis) and better odds of recovery ($p < .01$; see Figure 2), perceived more agency ($p < .01$; see Figure 3), and gave more optimistic BHS scores ($p < .01$; see Figure 4) than those in the control condition.

Next, we compared the biological-illness condition with the malleable condition among the high BDI-II scorers. Both conditions presented videos describing treatment options for depression; perhaps as a result, there were no significant differences between the two conditions in prognostic pessimism or hopelessness. Nonetheless, those in the malleable condition had significantly higher agency ratings than those in the biological-illness condition, $t(126) = 2.99, p < .01$.

We also used simple weighted contrasts to compare the biological-illness and control conditions among BDI-II high scorers. Participants in the biological-illness condition showed more optimism than those in the control condition only on two measures—lower BHS scores ($p < .05$) and higher agency ratings ($p = .02$). This suggests that the biological-illness video was less pow-
erful in changing the outlooks of symptomatic individuals from their baseline than the malleability intervention, which showed more differences from the control condition.

Next, we examined the effects of our manipulations on BDI-II low scorers. A series of ANOVAs revealed significant omnibus effects of condition on symptom-duration ratings (with and without rank transformation), perceived odds of recovery, and agency ratings (all $F$s > 12, all $p$s < .01). Replicating and extending previous findings with members of the general public (Deacon & Baird, 2009), BDI-II low scorers in the biological-illness condition were significantly more pessimistic about symptom duration (with and without rank transformation) and odds of recovery than those in the malleable and control conditions (all $p$s < .01, from weighted contrasts). Yet, those in the malleable and control conditions did not differ significantly on these measures, suggesting that the negative effects of biological explanations were absent when such information included psychoeducation about malleability. Furthermore, BDI-II low scorers in the malleable condition perceived depressed individuals to have more agency than did those in either of the other two conditions (all $p$s < .01 from weighted contrasts).

Finally, we compared BDI-II high- and low-scoring participants in each condition. At baseline (i.e., the control condition), prognostic pessimism was unsurprisingly stronger among BDI-II high scorers, as measured by symptom-duration ratings, $F(1, 191) = 13.02$, $p < .01$, and perceived odds of recovery, $F(1, 191) = 18.46$, $p < .01$. However, in the malleable condition, BDI-II high and low scorers did not differ significantly on either of these measures (see Figures 1 and 2). Agency scores showed an analogous pattern: In the control condition, BDI-II low scorers’ agency ratings were higher than those of high scorers, $F(1, 190) = 15.15$, $p < .01$, but there was no such difference in the malleable condition (see Figure 3). These patterns suggest that the malleability manipulation successfully elevated optimism among individuals with depressive symptoms to the level of nonsymptomatic individuals.

In the biological-illness condition, BDI-II high and low scorers did not differ significantly in their agency ratings. Interestingly, the biological-illness video actually resulted in more prognostic pessimism among BDI-II low scorers than among high scorers, as measured by expected symptom duration, $F(1, 172) = 5.38$, $p = .02$, and predicted odds of recovery, $F(1, 168) = 6.52$, $p = .01$.

**Discussion**

In the present studies, we examined how beliefs about the biology of depression might be related to prognostic pessimism among symptomatic individuals. Among people with elevated depressive symptomatology, endorsement of biochemical and ge-
Genetic causal attributions for depressive symptoms was significantly associated with more pessimistic symptom-duration predictions in all three studies and lower ratings of the likelihood of symptom desistance in Study 2. To our knowledge, this is the first time such effects have been documented in a sample of people who report significant depressive symptomatology. Given the increasing prevalence of biomedical conceptualizations of depression, the notion that depressed individuals who hold such beliefs might be more vulnerable to pessimism about the course of their disorder is alarming, particularly as positive outcome expectancies are an important determinant of actual prognosis (Rutherford et al., 2010).

Although this association was correlational, there are reasons to believe that it did not arise merely because people who are already pessimistic are more likely to endorse biochemical and genetic attributions. First, we controlled for BDI-II scores in all regression models, meaning that the effect of biochemical/genetic attributions in predicting pessimism was independent from severity of depressive symptomatology, given the increasing prevalence of biomedical conceptualizations of depression, the notion that depressed individuals who hold such beliefs might be more vulnerable to pessimism about the course of their disorder is alarming, particularly as positive outcome expectancies are an important determinant of actual prognosis (Rutherford et al., 2010).

In Study 2, we investigated whether such pessimism could be reduced by emphasizing the malleability of gene effects and brain chemistry in education about the biology of depression. Considering the ample evidence of the power of biological explanations in shaping views of psychopathology (Bennett et al., 2008; Darnimrod & Heine, 2011; Deacon & Baird, 2009; Haslam, 2011; Pescosolido et al., 2010; Phelan, 2005; Phelan et al., 2006), we sought to harness this power to promote optimism and feelings of agency by teaching that biology is not deterministic or fixed. Indeed, our malleability intervention successfully reduced symptomatic individuals’ prognostic pessimism and increased their feelings of agency concerning their moods, relative to those in the control condition. In fact, BDI-II high scorers who received this intervention were the only ones who rated the odds of recovery as greater than 50% on average. This intervention also yielded reduced feelings of general hopelessness.

Furthermore, the malleability intervention did not significantly increase feelings of guilt among BDI-II high scorers. Although caution is required in interpreting nonsignificant differences, this finding may suggest that the malleability intervention had specific, targeted effects rather than simply functioning as a positive mood induction. Also, the absence of increased guilt indicates that we found no evidence in our study of a potential negative side effect.
of emphasizing malleability—namely, that it could make symptomatic individuals feel accountable for their symptoms.

Comparisons of prognostic pessimism among high and low scorers also highlighted the benefits of the malleability intervention. Although all comparisons of BDI-II low and high scorers in the control condition predictably revealed that high scorers were more pessimistic, the same comparisons in the malleable condition showed no such differences. That is, after symptomatic individuals viewed the malleability intervention, their perceptions of their own agency and prognoses were as positive as nonsymptomatic individuals’ views of depression.

In contrast to those in the malleability condition, BDI-II high scorers in the biological-illness condition, who also received information about the biology of depression but without an emphasis on malleability, were no less pessimistic about their prognoses than those who received no intervention. This may suggest that this video’s content did not differ greatly from BDI-II high scorers’ preexisting beliefs about depression or that it was not powerful enough to change their preexisting beliefs. BDI-II high scorers in the biological-illness condition did report less hopelessness and more agency than those in the control condition, in a departure from previous research (Dar-Nimrod & Heine, 2011). This may have occurred because the biological-illness video referred to several effective treatments (Lebowitz & Ahn, 2012), which may have introduced the notion of malleability in some small way. However, they were significantly less confident in their own agency than participants in the malleable condition. The less generalizable and less powerful benefits of the biological-illness intervention for BDI-II high scorers suggest that the advantageous effects of the malleability intervention were not due merely to its inclusion of biological information, but rather to its specific emphasis on malleability.

Our manipulations did not affect high scorers’ expectations regarding symptom duration with treatment. One possible explanation is that mentioning treatment in the question strongly suggested the existence of effective treatment, which may have overpowered the differences in prognostic pessimism among the conditions. Additionally, research has shown that treatments are seen as more effective when they are congruent with causal accounts (e.g., medication is seen as more effective given biomedical explanations; Iselin & Addis, 2003). Thus, medication may have been seen as equally effective in all conditions, because none

Figure 3. Mean agency scores by condition. For BDI-II high scorers, agency scores represent the mean of participants’ agreement with the statements: “There are things I can do to eliminate my sad, blue, or depressed mood” and “I am able to improve my sad, blue, or depressed mood,” rated on a scale ranging from 1 (Completely Disagree) to 7 (Completely Agree). BDI-II low scorers responded to the same two agency items, but with respect to “the average depressed person” (e.g., “The average depressed person is able to improve their sad, blue, or depressed mood”). Higher bars indicate greater perceptions of personal agency. Bio. = Biological; BDI-II = Beck Depression Inventory-II. Error bars represent ± 1 SE.
questioned the importance of biological causes for depression. However, only the malleability intervention mentioned that psychotherapy can affect the brain, so prognostic expectations given psychotherapy (as opposed to treatment generally) could have differed among the conditions. Additionally, the absence of significant effects on our measure of symptom duration with treatment might be attributable to the fact that the measure did not specify a type of treatment, which could make this result somewhat difficult to interpret. Among participants who defined “treatment” as pharmacotherapy, the biological-illness video might have yielded more optimistic expectations of prognosis with treatment, with the opposite occurring among participants who defined “treatment” as psychotherapy. Such a pattern of responses could have “canceled out” any significant effects.

Interestingly, we did not find preexisting endorsement of biochemical/genetic causes for depression to moderate the effects of our manipulations. In previous research, people with strong genetic essentialist beliefs were affected more powerfully by the suggestion that biological differences underlie social categories (Keller, 2005), but this research used methodology quite different from ours, measuring nationalist prejudices. Future studies could clarify conditions in which preexisting biological essentialism affects sensitivity to biological explanations for psychopathology.

Study 2 also replicated previous findings that members of the general public, a majority of whom are not depressed, often associate biological explanations for psychopathology with prognostic pessimism (Haslam, 2011). Specifically, the biological-illness condition increased prognostic pessimism among low scorers so much that their responses on the symptom-duration scale and the rating of perceived odds of recovery were actually more pessimistic than those of BDI-II high scorers (who were unaffected by the biological-illness video, vis-à-vis the control condition). It remains unclear why our biological-illness video did not significantly increase prognostic pessimism among BDI-II high scorers, as it did among low scorers. One possibility is that some of its content (e.g., information about genetic heritability) may have seemed irrelevant to some high-scoring participants (e.g., those with no family history of depression). Another possibility is that

![Figure 4](image-url)
before any experimental manipulation, many BDI-II high scorers already held essentialist views of their depression symptoms as immutable consequences of largely unchangeable genetic and neurochemical factors. Indeed, this would be consistent with existing research suggesting that individuals with depression are more likely to view the disorder as resulting from stable biological causes (Prins, Verhaak, Bsing, & van der Meer, 2008). If BDI-II high scorers’ preexisting default beliefs already accorded with the content of the biological-illness video in such a manner, this could explain why the prognostic expectations of those in the biological-illness condition did not differ significantly from those of BDI-II high scorers in the control condition. By contrast, individuals without depression (i.e., a majority of the general public) may be more likely to view the disorder as resulting from psychosocial factors by default (Prins et al., 2008). If such causes are seen as more malleable, this could explain why BDI-II low scorers in the biological-illness condition—but not those in the malleability condition—differed in prognostic expectations from those in the control condition.

The benefits of our malleability intervention have clear clinical implications concerning psychoeducation. In particular, the intervention was delivered online in the form of a short video viewable on most Web browsers, making it highly scalable. Although Internet access, a computer with relatively up-to-date technical specifications, and some minimal familiarity with technology are necessary for consumers to access the video, it has the benefit of requiring no special expertise to administer. The development of psychological interventions that can be effectively administered remotely on a large scale—of which our malleability video is a prime example—has been identified as a pressing need in the field of mental healthcare (Kazdin & Blase, 2011).

The present studies also suggest important directions for further research. In particular, the long-term effects of our malleability intervention await further study. Because the intervention appears to promote a sense of agency and decrease feelings of pessimism among those with symptoms of depression, future studies could examine whether it might increase help-seeking behavior or enhance responsiveness to treatment.

Furthermore, future research could investigate how a history of treatment for depression—which we did not assess in our samples—might relate to our findings or moderate the effects of our manipulations. For example, symptomatic individuals’ previous experience with using antidepressant medications or with healthcare providers who promoted a particular causal theory regarding depression might influence their beliefs about the disorder’s causes or its malleability. Although our results speak to the ways in which individuals’ causal attributions for depression relate to their beliefs about its prognosis, our data do not allow us to examine how these beliefs formed, whether they were influenced by treatment history, or how such factors might relate to prognostic pessimism. Moreover, a history of successful treatment might bolster the notion that depression can be overcome, which could render the malleability intervention more plausible and efficacious. By contrast, a history of unsuccessful treatment (which might be more common among people with high BDI-II scores, because successfully treated individuals would likely not score as high) could make the malleability intervention seem less plausible. Nonetheless, our finding that the malleability intervention benefited people with current depression symptoms suggests that its effects are not limited to those who have been successfully treated for depression. More definite answers require future research.

Overall, our findings suggest reason for concern as well as reason for hopefulness. They establish that biological conceptualizations of depression—which are ever more popular both among scientists and the public—are associated with prognostic pessimism among symptomatic people. However, information about the biology of depression can be presented so that it actually reduces such pessimism.

The present results represent a call to arms for scientists studying the biology of mental disorders and those responsible for disseminating their findings. The association between biological attributions and prognostic pessimism may itself be malleable, and discussions of biology’s role in mental health need not suggest that psychopathology is permanent and immutable.

References


This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.


Received September 21, 2012
Revision received December 10, 2012
Accepted December 26, 2012