



Doubting the Diagnosis but Seeking a Talking Cure: An Experimental Investigation of Causal Explanations for Depression and Willingness to Accept Treatment

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Abstract

In the current literature there is a general lack of research examining the impact of causal explanations on beliefs about psychotherapy, willingness to accept treatment, and treatment expectancies. The present study was aimed at experimentally investigating effects of causal explanations for depression on treatment-seeking behavior and beliefs. Participants at a large Southern university ($N = 139$; 78% female; average age 19.77) received bogus screening results indicating high depression risk, then viewed an explanation of depression etiology (fixed biological vs. malleable biopsychosocial) before receiving a treatment referral (antidepressant vs. psychotherapy). Participants accepted the cover story at face value, but some expressed doubts about the screening task's ability to properly assess their individual depression. Within the skeptics, those given a fixed biological explanation for depression were relatively unwilling to accept either treatment, but those given a malleable biopsychosocial explanation were much *more* willing to accept psychotherapy. Importantly, differences in skepticism were not due to levels of actual depressive symptoms. Information about the malleability of depression may have a protective effect for persons who otherwise would not accept treatment.

Keywords Depression · Psychotherapy · Stigma · Etiology · Chemical imbalance · Prognosis · Attributions · Treatment-seeking

A growing number of studies indicate that biological causal explanations for depression are associated with negative beliefs about key aspects of depression and its treatment (Dar-Nimrod and Heine 2011; Lebowitz 2014). Depressed individuals who attribute depression to biological factors such as a medical illness, heredity, or chemical imbalances in the brain perceive their symptoms as less controllable and expect to be depressed for longer than depressed individuals who endorse other kinds of causal explanations (Brown et al. 2007; Lebowitz et al. 2013, Study 1). Moreover, relationships between biological causal explanations for depression and pessimistic beliefs about its controllability and course

are supported by experimental evidence (Deacon and Baird 2009; Kemp et al. 2014; Lebowitz et al. 2013, Study 2).

Biological Causal Explanations, Essentialism, and Treatment

One framework for understanding these effects relates to the concept of essentialism—the idea that persons belong to discrete kinds or categories based on innate, unchanging characteristics (Dar-Nimrod and Heine 2011; Haslam and Kvaale 2015; Kendler et al. 2011). Factors such as genes and neurobiology are often viewed as being outside of a person's control, fixed, and central to identity (Dar-Nimrod and Heine 2011; Haslam and Kvaale 2015). Thus biological explanations for depression—especially those that portray risk factors as fixed—contribute to a belief that one's own efforts to improve one's depressed mood will not make a difference (Deacon and Baird 2009; Lebowitz 2014; Lebowitz et al. 2013). This view can hamper motivation to engage in effective emotion regulation strategies or make positive

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lifestyle changes (Kneeland et al. 2016), and it may contribute to a belief that those positive changes will ultimately make things worse by inviting disappointment and failure (Winer et al. 2017; Winer and Salem 2016). Further, persons who believe that they are biologically fated to struggle with depression might have low expectations for treatment—especially non-drug treatments such as psychotherapy (Deacon and Baird 2009)—which, in turn, can interfere with help-seeking, therapeutic engagement, and psychotherapy outcomes (Kichuk et al. 2015).

The link between treatment expectancies and psychotherapeutic processes and outcomes is already well established (Delsignore and Schnyder 2007; Greenberg et al. 2006). Meta-analytic findings support the link between treatment expectancies and later post-treatment outcomes in clinical populations (Constantino et al. 2011).

However, much less is known about whether or not causal explanations for depression impact treatment expectancies for psychotherapy, though there is evidence suggesting that they might. For example, among psychotherapy clients—a population that necessarily holds a sufficiently positive view of psychotherapy to have sought it out as a form of treatment—biological causal explanations for depression are associated with lower credibility ratings for cognitive therapy, but higher credibility ratings for behavioral activation (Tompkins et al. 2017). Further, research indicates that hopelessness is associated with lower treatment expectancies (Goldfarb 2002). Although Lebowitz et al. did not find a significant link between biological explanations and scores on a general hopelessness measure (2013), biological explanations have been linked to hopelessness regarding perceived likelihood of recovery from depression (Deacon and Baird 2009; Kemp et al. 2014; Kvaale et al. 2013a, b; Lebowitz et al. 2013), which is arguably more relevant than general hopelessness in shaping treatment expectancies.

There is also evidence that treatment expectancies are lower when causal beliefs do not match treatment modality—for example, persons who endorse biological explanations rate non-pharmacological treatments such as psychotherapy as less likely to be helpful for them than do persons with different causal attributions for depression (Iselin and Addis 2003). Among clinicians and in the general public, causal attributions for depression have been linked to beliefs about treatment and preferences for specific treatment modalities (Ahn et al. 2009; Goldstein and Rosselli 2003; Lebowitz and Ahn 2014; Lebowitz and Applebaum 2017). Consistent with this idea, when causal explanations for depression were experimentally manipulated in participants instructed to imagine that they were depressed, biological explanations led to significantly lower perceived helpfulness ratings for psychotherapy, but higher perceived helpfulness for antidepressants (Deacon and Baird 2009). After participants read a biopsychosocial explanation these

effects were reversed, however, with psychotherapy rated as more helpful and antidepressants as less helpful (Deacon and Baird 2009). These findings represent initial experimental evidence suggesting that causal explanations for depression might shape treatment expectancies. However, use of a repeated-measures design in this study may have produced demand characteristics, because participants likely framed the task as a comparison of two competing models as a result of reading both a biological and biopsychosocial explanation for depression.

Two formative studies that experimentally manipulated causal beliefs in depressed participants did include measures assessing perceptions of treatment (Kemp et al. 2014; Lebowitz et al. 2013, Study 2), but in both cases elements of study design make it difficult to interpret the relevant results. Lebowitz et al. found no significant differences between individuals who viewed fixed biological, malleable, or no causal explanations on a question about how long they expected their symptoms to last if they received treatment (2013, Study 2). However, because participants were not asked about psychotherapy or antidepressant medications separately, it is possible that participants who viewed different causal explanations had differential perceptions about the helpfulness of these two treatment options, without there being a difference in perceived helpfulness of treatment overall.

Similarly, Kemp et al. (2014) found no significant difference in perceived credibility or helpfulness of psychosocial treatment when comparing participants in a serotonin deficiency condition to those in the control condition, but the serotonin deficiency group rated pharmacological treatment as significantly more credible and more helpful than the control group. These findings are also somewhat limited because the control group was not given an alternative explanation for their symptoms; therefore, receiving “normal” test results might have given the impression that treatment was not needed, or might have been experienced as invalidating, and thus may have decreased their belief that either type of treatment would be helpful (Kemp et al. 2014). Alternatively, depressed individuals might have stronger views about causes of depression—perhaps due to greater previous exposure to information about the disorder, or due to personal experiences—and thus a one-time manipulation might not be sufficient to change their beliefs.

Rationale for the Present Study

It is possible that causal explanations that emphasize fixed, biologically determined risk factors might interfere with psychotherapy, perhaps by limiting expectations for improvement in therapy and/or undermining the perceived credibility of non-medical treatments. It is also possible

that information about the malleability of biological and genetic risk factors may help to offset or even reverse the negative effects of biological explanations on perceptions of depressed persons (Lebowitz et al. 2013, Study 2). At present, these are empirical questions. There is a general lack of experimental research examining the impact of causal explanations on beliefs about psychotherapy. The few experimental findings that have been published on the topic are difficult to interpret due to aspects of study design. Further, the experimental studies published to date have measured outcomes via self-report; to our knowledge none have examined the effects of causal explanations for depression on behaviors that might be relevant to treatment. The present study was aimed at addressing these gaps.

Present Study

The primary purpose of the present study was to examine whether or not causal explanations for depression and type of treatment offered have an interactive effect on willingness to accept treatment. Participants were given a cover story stating that they were at high risk of developing depression. Then, based on random assignment, they were presented with either a fixed biological or a malleable biopsychosocial explanation for depression before being referred for treatment in the form of either psychotherapy or antidepressant medication.

Specifically, the present study was aimed at testing the following hypotheses:

- (1) Causal explanation (fixed biological vs. malleable biopsychosocial) and treatment referral type (antidepressant vs. psychotherapy) will have an interactive effect on willingness to accept treatment.
 - a. Within the antidepressant referral condition, participants who receive the fixed biological explanation for depression will report greater willingness to accept treatment than those who receive the malleable explanation.
 - b. Within the psychotherapy referral condition, participants who receive the fixed biological explanation for depression will report lower willingness to accept treatment than those who receive the malleable explanation.
- (2) Causal explanation (fixed biological vs. malleable biopsychosocial) and treatment type (antidepressant vs. psychotherapy) will have an interactive effect on treatment expectancies.

- a. Individuals in the malleable condition will report higher treatment expectancies (i.e., perceived helpfulness, perceived credibility) for psychotherapy and lower treatment expectancies for antidepressant medication.
- b. Individuals in the fixed biological condition will report lower treatment expectancies for psychotherapy and higher treatment expectancies for antidepressant medication.

Methods

Measures

Beliefs About Causes of Depression

As a means of evaluating whether the cover story and experimental manipulation had the intended effect of shaping participants' causal explanations for depression, participants were asked to rate on a scale from 1 to 7 (1 = very unlikely, 7 = very likely) the likelihood that each of several factors "might cause sad, blue, or depressed feelings". The items rated were the same as those measured by Lebowitz et al. (2013): "Genetics," "Brain chemistry or other biochemical imbalance," "Day-to-day problems and/or stress," "Beliefs or style of thinking," "Abnormal brain structure/development," "Brain injury," "Substance abuse," "Weakness of character," "Problems from childhood or the way you were raised," and "Recent traumatic events."

Depressive Symptoms

The Quick Inventory of Depressive Symptomatology—Self-Report (QIDS-SR; Rush et al. 2003) was used to assess current depressive symptoms. The QIDS-SR is a widely-used and validated self-report questionnaire that asks participants to rate the extent to which (0 = not at all, 3 = severely) they have experienced each of 16 symptoms over the previous week. Some items are aggregated for scoring, such that the total score covers 9 symptom domains: Insomnia/Hypersomnia, Sadness, Weight/Appetite Changes, Guilt/Worthlessness, Difficulty Concentrating, Thoughts of Death/Suicide, Loss of Interest, Fatigue, and Psychomotor Retardation/Agitation. Item 12 of the QIDS-SR was administered separately at the end of the study and used to assess for current suicidality, according to IRB-approved procedures. Thus, item 12 of the QIDS-SR was not included in computing the total score as this item was administered separately from the rest of the scale's items during the experiment. Researchers opted to assess suicidal ideation and/or intent in person at the end in order to provide referral sources after conclusion of the experiment. These items (without item 12) were

scored according to the standard procedure and included as a covariate in some analyses, as described in the Results section of this manuscript.

Treatment Expectancies for Psychotherapy

Expected helpfulness and credibility of psychotherapy was assessed using a brief written description of the treatment followed by the Credibility/Expectancy Questionnaire (CEQ; Devilly and Borkovec 2000), a validated 6-item self-report measure assessing 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 to reduce depressive symptoms?”). Because participants may not have personally felt depressed at the time of the study, items were modified slightly by dropping the words “you” and “your,” to allow participants to respond based on their perceptions of each treatment for depression in general. The CEQ has been shown to possess adequate test–retest reliability and internal consistency (Devilly and Borkovec 2000), and it has been used in previous research examining the effects of causal explanations for depression (Kemp et al. 2014). For the present study, the CEQ was scored using the procedures described by Nock and colleagues (Kemp et al. 2014; Nock et al. 2007). Items 1, 2, 3, and 5 were scored on a 9-point Likert-type scale (1 = Not a lot of sense/No improvement, 9 = A lot of sense/Very much improvement). Participants responded to Items 4 and 6 using an 11-point scale (i.e., 10% intervals ranging from 0 to 100%), but these two items were converted to a 9-point scale (responses from 40 to 60% are collapsed into a single value and scored a 5) at scoring, to match the other items in the measure. The total scale scored in this way has been shown to possess adequate internal consistency and construct validity (Nock et al. 2007). In previous research the credibility and expectancy components of the CEQ have been found to be strongly correlated (Nock et al. 2007), and have both been affected similarly by an experimental manipulation that induced a biological causal explanation for depression (Kemp et al. 2014). Indeed, in the present study the credibility and expectancy components of the CEQ (as described by Devilly and Borkovec 2000) were found to be correlated ($r = .62$ for the CEQ for psychotherapy, $r = .66$ for the CEQ for antidepressant medication). Thus, in the present study analyses were carried out on the CEQ total scale.

Treatment Expectancies for Antidepressant Medication

Similarly, expected helpfulness and credibility of antidepressant medication was assessed using a brief written description of the treatment, followed by the CEQ (Devilly and Borkovec 2000). All item wording and scoring procedures were the same as those described above in the Treatment

Expectancies for Psychotherapy section. Treatment expectancies for psychotherapy and antidepressant medication were assessed in counterbalanced order, and all participants completed the CEQ for both types of treatment.

Willingness to Accept Treatment

The computer program used to assess willingness to accept treatment was created and administered using E-Prime 2.0 Professional (Psychology Software Tools, Pittsburg, PA). The program comprised a series of questions, interspersed with short videos depicting a professionally-dressed actor, sitting at a desk in a laboratory. The actor selected to appear in these videos was blind to the hypotheses of the study, and was the same actor who presented the bogus “depression screening test” feedback and the causal explanations for depression. The stimuli for this screening test were taken from previous research using the IAT to study implicit depressive attitudes, and consisted of the following words: I, Me, Self, Myself, Mine, They, Them, Their, Theirs, Other, Depressed, Helpless, Hopeless, Gloomy, Withdrawn, Smiling, Glad, Cheerful, Joyful, Delighted (Meites et al. 2008). Data from this task were not logged as part of the present study. The task simply served as part of the cover story—participants were told after completing the task that it had been a “depression screening test”—so that they could be given bogus test results showing that they were at high risk for depression. Henceforth, we will refer to this task as the “depression screening test.”

In the first video viewed by all participants, the actor explained that early intervention is a key factor in successful treatment of depression. The actor stated that in light of the viewer’s score on the “depression screening test”, it is strongly recommended that he or she look into treatment right away. The actor then presented two referral options; the order in which they were presented depended on the treatment referral condition to which that participant had been assigned. Participants in the two antidepressant-first referral groups initially viewed a video presenting the opportunity to schedule an appointment with the student health center to discuss antidepressant medication with a health care provider, whereas participants in the two psychotherapy-first referral groups initially viewed a video presenting the opportunity to schedule an appointment with the student counseling center to discuss psychotherapy with a counselor. In both videos, the actor made clear that the participant could make the appointment right away via computer by watching the following set of videos and using the keyboard to respond to onscreen prompts.

After watching the initial video, participants were asked via computer if they were willing to schedule an appointment right away via computer. Participants who declined to do so were then shown additional videos asking questions

Table 1 Responses to treatment referrals and corresponding scores on measure of willingness to accept treatment

Response	Score
Scheduled an appointment, selected a date 0–7 days away	11
Scheduled an appointment, selected a date 8–14 days away	10
Scheduled an appointment, selected a date 15–21 days away	9
Scheduled an appointment, selected a date ≥ 22 days away	8
Declined appointment right away, but “definitely” intends to make one within a week	7
Declined appointment right away, but “maybe” will make one within a week, $> 50\%$ likelihood	6
Declined appointment right away, but “maybe” will make one within a week, $\leq 50\%$ likelihood	5
Declined appointment right away, will “probably not” make one within a week, but would “definitely” make appointment if started to feel worse	4
Declined appointment right away, will “probably not” make one within a week, “maybe” would make appointment if started to feel worse, $> 50\%$ likelihood	3
Declined appointment right away, will “probably not” make one within a week, “maybe” would make appointment if started to feel worse, $\leq 50\%$ likelihood	2
Declined appointment right away, will “probably not” make one within a week, and would “probably not” make appointment if started to feel worse	1

about their willingness to schedule an appointment on their own sometime within the following week, and their willingness to schedule an appointment in the future if they were to experience an increase in symptoms. Following each video, participants gave a response using the keyboard. For each treatment referral condition, willingness to accept treatment was operationally defined on the basis of (a) whether or not participants chose to schedule an appointment while in the laboratory, and (b) if so, what date they chose, or (c) if not, then whether they would be willing to schedule an appointment in the future. In this manner, treatment willingness was scored using the 11-point scale laid out in Table 1.

Participants

Sample size for the present study was determined using a priori power analyses carried out in G*Power (Faul et al. 2007). For Hypothesis 1, a power analysis was carried out for a 2×2 factorial ANOVA with the effect size set at $f = .25$ and alpha level of .05 (two-tailed). Results indicated that a total sample of $N = 128$ would be required to achieve estimated power of 80% for the interaction effect, which was the outcome of primary interest with regard to Hypothesis 1. For Hypothesis 2, a power analysis was carried out for a mixed-design ANOVA with the effect size set at $f = .25$, and alpha level of .05, and correlation between repeated measures conservatively set to zero. Results indicated that a total sample of $N = 66$ would be required to achieve estimated power of 80%.

Participants were recruited via SONA from the undergraduate subject pool at a large Southern university, and were awarded 1 h of course credit in exchange for participating. Participants who volunteered through SONA to take

part in the study were scheduled to come to the lab for the experimental session.

Procedure

Upon arriving, participants were presented with an informed consent form stating that the goal of the study is to examine how individual differences relate to responses on computerized tasks. The form also stated that the study would involve completing tasks on a computer and responding to questions, and that all efforts would be made to ensure the confidentiality of responses.

A researcher seated the participant in front of a computer and stated that the computer would guide the participant through each part of the session. The researcher gave the participant a set of headphones and instructed the participant to wear them throughout the session, as some parts of the session would include audio. The researcher then opened the computer program in E-Prime 2.0 Professional, which began presenting a computerized task modeled after the implicit association test (IAT; Greenwald et al. 1998). The stimuli for this task were taken from previous research using the IAT (Meites et al. 2008). Once the task instructions appeared onscreen, the researcher left the room. After the task was finished, the computer automatically administered the QIDS-SR (without item 12). Participants responded using the keyboard.

Experimental Manipulation of Causal Explanations

Participants were randomly assigned to one of four cells: the fixed biological explanation/antidepressant referral cell, the fixed biological explanation/psychotherapy referral cell, the malleable explanation/antidepressant referral cell, or the

malleable explanation/psychotherapy referral cell. After completing the QIDS-SR, the computer automatically presented the bogus “depression screening test” results and either the fixed biological explanation video or the malleable explanation video. The script of each video was presented by an actor wearing professional attire and seated at a desk in a laboratory. The same actor appeared in both causal explanation videos, as well as in both sets of treatment referral videos, and was blind to the study hypotheses. The causal explanation videos followed the same scripts used by Lebowitz et al. (2013), with additional material added to the beginning presenting the bogus “depression screening test” results. Both videos acknowledged the existence of biological risk factors for depression, but the malleable explanation emphasized the multicausal nature of depression and the importance of environmental and behavioral factors in moderating the influence of biological risk factors. For example, in the fixed biological explanation, participants were told:

Genetics are a large part of what puts a person at risk for becoming depressed. For example, immediate family members of a depressed person have a significantly higher risk of becoming depressed than would an average person, and having two parents with a history of depression more than doubles a person’s risk of becoming depressed. This is because genetics play such a large part in determining whether someone becomes depressed or not.

Genes are like manufacturing instructions for building our brains and bodies. As you may know, genes determine how we look, and what kinds of traits we have. Likewise, certain genes create chemical imbalances in the brain, which are known as major causes of depression. These genes have even been called depression genes. Furthermore, genes determine whether or not people become depressed in response to stressful events.

By contrast, the malleable explanation stated:

There are many reasons why genes are not the deciding factor in depression. For example, even if a person has depression-related genes, these genes may not be active. Like a light switch, genes can be turned on or off. Research has shown that lifestyle factors like diet, exercise, and levels of stress will affect whether or not genes related to depression will actually be turned on.

Some people have heard that depression is caused by a chemical imbalance in the brain. In reality though, biological tests of brain activity or brain chemicals cannot even be used to diagnose depression. What’s more, scientists have found that the brain is constantly changing because of the experiences and environments

we choose. The brain can be compared to a muscle: it grows and changes according to how it is used or exercised.

The full scripts of both causal explanation videos are available from the corresponding author upon request.

Post-manipulation Questionnaires

After the causal explanation video ended, the computer automatically presented the manipulation check and treatment expectancy measures and instructed participants to respond using the keyboard. Other self-report measures unrelated to the present hypotheses were also administered via computer at this time.

Treatment Referrals

After the self-report measures were completed, the computer automatically presented the treatment referral video and questions assessing willingness to accept treatment in the form of either psychotherapy or antidepressant medication, depending upon the referral condition to which participants had been randomly assigned.

Debriefing

After participants responded to questions assessing willingness to accept treatment, the demographic questionnaire was automatically administered via computer, followed by the funnel debriefing questionnaire. The funnel debriefing questionnaire initially asked participants to provide their overall thoughts about the present study, with further questions asking for greater detail about their thoughts (e.g., if they had doubts about any part of the study). Participants were then debriefed via video and given the opportunity to decline to have their data used for research purposes. The full script of the debriefing video is available from the corresponding author upon request. Participants were also given referral information and offered assistance in making an appointment with either the student health center or the counseling center if they wished to do so before they left the laboratory.

Results

Participant Characteristics

Informed consent was obtained from all individual participants included in the study. Of the 145 participants who completed the study, six were excluded from analyses for the following reasons: declining to sign data use permission form (2), sound initially muted on videos (1), listening

to music during the experimental session (1), using phone to video chat during the experimental session (1), previously took a class taught by PI and stated that this factor influenced desire to participate (1). Thus, a total of 139 participants were included in analyses. Demographic characteristics of the sample were as follows: 77.7% female; mean age of 19.77, $SD = 1.64$; 63.3% White non-Hispanic, 32.4% Black non-Hispanic, 1.4% Hispanic or Latino, 0.7% Asian or Pacific Islander, 2.2% Other.

Missing Data and Internal Consistency

Only 0.38% of the data was missing from measures of beliefs about the causes of depression, depressive symptoms, and treatment expectancies. Little's test yielded non-significant results, indicating that missing values in the present data set are missing completely at random. Given the low percentage of missing data and the outcome of Little's test, missing values from self-report measures were filled in using mean substitution (Tabachnick and Fidell 2013).

In addition, five values were missing from the treatment willingness data, and all occurred on items where participants were asked to enter a percentage indicating the likelihood that they would seek treatment either within a week or in the future if depressive symptoms increased. Thus, in these five cases, scores were computed by taking the midpoint between the two values that might have been assigned if participants had responded to all of the items validly. More specifically, these values were entered in by utilizing the participant's data from other questions concerning treatment willingness. For instance, if a participant noted they did not wish to make an appointment right away but would "maybe" make one on their own within a week, they would score either a 5 or 6 on the relevant treatment willingness measure. In this case, if the percent likelihood item was missing, then a score of 5.5 would be entered.

To assess internal consistency, Cronbach's alpha was calculated for the QIDS-SR (without item 12), the CEQ for antidepressant medication, and the CEQ for psychotherapy. All three measures evidenced adequate internal consistency; $\alpha = .79$, $\alpha = .89$, and $\alpha = .85$, respectively.

Beliefs About the Causes of Depression and Credulity

After viewing either the fixed biological or malleable explanation video, participants were asked to rate the likelihood that various factors might cause depressed mood. In order to assess the efficacy of the causal explanation manipulation in altering participants' beliefs about the causes of depression, independent-samples t tests were carried out using these ratings as the DVs. For items where Levene's test indicated inequality of variance (i.e., "brain chemistry

or biochemical imbalance," "abnormal brain structure or development," "substance abuse") the degrees of freedom were adjusted accordingly. Results indicated significant group differences in ratings for "genetics," $t(137) = 7.88$, $p < .001$, Cohen's $d = 1.35$, "brain chemistry or biochemical imbalance," $t(135.82) = 5.94$, $p < .001$, Cohen's $d = 1.02$, "abnormal brain structure or development," $t(134.74) = 6.12$, $p < .001$, Cohen's $d = 1.05$, and "brain injury," $t(137) = 3.42$, $p = .001$, Cohen's $d = 0.58$.¹ As expected, participants who viewed a fixed biological explanation for depression rated each of these factors as more likely to cause depressed mood than did participants who viewed a malleable explanation.

Prior to watching the debriefing video, participants completed a funneled debriefing questionnaire that included the yes/no question: "Did you have doubts about any part of the study?" Over half of participants (56.8% of full sample; 58.2% in the fixed biological explanation condition, 55.6% in the malleable explanation condition) reported having no doubts about any part of the study. A Chi square test examined whether the percentage of participants who expressed doubts differed across the four combinations of causal explanation and referral type. Results were non-significant, $X^2(3, N = 139) = 0.47$, $p = 0.93$, indicating no differences in doubts across conditions.

Participants who answered "yes" to the binary doubts item were then presented with a free-response item asking them to describe their doubts. Almost all responses focused on the accuracy or validity of the "depression screening test" results (e.g., "I feel my reaction time results overestimated my depression risk," "I doubt that I have depression," "I am still a little bit confused about how reaction time can relate to someone's depression"). No participants indicated any suspicion that the "depression screening test" had been part of a cover story or that the study had involved deception at any point, nor did anyone correctly identify the true purpose of the study. Thus, participants likely accepted the causal explanations for depression, even if they had doubts about the "depression screening test" as it pertained to them.

In order to quantitatively assess this possibility, independent samples t tests using causal explanation condition as the IVs and responses to the manipulation check items as the DVs were repeated, this time only including those participants ($n = 60$) who reported doubts about the study (Fig. 1).

¹ Within the fixed biological explanation group ($n = 67$), the item "recent traumatic events" exhibited the most extreme skewness (-1.76) and kurtosis (3.56) values. Within the malleable explanation group ($n = 72$), "day to day problems or stress" exhibited the greatest skew (-2.06) and "substance abuse" exhibited the most extreme kurtosis (4.86). Although somewhat elevated, these skewness and kurtosis values are still within acceptable limits (Kline 2010). Mann-Whitney tests were also conducted comparing causal explanation groups and yielded similar results.

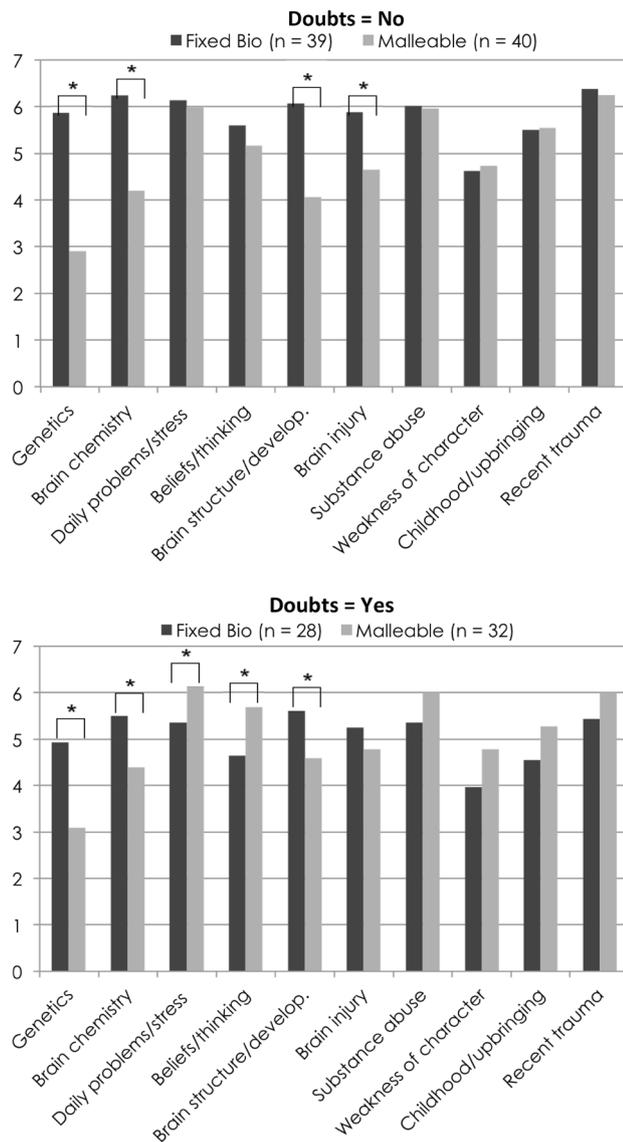


Fig. 1 Mean beliefs about the causes of depression in participants who reported no doubts about the study (top) and those who did report doubts (bottom). * $p < .05$

Results still indicated significant differences between explanation conditions, with participants in the fixed biological condition giving higher ratings for “genetics,” $t(58) = 3.69$, $p < .01$, Cohen’s $d = 0.97$, “brain chemistry or biochemical imbalance,” $t(58) = 2.35$, $p = .02$, Cohen’s $d = 0.62$, and “abnormal brain structure or development,” $t(58) = 2.31$, $p = .03$, Cohen’s $d = 0.61$, though not for “brain injury,” $t(58) = 1.01$, $p = .32$, Cohen’s $d = 0.27$. In addition, two other factors were now significant with participants in the malleable condition giving higher ratings: “day to day problems or stress,” $t(58) = 2.18$, $p = .03$, Cohen’s $d = 0.57$, and “beliefs or style of thinking,” $t(58) = 2.42$, $p = .02$, Cohen’s $d = 0.64$.

Taken together, these results indicate that participants who expressed doubts about the study were still swayed by the causal explanations for depression. Nonetheless, in order to assess whether credulity toward the study influenced the effect of the experimental manipulation, hypothesis tests were carried out both with and without the binary doubts variable included as a factor.

Hypothesis 1

Hypothesis 1, that the effect of causal explanation on willingness to accept treatment would differ based on type of treatment referral, was tested using a 2×2 factorial ANOVA. Causal explanation (fixed or malleable) and type of treatment referral (antidepressant medication or psychotherapy) were included as independent variables (IVs) and willingness to accept treatment served as the dependent variable (DV).

Means and standard deviations for each cell were as follows: fixed explanation, medication referral ($n = 34$, $M = 3.97$, $SD = 2.67$); fixed explanation, psychotherapy referral ($n = 33$, $M = 4.47$, $SD = 2.64$); malleable explanation, medication referral ($n = 31$, $M = 3.63$, $SD = 2.69$); malleable explanation, psychotherapy referral ($n = 41$, $M = 5.02$, $SD = 2.82$). There was a significant main effect of type of treatment referral on willingness to accept treatment, $F(1, 135) = 4.20$, $p = .04$, partial $\eta^2 = .03$. Examination of marginal means indicates that participants referred to the counseling center for psychotherapy were more willing to accept the referral than were those referred to the student health center for antidepressant medication. The interaction of causal explanation and treatment referral was non-significant, $F(1, 135) = .94$, $p = .33$, partial $\eta^2 = .01$, however. Thus, Hypothesis 1 was not supported, as no main effects were predicted and a significant interaction was predicted.

Credulity and Willingness to Accept Treatment

In order to examine whether participants’ credulity toward the study moderated the impact of the experimental manipulation on willingness to accept treatment, a $2 \times 2 \times 2$ ANOVA was conducted. Causal explanation (fixed or malleable), type of treatment referral (antidepressant medication or psychotherapy), and doubts (yes or no) were included as IVs, and willingness to accept treatment served as the DV.

Consistent with the results from the original test of Hypothesis 1, there was a significant main effect of treatment referral type on willingness to accept treatment, $F(1, 131) = 5.01$, $p = .03$, partial $\eta^2 = .04$, with participants referred for psychotherapy showing greater willingness to accept the referral than those referred for antidepressant medication. There was also a significant main effect of the binary doubts variable, $F(1, 131) = 4.39$, $p = .04$, partial

$\eta^2 = .03$, with participants who reported no doubts about the study exhibiting greater willingness to accept treatment. Notably, the 3-way interaction of explanation condition, referral type, and doubts was also significant, $F(1, 131) = 6.00$, $p = .02$, partial $\eta^2 = .04$.

To probe the significant 3-way interaction, separate 2×2 ANOVAS were conducted within each level of the binary doubts variable, with explanation condition and referral type as IVs and treatment willingness as the DV. Among participants who reported no doubts about the study ($n = 79$), no significant main effects or interactions emerged. However, among participants who reported having doubts about the study ($n = 60$), the main effect of referral type approached significance, $F(1, 56) = 3.93$, $p = .05$, partial $\eta^2 = .07$, and there was a significant 2-way interaction of explanation condition and referral type, $F(1, 56) = 6.47$, $p = .01$, partial $\eta^2 = .10$.

Examination of marginal means indicated that among participants who responded “yes” to the doubts question, those who viewed the malleable explanation for depression were more willing to accept a referral for psychotherapy than were those who viewed the fixed biological explanation, whereas mean scores for treatment willingness in those referred for antidepressant medication were similar across causal explanation conditions (Fig. 2). To test the simple effect of causal explanation within participants who received psychotherapy referrals and reported having doubts about the study, an independent samples t test was conducted with causal explanation as the IV and willingness to accept treatment as the DV. Results showed a large effect of causal explanation condition, $t(32) = 2.92$, $p = .01$, Cohen’s $d = 1.04$, with those who heard the malleable explanation for depression evidencing much greater willingness to accept psychotherapy than those who heard the fixed biological explanation.

In order to rule out the possibility that the observed interaction of causal explanation, referral type, and doubts was an artifact of differences in depressive symptoms across cells, we repeated the $2 \times 2 \times 2$ ANOVA with causal explanation (fixed or malleable), type of treatment referral (antidepressant medication or psychotherapy), and doubts (yes or no) as IVs, but this time with current depression symptoms serving as the DV. In this case, the 3-way interaction was not significant, $F(1, 131) = 1.03$, $p = .31$, partial $\eta^2 = .01$. This outcome serves as evidence against the possibility that the 3-way interaction predicting treatment willingness was merely an artifact of differences in depressive symptoms.

However, in the analyses with treatment willingness as the DV we saw the significant explanation condition by referral type interaction specifically within the group of participants who reported having doubts about the study. As a means of assessing evidence for an artifactual

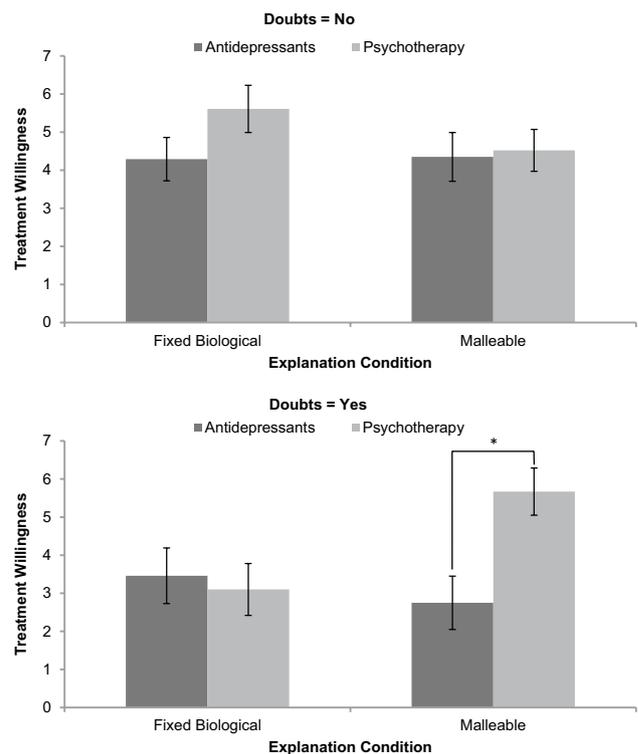


Fig. 2 Mean levels of willingness to accept treatment within each combination of causal explanation for depression and treatment referral type, for participants for reported no doubts about the study (top) and those who did report doubts (bottom). * $p < .05$

explanation at this level, separate 2×2 ANOVAS were conducted within each level of the binary doubts variable, with explanation condition and referral type as IVs, but now with depression scores serving as the DV. The 2-way interaction neared significance among participants who reported having doubts, $n = 60$, $F(1, 56) = 3.97$, $p = .05$, partial $\eta^2 = .07$. However, the effect size for the 2-way interaction of explanation condition and referral type with treatment willingness as the DV was 58% larger than the effect size with depression scores as the DV.

Finally, we conducted ANCOVAs that repeated the original $2 \times 2 \times 2$ ANOVA and subsequent pair of 2×2 ANOVAs within each level of the doubts variable (i.e., with explanation condition, referral type, and doubts as IVs and willingness to accept treatment as the DV), but this time depression scores were included as a covariate. After adjusting for current depressive symptoms, the $2 \times 2 \times 2$ ANCOVA still yielded a significant 3-way interaction of explanation condition, referral type, and doubts, $F(1, 130) = 5.11$, $p = .03$, partial $\eta^2 = .04$. In addition, the 2×2 ANCOVA within those participants who reported having doubts ($n = 60$) still yielded a significant 2-way interaction of explanation condition and referral type after adjusting for current depressive symptoms, $F(1, 55) = 4.57$, $p = .04$,

Table 2 Variable means in fixed biological and malleable explanation conditions for participants with and without doubts

Referral type	Doubts = No				Doubts = Yes			
	Fixed biological (n = 39)		Malleable (n = 40)		Fixed biological (n = 28)		Malleable (n = 32)	
	M	SD	M	SD	M	SD	M	SD
CEQ antidepressant	38.31	9.74	34.48	8.15	36.46	10.03	32.72	8.73
CEQ psychotherapy	38.56	9.20	37.03	8.47	35.61	7.53	35.84	8.26
Depressive symptoms	8.38	5.08	8.00	3.94	5.71	4.04	6.94	4.09

CEQ antidepressant = credibility and expectancy questionnaire, antidepressant medication, CEQ psychotherapy = credibility and expectancy questionnaire, psychotherapy, Doubts = response to question “Did you have doubts about any part of the study?”

partial $\eta^2 = .08$. Thus, depressive symptoms alone cannot explain the constellation of findings that emerged due to participants’ doubts about their diagnosis.

Hypothesis 2

Hypothesis 2, that participants given a fixed biological explanation for depression would report lower perceived credibility and effectiveness for psychotherapy and higher perceived credibility and effectiveness for antidepressant medication than those given a malleable explanation, was tested using a mixed-design ANOVA. Causal explanation (fixed or malleable) served as the between-subjects variable and type of treatment (psychotherapy and antidepressant medication as measured by the CEQ for psychotherapy and CEQ for antidepressant medication, respectively) as the within-subjects variable.

The main effect of causal explanation approached but did not reach significance, $F(1, 137) = 3.70, p = .06$, partial $\eta^2 = .03$, and the interaction of causal explanation and treatment type was non-significant, $F(1, 137) = 3.00, p = .09$, partial $\eta^2 = .02$, and thus the results should be interpreted with caution. Examination of marginal means revealed that among participants given a fixed biological explanation for depression ($n = 67$) CEQ ratings for psychotherapy ($M = 37.33, SD = 8.61$) and for antidepressant medication ($M = 37.54, SD = 9.83$) were similar, whereas among participants given a malleable explanation ($n = 72$) CEQ ratings for psychotherapy ($M = 36.50, SD = 8.34$) were higher than those for antidepressant medication ($M = 33.69, SD = 8.40$). Moreover, CEQ ratings for antidepressant medication among participants given a malleable explanation were lower than CEQ ratings for either treatment type among participants given a fixed biological explanation.

To better understand these results, paired-samples t tests were carried out examining the simple effects of treatment type within each level of causal explanation. Results indicated that within the malleable explanation group, participants rated psychotherapy as significantly more credible and more effective for treating depression than antidepressant

medication, $t(71) = 2.38, p = .02$, Cohen’s $d = .34$. Within the fixed biological explanation group, no significant differences between treatment types were observed. Thus, Hypothesis 2 was partially supported, but again, caution is urged in interpreting the simple effects given that the interaction was non-significant.

Credulity and Perceived Credibility and Effectiveness of Treatments

In order to examine whether participants’ credulity toward the study moderated the impact of the experimental manipulation on the perceived credibility and effectiveness of treatments, a mixed ANOVA was carried out with explanation condition (fixed biological or malleable) and doubts about the study (yes or no) as between-subjects IVs, CEQ treatment modality (antidepressant medication or psychotherapy) as the within subjects IV, and CEQ scores as the DV.

Results were largely consistent with those obtained in the original test of Hypothesis 2, before the addition of the binary doubts variable. Again, the main effect of causal explanation approached significance, $F(1, 135) = 3.29, p = .07$, partial $\eta^2 = .02$, whereas the main effect of doubts was not significant despite yielding a similar effect size, $F(1, 135) = 2.50, p = .12$, partial $\eta^2 = .02$. Again, examination of marginal means (see Table 2 for means and SDs) showed that among participants given a fixed biological explanation for depression, CEQ ratings for psychotherapy and for antidepressant medication were similar, whereas among participants given a malleable explanation CEQ ratings for psychotherapy were higher than those for antidepressant medication. However, the 2-way interaction of causal explanation and CEQ treatment type did not reach significance, $F(1, 135) = 3.14, p = .08$, partial $\eta^2 = .02$. The 3-way interaction of explanation condition, CEQ treatment type, and the binary doubts variable was also non-significant, $F(1, 135) = 0.23, p = .63$, partial $\eta^2 < .01$.

Discussion

The present study yielded some evidence in support of the hypothesis that causal explanations for depression and the type of treatment offered interactively impact willingness to accept treatment, such that individuals given a malleable explanation for depression are more likely to accept referrals for psychotherapy than those given a fixed biological explanation. However, this effect was moderated by participants' level of credulity with regard to the "depression screening test" presented as part of the cover story. Further, the direction of this moderation effect was unexpected, in that the interaction of causal explanation condition and treatment referral type paradoxically had a greater impact on treatment willingness among participants who reported having doubts about the study. This outcome was especially surprising given that on the whole participants who had no doubts about the study showed greater willingness to accept treatment than those who reported having doubts.

Differences in current depressive symptoms across the different combinations of causal explanation and treatment referral type did not account for the observed interaction effect on willingness to accept treatment, given that the interaction effect remained significant after adjusting for current depressive symptoms. It appears that the experimental manipulation of causal explanations for depression in the present study did have a large effect on willingness to accept psychotherapy, but only in participants who were skeptical of the "depression screening test."

One possible explanation for the moderating role of doubts observed in the present study is that the experimental manipulation might have required active cognitive scrutiny and elaboration on the causal explanations for depression in order for the interactive effect on treatment willingness to emerge, and reporting doubts about the study may have been a by-product and marker of that active scrutiny (Petty et al. 2009; Petty and Cacioppo 1986). Participants who actively scrutinized the cover story and bogus feedback and critically compared it to their prior knowledge of how depression symptoms or risk are measured would arguably be more likely to form doubts about our "depression screening test." Likewise, a participant who was actively scrutinizing the content of these messages and making inferences would likely notice a contradiction—the root of the problem in depression is genetic and biological, yet psychotherapy does not alter the underlying biology—and might therefore be unwilling to engage in psychotherapy as a treatment for depression, even if they were to actually feel depressed in the future. Alternatively, participants who evaluated the cover story and bogus feedback by applying heuristics related to pre-existing trust in doctors, mental health experts, or the

university as an institution, might have been more likely to passively accept the messages without forming any doubts and might not have perceived any contradiction between the fixed biological explanation for depression and psychotherapy as a form of treatment (Petty et al. 2009; Petty and Cacioppo 1986).

A range of factors can impact decisions about whether to engage in effortful or passive processing, including situational factors such as perceived trustworthiness and expertise of the source, personal interest in the subject matter, extent to which the message is consistent with prior beliefs, and more stable individual differences such as need for cognition—a preference for effortful cognitive engagement across a range of situations (Cacioppo and Petty 1982; Heesacker et al. 1983; Petty et al. 2009). Future research can directly examine these factors in relation to causal explanations for depression and treatment willingness.

Finally, it is notable that among participants who did report having doubts about the study, those who viewed the malleable explanation for depression and received a psychotherapy referral exhibited much greater willingness to accept treatment than *any* of the other three cells, including the cell of participants who viewed the fixed biological explanation and received a medication referral. In other words, the combination of a malleable explanation for depression and the option of psychotherapy as a treatment had a protective effect, allowing this group of participants to be just as accepting of treatment as those who did not have doubts about the study. This, too, seems to suggest that the interactive effect of causal explanation and referral type on treatment willingness may be linked to differences in scrutinizing the credibility of depression assessments.

The present study also yielded a main effect of referral type, with participants who received a referral for psychotherapy exhibiting greater willingness to accept treatment than those who received a referral for antidepressant medication. This is consistent with the robust literature indicating that psychological treatments are preferred over pharmacological treatments for mental health problems (McHugh et al. 2013).

With regard to Hypothesis 2, that individuals in the malleable condition would report higher treatment expectancies for psychotherapy and lower treatment expectancies for antidepressant medication, whereas individuals in the fixed biological condition would report lower treatment expectancies for psychotherapy and higher treatment expectancies for antidepressant medication, results of the present study were inconclusive. Although a trend in the predicted direction was observed in the malleable explanation condition, the predicted interaction did not reach significance. Inclusion of credulity in the analysis also yielded non-significant results. The present study was powered to detect medium-sized effects, and it is possible that the predicted interaction

reflects a small effect that would reach significance with a larger sample. It is also possible that the experimental manipulation of causal explanations for depression had a stronger impact on willingness to accept treatment than on treatment expectancies in the present study because participants responded to treatment referrals while under the impression that they were making decisions about their own lives and the likelihood that they might *personally* benefit from treatment, whereas they may have answered treatment credibility and expectancy items on the basis of beliefs about the potential benefits of treatments for others, or for people with depression more generally. These potential explanations can be tested in future research.

Implications of the Present Study for Treatment of Depression with Psychotherapy

This constellation of findings, which will benefit from replication, extension, and translation, provides further evidence of the benefit of disseminating information about the malleability of biological risk factors for depression. One implication of the present study is that not everyone will see a contradiction between a fixed biological causal explanation for depression and the use of psychotherapy as a treatment, possibly because some individuals will accept both messages on the grounds that they come from a trusted source (for instance, both messages might come from a clinician practicing interpersonal psychotherapy). However, for others—perhaps those inclined to scrutinize and make inferences about causal explanations—a contradiction will emerge, and to the extent that they have adopted a fixed biological explanation for depression they may be less willing to accept psychotherapy as a treatment.

To our knowledge, no studies to date have examined relationships between need for cognition and treatment preferences in depression. However, the literature does indicate that individuals high in need for cognition are less stressed by cognitively demanding tasks (Cacioppo et al. 1996) and prefer taking an active role in problem solving. For instance, there is a strong association between need for cognition and endorsement of statements such as, “I prefer to figure things out for myself” (Amabile et al. 1994). In the present study, among participants who reported doubts there was a significant difference between the causal explanation conditions in perceived likelihood that depression could be caused by “beliefs or style of thinking,” whereas no such effect was found among those who had no doubts about the study. Perhaps active thinkers found the malleable explanation more compelling or consistent with their experiences than those who engaged in more passive processing, and therefore were more swayed by it. It is possible that persons

who highly value effortful thought and tend to scrutinize the world are not inclined to accept passive treatments for depression such as antidepressant medication, regardless of their beliefs about the causes of depression. Yet, the present study suggests they may be open to a treatment that affords more autonomy such as psychotherapy, but only *if* it is logically consistent with their understanding of the causes of depression. At present this idea is largely conjecture, but these findings lay out a clear target for future research.

Strengths and Limitations

The key finding from the present study—that causal explanation, referral type, and doubts had a significant interactive effect on participants’ willingness to accept treatment—arose from exploratory analyses, and thus carries certain limitations. Follow-up studies will be crucial to determine whether or not the pattern of results observed here replicates, and to provide more insight into the present findings. For example, because the role of doubts as a moderator of our experimental results was unanticipated, the present study did not directly assess how participants went about evaluating the information presented.

It is possible that the provision of inaccurate feedback about depression risk prompted a particular type of processing (e.g., greater scrutiny). Nonetheless, the observed interaction cannot be explained as an experimental artifact, because all of the participants heard the same cover story and bogus feedback. Most participants were not actually depressed and therefore had reason to believe the feedback was inaccurate, yet only some of them had doubts about the study. This suggests that individual differences—such as, perhaps, preference for more effortful processing—played a role in determining whether or not participants formed doubts about the study, and that these individual differences may be a novel moderator of behavior in response to different types of messages about depression. However, at present it remains an empirical question whether effortful versus passive processing acts as a moderator of the relationship between causal explanations for depression and treatment willingness, and additional research will be needed to test this possibility.

Recruiting from a student population allowed us to assess individuals with a wide range of prior knowledge and attitudes toward psychological disorders and treatment, whereas clinical samples might disproportionately include individuals who already have favorable attitudes toward treatment. Depressed individuals might also have more fixed beliefs about causes of depression—perhaps due to greater previous exposure to information about the disorder, or due to personal experiences—and therefore a one-time manipulation might not be sufficient to change their beliefs. Finally,

recruitment of an undergraduate sample allowed us to test our hypotheses with emerging adults, and given that the median age of onset for mood disorders in the United States ranges from 25 to 32 years old (Kessler et al. 2005), beliefs about depression may be especially important in emerging adulthood.

One drawback to recruitment of a student sample, rather than a sample pre-screened for elevated depressive symptoms, is that the findings that emerged may not generalize to a clinical population. In addition, the fact that the majority of our participants did not have elevated depressive symptoms may have made it more difficult to convince participants of the accuracy and validity of the “depression screening test” (although no participants identified that the present study used deception or guessed the true purposes of the study). These were limitations of the present study, but in our view these limitations were outweighed by the benefits of a sample drawn from emerging adults who were not pre-screened for depression.

The present study was not aimed at determining how much each causal explanation might differ from no explanation (i.e., a control condition), but rather at examining the effects of the fixed biological and malleable causal explanations in relation to one another, and the extent to which those effects were moderated by type of treatment offered. The decision not to compare causal explanations to a “no explanation” condition was based in part on an interest in ecological validity. Outside of a laboratory setting it would be very unusual for an individual to be diagnosed with depression or told they are at risk for depression without being given any information regarding the causes or nature of the disorder. In fact, certain types of assessments may, in and of themselves, communicate certain causal explanations; for example, assessment of family history, tests of genetic factors, or brain imaging may imply a biological basis for depression even if no such explanation is provided explicitly. Thus, the malleability condition might provide a more ecologically valid alternative to a fixed biological explanation than a condition in which participants are given no information about depression or the implications of their test results.

Nonetheless, inclusion of a control group could further understanding of the absolute impact of specific causal explanations, whereas the present study only provides information about the comparative impact. This is a limitation of the present study. However, the findings that emerged can help inform the design of follow-up studies that do include control groups, and therefore allow for more detailed parsing of the effects of each type of causal explanation.

Finally, the willingness to accept treatment scale was a strength of the present study in that it provided a behaviorally-anchored outcome measure; participants responded to a scheduling prompt while under the impression that they were making a real, immediate decision about whether or not

to seek treatment. However, the measure also confers limitations in that it is novel and unvalidated, and the intervals between scale values may not be equal. There is a substantial literature demonstrating the robustness of ANOVA and *t* tests even when used with ordinal data, so long as the data includes sufficient levels (Glass et al. 1972; Norman 2010), and therefore we believe the benefits of using this measure of willingness to accept treatment ultimately outweighed the drawbacks.

Future Directions

Responses from the funnel debriefing questionnaire provided insight into potential ways of strengthening the cover story in future research. Although participants did take the cover story at face value and accepted that the computerized task they completed was intended as a “depression screening test,” many participants questioned whether the test could accurately measure one’s depression risk. In future studies the cover story could likely be modified by increasing the number of task blocks that participants complete so that the test seems more thorough, or by incorporating other emotional processing tasks and presenting the depression screener as a battery of measures rather than a single test. The cover story may also be modified by expanding the explanation of how reaction time tasks can measure individual differences in the processing of emotional information, and of how these differences relate to depression risk.

The literature on processing styles indicates that a wide range of factors such as individual differences in need for cognition, personal interest in the subject matter, extent to which the message is consistent with the listener’s self-concept, and perceived trustworthiness of the information source can jointly determine elaboration likelihood in a given situation (Heesacker et al. 1983; Petty et al. 2009). Thus, research will be needed to assess which factors are most relevant in determining how individuals evaluate messages about the causes of depression.

In addition, assessing other variables that may influence attitudes toward treatment would be important in future research. For example, the QIDS-SR measures only current depressive symptoms (within the past week); thus, one cannot rule out the influence of lifetime history of depression and its course, such as whether chronicity affects willingness to accept treatment. Furthermore, assessing family history of psychopathology may also prove important, as knowledge of one’s own family history can foster attitudes about the effectiveness of certain treatments.

Based on our results, it seems that some of the key effects of causal explanations for depression could only be observed within specific combinations of causal explanation and treatment referral type. Although we ensured proper power for

the current study, follow-up studies designed with this point in mind could include larger sample sizes to increase statistical power for detecting and comparing differential influences on treatment decisions, especially the sub-group analyses (i.e., doubts versus no doubts), given the effect sizes from the analyses. In order to better parse the impact of each causal explanation, future research can include control groups and incorporate pre-manipulation measurements of beliefs, for comparison. Further studies will also be needed to examine whether the effects observed here extend to populations with elevated symptoms of depression.

Finally, it is certainly possible to conceive of causal explanations attributing depression to factors that are not biological or genetic, but are nonetheless fixed—for example, models that present early childhood adversities as leaving some permanent psychological scar. Future research is needed to determine whether the differential effects of fixed biological and malleable explanations for depression on treatment willingness would also emerge when comparing other forms of fixed and malleable causal explanations.

Conclusion

The present study was the first to our knowledge to experimentally examine whether causal explanations for depression impact willingness to accept a referral for psychotherapy. Our findings indicate that information about the malleability of risk factors for depression may have a protective effect that specifically benefits skeptical individuals who otherwise would not accept treatment, allowing them to be open to psychotherapy.

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Data Availability The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Compliance with Ethical Standards

Conflict of interest Taban Salem, E. Samuel Winer, D. Gage Jordan, and Morgan M. Dorr declare that they have no conflict of interest.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Research Involving Human and Animal Rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research

committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References

- Ahn, W. K., Proctor, C. C., & Flanagan, E. H. (2009). Mental health clinicians' beliefs about the biological, psychological, and environmental bases of mental disorders. *Cognitive Science*, *33*, 147–182.
- Amabile, T. M., Hill, K. G., Hennessey, B. A., & Tighe, E. M. (1994). The Work Preference Inventory: Assessing intrinsic and extrinsic motivational orientations. *Journal of Personality and Social Psychology*, *66*, 950–967. <https://doi.org/10.1037/0022-3514.66.5.950>.
- Brown, C., Battista, D. R., Sereika, S. M., Bruehlman, R. D., Dunbar-Jacob, J., & Thase, M. E. (2007). Primary care patients' personal illness models for depression: Relationship to coping behavior and functional disability. *General Hospital Psychiatry*, *29*, 492–500. <https://doi.org/10.1016/j.genhosppsych.2007.07.007>.
- Cacioppo, J. T., & Petty, R. E. (1982). The need for cognition. *Journal of Personality and Social Psychology*, *42*, 116–131. <https://doi.org/10.1037/0022-3514.42.1.116>.
- Cacioppo, J. T., Petty, R. E., Feinstein, J. A., & Jarvis, W. G. (1996). Dispositional differences in cognitive motivation: The life and times of individuals varying in need for cognition. *Psychological Bulletin*, *119*, 197–253. <https://doi.org/10.1037/0033-2909.119.2.197>.
- Constantino, M. J., Arnkoff, D. B., Glass, C. R., Ametrano, R. M., & Smith, J. Z. (2011). Expectations. *Journal of Clinical Psychology*, *67*, 184–192.
- Dar-Nimrod, I., & Heine, S. J. (2011). Genetic essentialism: On the deceptive determinism of DNA. *Psychological Bulletin*, *137*, 800–818. <https://doi.org/10.1037/a0021860>.
- Deacon, B. J., & Baird, G. L. (2009). The chemical imbalance explanation of depression: Reducing blame at what cost? *Journal of Social and Clinical Psychology*, *28*, 415–435. <https://doi.org/10.1521/jscp.2009.28.4.415>.
- Delsignore, A., & Schnyder, U. (2007). Control expectancies as predictors of psychotherapy outcome: A systematic review. *British Journal of Clinical Psychology*, *46*, 467–483. <https://doi.org/10.1348/014466507X226953>.
- Devilly, G. J., & Borkovec, T. D. (2000). Psychometric properties of the credibility/expectancy questionnaire. *Journal of Behavior Therapy and Experimental Psychiatry*, *31*, 73–86. [https://doi.org/10.1016/S0005-7916\(00\)00012-4](https://doi.org/10.1016/S0005-7916(00)00012-4).
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). GPower 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175–191. <https://doi.org/10.3758/BF03193146>.
- Glass, G. V., Peckham, P. D., & Sanders, J. R. (1972). Consequences of failure to meet assumptions underlying the fixed effects analyses of variance and covariance. *Review of Educational Research*, *42*, 237–288.
- Goldfarb, D. E. (2002). College counseling center clients' expectations about counseling: How they relate to depression, hopelessness, and actual-ideal self-discrepancies. *Journal of College Counseling*, *5*, 142–152. <https://doi.org/10.1002/j.2161-1882.2002.tb00216.x>.
- Goldstein, B., & Rosselli, F. (2003). Etiological paradigms of depression: The relationship between perceived causes, empowerment, treatment preferences, and stigma. *Journal of Mental Health*, *12*, 551–563.

- Greenberg, R. P., Constantino, M. J., & Bruce, N. (2006). Are patient expectations still relevant for psychotherapy process and outcome? *Clinical Psychology Review*, 26, 657–678. <https://doi.org/10.1016/j.cpr.2005.03.002>.
- Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology*, 74, 1464–1480. <https://doi.org/10.1037/0022-3514.74.6.1464>.
- Haslam, N., & Kvaale, E. P. (2015). Biogenetic explanations of mental disorder: The mixed-blessings model. *Current Directions in Psychological Science*, 24, 399–404. <https://doi.org/10.1177/0963721415588082>.
- Heesacker, M., Petty, R. E., & Cacioppo, J. T. (1983). Field dependence and attitude change: Source credibility can alter persuasion by affecting message-relevant thinking. *Journal of Personality*, 51(4), 653–666.
- Iselin, M. G., & Addis, M. E. (2003). Effects of etiology on perceived helpfulness of treatments for depression. *Cognitive Therapy and Research*, 27, 205–222. <https://doi.org/10.1023/A:1023513310243>.
- Kemp, J. J., Lickel, J. J., & Deacon, B. J. (2014). Effects of a chemical imbalance causal explanation on individuals' perceptions of their depressive symptoms. *Behaviour Research and Therapy*, 56, 47–52. <https://doi.org/10.1016/j.brat.2014.02.009>.
- Kendler, K. S., Zachar, P., & Craver, C. (2011). What kinds of things are psychiatric disorders? *Psychological Medicine*, 41, 1143–1150. <https://doi.org/10.1017/S0033291710001844>.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62, 593–602. <https://doi.org/10.1001/archpsyc.62.6.593>.
- Kichuk, S. A., Lebowitz, M. S., & Adams, T. G., Jr. (2015). Can biomedical models of psychopathology interfere with cognitive-behavioral treatment processes? *The Behavior Therapist*, 38, 181–186.
- Kline, R. B. (2010). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: Guilford Press.
- Kneeland, E. T., Dovidio, J. F., Joormann, J., & Clark, M. S. (2016). Emotion malleability beliefs, emotion regulation, and psychopathology: Integrating affective and clinical science. *Clinical Psychology Review*, 45, 81–88. <https://doi.org/10.1016/j.cpr.2016.03.008>.
- Kvaale, E. P., Gottdiener, W. H., & Haslam, N. (2013a). Biogenetic explanations and stigma: A meta-analytic review of associations among laypeople. *Social Science and Medicine*, 96, 95–103. <https://doi.org/10.1016/j.socscimed.2013.07.017>.
- Kvaale, E. P., Haslam, N., & Gottdiener, W. H. (2013b). The 'side effects' of medicalization: A meta-analytic review of how biogenetic explanations affect stigma. *Clinical Psychology Review*, 33, 782–794. <https://doi.org/10.1016/j.cpr.2013.06.002>.
- Lebowitz, M. S. (2014). Biological conceptualizations of mental disorders among affected individuals: A review of correlates and consequences. *Clinical Psychology*, 21, 67–83. <https://doi.org/10.1111/cpsp.12056>.
- Lebowitz, M. S., & Ahn, W. (2014). Effects of biological explanations for mental disorders on clinicians' empathy. *Proceedings of National Academy of Sciences*, 111, 7786–7790. <https://doi.org/10.1073/pnas.1414058111>.
- Lebowitz, M. S., Ahn, W. K., & Nolen-Hoeksema, S. (2013). Fixable or fate? Perceptions of the biology of depression. *Journal of Consulting and Clinical Psychology*, 81, 518–527. <https://doi.org/10.1037/a0031730>.
- Lebowitz, M. S., & Appelbaum, P. S. (2017). Beneficial and detrimental effects of genetic explanations for addiction. *International Journal of Social Psychiatry*, 63, 717–723.
- McHugh, R. K., Whitton, S. W., Peckham, A. D., Welge, J. A., & Otto, M. W. (2013). Patient preference for psychological vs pharmacological treatment of psychiatric disorders: A meta-analytic review. *The Journal of Clinical Psychiatry*, 74, 595.
- Meites, T. M., Deveney, C. M., Steele, K. T., Holmes, A. J., & Pizzagalli, D. A. (2008). Implicit depression and hopelessness in remitted depressed individuals. *Behaviour Research and Therapy*, 46, 1078–1084. <https://doi.org/10.1016/j.brat.2008.05.008>.
- Nock, M. K., Ferriter, C., & Holmberg, E. (2007). Parent beliefs about treatment credibility and effectiveness: Assessment and relation to subsequent treatment participation. *Journal of Child and Family Studies*, 16, 27–38. <https://doi.org/10.1007/s10826-006-9064-7>.
- Norman, G. (2010). Likert scales, levels of measurement and the "laws" of statistics. *Advances in Health Sciences Education*, 15, 625–632. <https://doi.org/10.1007/s10459-010-9222-y>.
- Petty, R. E., Brinol, P., & Priester, J. (2009). Mass media attitude change: Implications of the elaboration likelihood model of persuasion. In J. Bryant & M. B. Oliver (Eds.), *Media effects: Advances in theory and research* (3rd ed., pp. 125–164). New York, NY: Routledge.
- Petty, R. E., & Cacioppo, J. T. (1986). The elaboration likelihood model of persuasion. *Advances in Experimental Social Psychology*, 19, 123–205. [https://doi.org/10.1016/S0065-2601\(08\)60214-2](https://doi.org/10.1016/S0065-2601(08)60214-2).
- Psychology Software Tools, Inc. [E-Prime 3.0]. (2017). Retrieved from <http://www.pstnet.com>.
- Rush, A. J., Trivedi, M. H., Ibrahim, H. M., Carmody, T. J., Arnow, B., Klein, D. N., et al. (2003). The 16-item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): A psychometric evaluation in patients with chronic major depression. *Biological Psychiatry*, 54, 573–583. [https://doi.org/10.1016/S0006-3223\(02\)01866-8](https://doi.org/10.1016/S0006-3223(02)01866-8).
- Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (6th ed.). Boston, MA: Pearson.
- Tompkins, K. A., Swift, J. K., Rousmaniere, T. G., & Whipple, J. L. (2017). The relationship between clients' depression etiological beliefs and psychotherapy orientation preferences, expectations, and credibility beliefs. *Psychotherapy*, 54, 201. <https://doi.org/10.1037/pst0000070>.
- Winer, E. S., Bryant, J., Bartoszek, G., Rojas, E., Nadorff, M. R., & Kilgore, J. (2017). Mapping the relationship between anxiety, anhedonia, and depression. *Journal of Affective Disorders*, 221, 289–296. <https://doi.org/10.1016/j.jad.2017.06.006>.
- Winer, E. S., & Salem, T. (2016). Reward devaluation: Dot-probe meta-analytic evidence of avoidance of positive information in depressed persons. *Psychological Bulletin*, 142, 18–78. <https://doi.org/10.1037/bul0000022>.

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