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An Experimental Investigation of Causal Explanations for Depression and Willingness to Accept Treatment

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An experimental investigation of causal explanations for depression and willingness to accept treatment

By

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An experimental investigation of causal explanations for depression and willingness to accept treatment

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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) 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 explanation were much *more* willing to accept psychotherapy. Importantly, differences in skepticism were not due to levels of actual depressive symptoms. The present findings indicate that information about the malleability of depression may have a protective effect for persons who otherwise would not accept treatment.

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CHAPTER I

INTRODUCTION

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 & Heine, 2011; Lebowitz, 2014). In light of these findings, the present study was designed to examine effects of causal explanations for depression on willingness to accept psychotherapy and pharmacotherapy, respectively.

Causal Explanations for Depression

Depressed individuals who view their symptoms as being caused by biological factors such as a medical illness, genes/heredity, or a chemical imbalance in the brain perceive their depression 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, Ahn, & Nolen-Hoeksema, 2013, Study 1).

Moreover, relationships between biological causal explanations for depression and negative beliefs are supported by experimental evidence (Kemp, Lickel, & Deacon, 2014; Lebowitz et al., 2013, Study 2). For example, depressed participants who received bogus test results indicating that they had a serotonin deficit rated themselves as less capable of regulating their negative moods, expected their symptoms to last longer, and predicted lower odds of eventual recovery, when compared to depressed participants who got bogus test results showing normal neurotransmitter levels (Kemp et al., 2014).

Similarly, Lebowitz and colleagues manipulated causal explanations for depression in individuals with high and low levels of depressive symptoms by showing them informational videos (2013, Study 2). One group watched a "biological illness" video that described depression as a brain disorder largely caused by genes and stated "depressed people have abnormalities in critical areas of the brain." A second group watched a "malleable" video, which stated that "lifestyle factors like diet, exercise, and levels of stress" affect whether or not depression-related genes get "turned on," and that learning new ways of thinking and interacting with others changes patterns of brain activity in depressed persons. Both videos described various treatment options, but the biological illness video emphasized antidepressant medication, whereas the malleable video emphasized psychotherapy. A third group of participants served as controls and did not watch a video (Lebowitz et al., 2013, Study 2).

Among individuals with higher levels of depressive symptoms, those assigned to the malleable condition perceived themselves as more capable of improving their negative moods than did participants in either the biological illness or control conditions, and they reported lower levels of hopelessness, expected their own symptoms to be shorter-lived, and believed they had higher odds of recovery than did depressed participants in the control group (Lebowitz et al., 2013, Study 2). Interestingly, depressed participants in the biological illness group also reported less hopelessness and more ability to change their negative moods than did controls, though they did not differ from the control group on perceived duration of symptoms or odds of recovery. These findings were attributed to the fact that both videos portrayed depression as a treatable problem and presented treatment options. Among mildly depressed and nondepressed individuals,

those assigned to the malleable condition perceived “the average depressed person” as more able to improve their negative moods than did participants in either the biological illness or control conditions. In contrast, individuals in the biological illness condition expected the average depressed person’s symptoms to last longer and be less likely to remit than did participants in either of the other two groups, which did not differ. Finally, across depressed and nondepressed participants, there were no significant effects of condition on guilt associated with depression or perceived odds of recovery with treatment (the latter will be discussed further in the Biological Explanations, Coping Strategies, and Treatment section of this document).

In an experimental study in which non-depressed participants were asked to imagine that they had been diagnosed with depression, participants reported less sense of personal responsibility for symptoms after reading a biological causal explanation for depression than they did after reading a biopsychosocial explanation (Deacon & Baird, 2009). However, after reading the biological explanation participants also reported lower perceived ability to control their symptoms, believed their depression would be more chronic, and said they would be less likely to eventually recover. Similarly, among the general public (i.e., persons not pre-selected for elevated symptoms or history of psychological disorders), experimental induction of biological causal explanations for mental disorders leads to increases in perceived severity of psychological problems as well as perceived dangerousness of individuals with such problems (Kvaale, Haslam, & Gottdiener, 2013).

Biological Causal Explanations and Essentialism

Findings from qualitative research illustrate a belief that the biological characteristics of persons with psychological disorders make them categorically different from persons without such disorders, and that their status as disordered will determine many aspects of their future. For example, in a synthesis of qualitative studies, Malpass and colleagues (2009) identified a pattern whereby depressed individuals described the experience of being prescribed an antidepressant—which necessarily communicates a biological explanation for depression—as something that reduced self-blame but also categorically changed their perceptions of themselves by confirming that they were not "normal" (Malpass et al., 2009). Another qualitative study included this quote from the mother of an adolescent girl with psychological difficulties (Hess, Gantt, Lacasse, & Vierling-Claassen, 2014, p. 196):

She's schizoaffective bipolar; the prognosis is that there is no cure—that she needs to learn to live with it the best she can. I'm expecting her to regress. ... Her brain is wired in a way that her mental illness will be a monkey on her back the rest of her life. It is unlikely she will ever be able to hold down a real job.

Hess and colleagues also quoted an adult woman saying the following about her own depressive symptoms (2014, p. 193):

It's a chemical imbalance—it's not gonna go away with ... you know; I can't—I'm not one of those that could take medication for a couple of years and then be good. ... I'm stuck.

One framework for understanding these comments relates to the concept of essentialism—the idea that persons belong to discrete kinds or categories based on innate, unchanging aspects of their nature or identities (Dar-Nimrod & Heine, 2011; Haslam & Kvaale, 2015; Kendler, Zachar, & Craver, 2011). Factors such as genes or neurobiology are often viewed as being outside of a person's control, fixed, and central to identity (Dar-Nimrod & Heine, 2011; Haslam & Kvaale, 2015). Because personal responsibility is predicated on a behavior or situation being controllable (Weiner, 1993), it is not surprising that attributing mental disorders to biological factors tends to reduce blame. However, as Haslam and Kvaale (2015) argue, biological explanations can trigger a type of essentialism grounded in the belief that biological features make persons with mental disorders "categorically different: possessors of the pathological essence." In turn, essentialist views give rise to increases in perceived dangerousness, increased social distancing, and poorer perceived prognosis, as member of the public perceive negative outcomes as being deterministically written into the biology of persons with psychological problems. This framework helps to explain how 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).

Is Biological Essentialism Warranted?

The heritability of major depressive disorder (MDD) is estimated at 37% [95%CI, 31%-42%], based on twin studies comparing concordance rates for monozygotic (identical) and dizygotic (fraternal) twins, and family studies comparing MDD prevalence rates in relatives of MDD-diagnosed probands and those of comparison

subjects (Sullivan, Neale, & Kendler, 2000). Also, heritability may be higher than 37% for recurrent cases that begin earlier in life (Levinson, 2006). However, researchers have yet to identify specific genetic markers that reliably account for the observed MDD heritability. For example, a recent "mega-analysis" of genome-wide association studies (GWAS) examined genetic polymorphisms in approximately 16,000 MDD-diagnosed subjects and 60,000 controls divided across discovery and replication phases, and the analysis produced null findings (i.e., no locus achieved genome-wide significance across discovery and replication phases; Major Depression Working Group of the Psychiatric GWAS Consortium, 2013). In addition, a polygenic risk profile developed using 80% of the cases from the discovery sample and used to predict MDD vs. control status in the other 20% only accounted for 6% of the variance in status (Major Depression Working Group of the Psychiatric GWAS Consortium, 2013).

One possible explanation for the difficulty researchers have had finding genetic variants linked to MDD has to do with the complexity and heterogeneity of cases that are grouped together within the diagnostic label of MDD (Bogdan, Nikolova, & Pizzagalli, 2013; Major Depression Working Group of the Psychiatric GWAS Consortium, 2013). If MDD is not a unified construct, it may be more useful to search for genetic markers at the level of specific endophenotypes (or, intermediate phenotypes) such as reward processing or stress sensitivity (Bogdan et al., 2013; Miller & Rockstroh, 2013). Indeed, there is evidence to suggest that genetic variants that decrease the activity of dopamine in subcortical regions such as the striatum and increase the activity of dopamine in the cortex may be linked to deficits in reward learning and anticipation of reward. However,

most of the studies conducted thus far have not found links between MDD and genetic variants associated with decreased reward processing (Bogdan et al., 2013).

Genetic variants that play a role in the hypothalamic-pituitary-adrenal (HPA) axis have been linked to differential responses to stress, as well as to MDD in some GWAS results (though recall that the largest GWAS to date produced null findings; Major Depression Working Group of the Psychiatric GWAS Consortium, 2013). In fact, arguably the strongest findings linking genes to MDD to date are those showing that certain genetic variants involved in the HPA-axis and stress reactivity may confer elevated risk for developing depression when exposed to highly stressful environments (Binder & Nemeroff, 2010; Bogdan et al., 2013; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010). Notably, however, these findings support the existence of gene x environment interactions, rather than main effects of gene variants on MDD.

It is possible that future research will yield more robust evidence of genetic or neurobiological factors that account for the observed heritability of depression. However, it is also possible that our current heritability figures overestimate the role of genes in the etiology of depression. For example, heritability estimates derived from twin studies are based on the assumption that twins reared together share an identical environment regardless of whether they are monozygotic or dizygotic (i.e., the "equal environment assumption"), and some critics argue that this assumption is flawed and leads to inflated estimates of genetic contributions to variance (Joseph, 1998). Family studies also have limitations; for example, having family members who have been depressed might increase the chances of an individual seeking clinical services, and thereby increase rates of diagnosis (Sullivan et al., 2000). Moreover, clinicians frequently gather information

about a patient's family history as part of routine care, and that knowledge may influence diagnostic impressions; in fact, some would argue that family history should be a key consideration in diagnostic decision-making, because psychological disorders are heritable. If clinicians' belief that depression is heritable is, in turn, based on data showing that depression is diagnosed more often in family members of depressed individuals, then a tautological feedback loop may arise between research and clinical practice.

Thus, genetically-determined neurobiology almost certainly plays some role in the development of depression, but the extent and nature of that role are currently unknown, and it may take many more years of research before those questions can be answered. Furthermore, even if we were to find robust evidence supporting strong genetic and biological contribution to the etiology of depression, the way in which that information is presented could have a negative impact on depressed individuals.

Biological Explanations, Coping Strategies, and Treatment

Evidence linking biological explanations for depression to negative, fatalistic views of prognosis (e.g., expectations that symptoms will be (a) chronic, (b) resistant to any efforts to change, and (c) unlikely to remit) is especially troubling given that expected prognosis influences actual outcomes across a range of clinical conditions, including depression (Glattacker, Heyduck, & Meffert, 2013; Kirsch & Low, 2013; Mondloch, Cole, & Frank, 2001). Thus, it is possible that biological explanations for depression might have a negative impact on actual outcomes in the lives of depressed persons.

There are several plausible means by which such an impact could occur. First, as Kirsch and Low point out (2013), a negative view of one's prognosis is a form of hopelessness, and hopelessness is a core component of depression and one that tends to perpetuate other depressive symptoms (Roepke & Seligman, 2016). In short, believing you will always be depressed is depressing. Second, biological explanations contribute to a belief that one's own efforts to improve one's depressed mood will not make a difference (Deacon & Baird, 2009; Lebowitz, 2014; Lebowitz et al., 2013), and this belief can make a person less likely to engage in effective emotion regulation strategies or make positive lifestyle changes (Kneeland, Dovidio, Joormann, & Clark, 2016), or more likely to believe that those positive changes will ultimately result in something negative (Winer et al., 2017; Winer & Salem, 2016). Third, persons who believe they are biologically fated to struggle with depression might also have low expectations for treatment—especially non-drug treatments such as psychotherapy (Deacon & Baird, 2009)—and treatment expectancies are strongly linked to treatment engagement and outcomes (Greenberg, Constantino, & Bruce, 2006).

Emotion Malleability Beliefs

Perceived ability to improve one's depressed mood is closely related to emotion malleability beliefs, or the extent to which one believes that emotions are malleable and can be changed through personal efforts (De Castella et al., 2013; Kneeland et al., 2016; Tamir, John, Srivastava, & Gross, 2007; Veilleux, Salomaa, Shaver, Zielinski, & Pollert, 2015). Belief that one's emotions are malleable is associated with more successful emotion regulation, due to the use of more effective strategies enacted earlier on in emotion-eliciting situations (Kneeland et al., 2016; Tamir et al., 2007). Conversely, belief

that one's emotions are uncontrollable is associated with reliance on ineffective and potentially harmful strategies such as rumination and emotion suppression (Kneeland et al., 2016; Tamir et al., 2007). Given that biological explanations reduce perceived controllability of depressed moods, it is possible that biological explanations for depression might impede effective emotion regulation by depressed persons, who are already prone to relying on maladaptive emotion regulation strategies (Joormann & Vanderlind, 2014; Kneeland et al., 2016).

Treatment Expectancies

It is also possible that biological explanations for depression could reduce perceived helpfulness of psychotherapy, thus interfering with help-seeking, therapeutic engagement, and psychotherapy outcomes (Kichuk, Lebowitz, & Adams, 2015). The link between treatment expectancies and psychotherapeutic process and outcomes is already well established (Delsignore & Schnyder, 2007; Greenberg et al., 2006). Much less is known about whether or not causal explanations for depression impact treatment expectancies for psychotherapy, but there is evidence suggesting that they might. For example, research indicates that hopelessness is associated with lower treatment expectancies (Goldfarb, 2002). Although Lebowitz and colleagues 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 & Baird, 2009; Kemp et al., 2014; Kvaale, Gottdiener, & Haslam, 2013; Kvaale, Haslam, et al., 2013; 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 those with different causal attributions for depression (Iselin & Addis, 2003). Consistent with this idea, when causal explanations for depression were experimentally manipulated in participants instructed to imagine they were depressed, biological explanations led to significantly lower perceived helpfulness ratings for psychotherapy, but higher perceived helpfulness for antidepressants (Deacon & Baird, 2009). After participants read a biopsychosocial explanation these effects were reversed, with psychotherapy rated as more helpful and antidepressants as less helpful (Deacon & 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.

The two extant studies that experimentally manipulated causal beliefs in depressed participants included measures of perceived helpfulness 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 and colleagues 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 and colleagues (2014), found no significant difference in perceived credibility or helpfulness of psychosocial treatment when comparing participants in the serotonin deficiency condition and 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 difficult to interpret because the control group was not given an alternative explanation for their symptoms; therefore, receiving "normal" test results might have limited their belief that either type of treatment would be helpful (Kemp et al., 2014). Alternatively, depressed individuals might 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 thus a one-time manipulation might not be sufficient to change their beliefs.

Rationale for the Present Study

The studies discussed thus far demonstrate that biological causal explanations for depression—in particular, explanations that promote a fixed view of biological or genetic factors—can lower perceived ability to improve one's mood and may contribute to negative, fatalistic views of prognosis. In light of those findings, it is reasonable to ask whether biological causal explanations might interfere with psychotherapy, perhaps by limiting expectations for improvement in therapy and/or undermining the perceived credibility of non-medical treatments. At present, this is an empirical question. There is a general lack of experimental research examining the impact of biological causal

explanations on beliefs about treatment, and aspects of prior study design (i.e., use of a repeated-measures design, lack of differentiation between antidepressants and psychotherapy in treatment questions, only including a biological explanation condition and control condition) make it difficult to interpret the handful of experimental findings that have been conducted on the topic. 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 actual behaviors that might be relevant to treatment. The present study was aimed at addressing these gaps.

Hypotheses

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 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) The effect of explanation (fixed vs. malleable) on willingness to accept treatment will differ based on treatment option (antidepressant vs. psychotherapy).
    - Within the antidepressant referral condition, participants who receive the fixed explanation for depression will report greater willingness to accept treatment than those who receive the malleable explanation.

- Within the psychotherapy referral condition, participants who receive the fixed explanation for depression will report lower willingness to accept treatment than those who receive the malleable explanation.

2) Individuals in the fixed condition will report lower treatment expectancies (i.e., perceived helpfulness, perceived credibility) for psychotherapy and higher treatment expectancies for antidepressant medication than those in the malleable condition.
3) Individuals in the fixed condition will endorse a more negative perceived prognosis for depression (i.e., more severe and chronic symptoms, lower likelihood of full recovery) than those than those in the malleable condition.
4) Individuals in the fixed condition will report stronger endorsement of the idea that individuals are constrained by their emotional states.

CHAPTER II

METHODS

Study Overview

First, participants completed a computerized task and were presented with bogus test results indicating that, based on their scores on the task, they are at high risk for experiencing depression within the next 6 months. The computer task itself had the advantage of not being associated with a biological model of depression in the same way that other procedures, such as swabbing of cheek cells, might be. Together, the computer task and the bogus results were intended to serve as a plausible cover story (see King et al., 2008, for a real-world example of a major depression risk prediction algorithm) that could reasonably be expected to yield more valid data than might be obtained by simply asking individuals to imagine being depressed. Next, following the design used by Lebowitz and colleagues (Lebowitz et al., 2013), participants were randomly assigned to watch either a video presenting a fixed biological explanation for depression, or a video presenting an explanation that emphasizes the malleability of risk factors for depression. Participants then completed self-report measures assessing treatment expectancies for antidepressant drugs and psychotherapy separately. Beliefs about emotion regulation and other relevant variables were also measured. Finally, participants viewed and responded to a series of video prompts encouraging them to seek treatment and emphasizing the

importance of early intervention in treating depression. The videos offered participants the chance to make an initial appointment to discuss treatment with a care provider. Again based on random assignment, half of the individuals in the fixed explanation condition and half of those in the malleable explanation condition were offered an appointment with a health care provider to discuss antidepressant medication, whereas the other half in each condition was offered an appointment with a counselor to discuss psychotherapy. Participants' decisions to accept or decline the appointment served as a behavioral measure of treatment acceptance, and those who declined the appointment were asked follow-up questions about their willingness to accept treatment in the future.

Measures

Beliefs about Causes of Depression

As a manipulation check, participants were asked to rate on a scale from 1-7 (1 = very unlikely, 7 = very likely) the likelihood that each of several factors "might cause sad, blue, or depressed feelings". The factors rated were based on the factors measured by Lebowitz and colleagues (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. The remaining items (sans 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.

Emotion and Regulation Beliefs

The Emotion and Regulation Beliefs Scale (ERBS; Veilleux et al., 2015) was administered to assess participants' beliefs about whether or not emotions can be regulated and whether or not it is worthwhile to attempt to regulate emotions. The ERBS is a validated, 21-item self-report scale that measures beliefs in three domains: Emotion Constraint, or the extent to which one feels constrained by emotions; Regulation Worth, or the extent to which one believes it is valuable to try to regulate emotions; and Hijacking, or the extent to which one believes that emotions can cause a person to lose control of his or her behavior. Responses are given on a 5-point scale (1 = strongly agree, 5 = strongly disagree).

Prognostic Pessimism

Perceptions about the typical prognosis associated with depression was assessed via the following questions: “How long do you think that the average depressed person will continue to feel sad, blue, or depressed,” and “How long do you think that the average depressed person will continue to feel sad, blue, or depressed if they receive treatment?” Responses were given using a 9-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, 1 to 2 years, 2 to 5 years, More than 5 years, but not indefinitely, and Indefinitely (coded as 9; Lebowitz et al., 2013). In addition, participants were asked to estimate on a 0-100% scale the odds that a person’s depressive symptoms will “go away”, as well as the odds that a person’s depressive symptoms will “return or grow worse” in the future.

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 & Borkovec, 2000), a validated 6-item self-report measure. The CEQ includes 4 items measuring credibility (such as, “At this point, how logical does the above treatment seem?”) and 2 items measuring expectancy (such as, “At this point, how much do you really *feel* that this treatment would help to reduce depressive symptoms?”). Because participants may not personally have 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 the treatment for depression in general. The CEQ has been shown to possess adequate test-retest reliability and internal consistency (Devilly & 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, Ferriter, & Holmberg, 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% - 100%), but these two items were converted to a 9-point scale (responses from 40%-60% are collapsed into a single value and scored a 5) at scoring, to match the other items in the measure.

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 & Borkovec, 2000). All item wording and scoring procedures was 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.

Implicit Association Test (IAT)

In the IAT, participants are instructed to match different categories of stimuli as quickly as possible, and scores are computed by comparing average response times for different category combinations, with the assumption that participants will give faster responses when matching categories that they perceive to be similar than when matching categories that they perceive to be dissimilar (Greenwald, McGhee, & Schwartz, 1998).

For the present study, the stimuli were taken from previous research using the IAT to study implicit depressive attitudes, and included: I, Me, Self, Myself, Mine, They, Them, Their, Theirs, Other, Depressed, Helpless, Hopeless, Gloomy, Withdrawn, Smiling, Glad, Cheerful, Joyful, Delighted (Meites, Deveney, Steele, Holmes, & Pizzagalli, 2008). Data from the IAT were not intended to be examined as part of the present study, and these data were not logged due to experimenter error. 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.

Willingness to Accept Treatment

The computer program used to assess willingness to accept treatment was created and administered using E-Prime 2.0 Professional. The program comprised a series of questions, interspersed with short videos depicting an actor dressed professionally, 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.

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 that day. Participants who declined to schedule an appointment viewed follow-up videos and answered questions about their willingness to schedule an appointment if they were to experience an increase in symptoms. After participants responded to the first treatment referral, they repeated the process with the second treatment option (i.e., those who were initially offered the psychotherapy referral were then offered the antidepressant medication referral, and vice versa). Only data from the initial treatment referral is included in analyses in the present manuscript, and henceforth the term "referral type" will be used to denote the *first* referral presented to each participant.

For each treatment option, willingness to accept treatment was operationally defined on the basis of (a) whether or not participants chose to schedule an appointment, 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. Specifically, treatment willingness was scored using the following 11-point scale:

11 = Participant scheduled an appointment and selected a date 0-7 days away.

10 = Participant scheduled an appointment and selected a date 8-14 days away.

09 = Participant scheduled an appointment and selected a date 15-21 days away.

08 = Participant scheduled an appointment and selected a date 22 or more days away.

07 = Participant declined to schedule an appointment right away, but “definitely” intends to call the relevant resource (i.e., either the Student Health Center or Student Counseling Services) within a week to schedule.

06 = Participant declined to schedule an appointment right away, but might call the relevant resource within a week to make an appointment (indicated greater than 50% likelihood).

05 = Participant declined to schedule an appointment right away, but might call the relevant resource within a week to make an appointment (indicated 50% or lower likelihood).

04 = Participant declined to schedule an appointment right away and will "probably not" call the relevant resource within a week to make an appointment, but would "definitely" make an appointment if depressive symptoms were to increase in the future.

03 = Participant declined to schedule an appointment right away and will "probably not" call the relevant resource within a week to make an appointment, but might make an appointment if symptoms were to increase in the future (indicated greater than 50% likelihood).

02 = Participant declined to schedule an appointment right away and will "probably not" call the relevant resource within a week to make an

appointment, but might make an appointment if symptoms were to increase in the future (indicated 50% or lower likelihood).

01 = Participant declined to schedule an appointment right away, will "probably not" call the relevant resource within a week to make an appointment, and would “probably not” make an appointment even if symptoms were to increase in the future.

Procedure

Participants who volunteered through SONA to take part in the study were scheduled to come to the lab for the experimental session. Upon arriving, participants were presented with an initial 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 opened the computer program in E-Prime 2.0 Professional, which began with the Implicit Association Test (IAT). Once the instructions appeared onscreen, the researcher left the room. After the IAT, the computer automatically presented the QIDS-SR (sans 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 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.

Fixed biological explanation. The script for the fixed biological explanation video was as follows:

> Hello, I work in the field of clinical psychology as a researcher and clinician, and I want to thank you for your time today. You just completed a computer task called the "Reaction Time Test." The Reaction Time Test is a recently-developed depression screening test that measures dysfunction in the brain's processing of emotional information, which is an early and highly reliable marker of depression risk.
>
> Researchers at Mississippi State University are currently developing an online service that will allow students to log in, take the depression screening test, and get immediate feedback about their risk of developing depression, based on their scores. They will also be provided with information to help them reduce their risk.

After you finished the test, the computer automatically calculated your score. Your score will now be presented, along with some information to help you understand what your score means.

If you scored in the Low Risk range, a Low Risk video will play to explain your score. If you scored in the Moderate Risk range, a Moderate Risk video will play, and if you scored in the High Risk range, a High Risk video will play.

[*voice over as graph is displayed*]

The graph you see now shows your results from the depression screening test. This video is playing because your reaction times were faster when Me related words were in the same category with Sad words, and Not-Me related words were in the same category with Happy words. As you can see on the graph, your scores fall in the High Risk range. Your results indicate that certain parts of your brain are hypersensitive to negative information. Even if you have not consciously noticed mood problems yet, your test results indicate that you are at very high risk of becoming depressed in the near future, because the parts of your brain that process negative emotional information are overactive. In order to help you understand what your scores mean, some information about depression will now be presented.

Many people who have symptoms of depression, or know someone who does, wonder what causes these kinds of problems. Mood problems, such as depression, run in families. 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. For example, not everybody who gets mistreated as a child becomes depressed later in life; it is the person's genetic makeup that determines whether that person will become depressed in response to the abuse. Our genes can even influence what environments we end up in, so some people may find themselves in depressing circumstances because of their genes.

In recent years, neuroscience has shown us that depression is truly a brain disorder. Brain imaging has shown that there are real differences between the brains of depressed people and the brains of non-depressed people. Depressed people have abnormalities in critical areas of the brain. The area that is involved in emotional reactions to the environment is over-active; this explains why many depressed people over-react to stressful situations in their lives. At the same time, the brain area that is involved in solving problems is under-active, making it difficult for depressed individuals to think clearly or act effectively to solve their

problems. The following clip will explain a little bit about the biology of depression.

[*Clip from http://www.thevisualmd.com/health_centers/neurological_health/depression/what_is_depression_video*]

As you may know, antidepressants are sometimes given to people with depression. The benefits of these medications, however, tend to be only temporary; scientific studies have shown that depression frequently comes back when people stop taking their medication. Once the medication wears off, there is no longer anything stopping the person's genes from causing chemical imbalances in the brain, so brain chemistry often returns to the way it was before the person started taking the medication. Therefore, it is important for depressed people to continue taking their medication consistently, much like people with diabetes, high cholesterol, high blood pressure or other chronic illnesses must take their medication every day. Some scientists believe that psychotherapy—the process of talking to a professional therapist—is beneficial mainly because it helps patients make sure they take their medication regularly. Psychotherapy may also help depressed individuals cope with their symptoms and endure the negative effects of their disorder. There are also self-help books that can be used for this purpose. Sometimes, when other treatments are not working, doctors will try electroconvulsive therapy – sometimes called electric shock therapy -- to treat depression. This treatment causes a seizure in the brain, and while scientists do not fully understand how it works, it is known to benefit some very depressed

individuals. Although no treatment for depression has a 100% success rate, there are several options that professionals can use when treating a person with this disease.

Malleable explanation. The script for the malleable explanation video was as follows:

Hello, I work in the field of clinical psychology as a researcher and clinician, and I want to thank you for your time today. You just completed a computer task called the "Reaction Time Test." The Reaction Time Test is a recently-developed depression screening test that measures dysfunctional habits of responding to emotional information, which is an early and highly reliable marker of depression risk.

Researchers at Mississippi State University are currently developing an online service that will allow students to log in, take the depression screening test, and get immediate feedback about their risk of developing depression, based on their scores. They will also be provided with information to help them reduce their risk.

After you finished the test, the computer automatically calculated your score. Your score will now be presented, along with some information to help you understand what your score means.

If you scored in the Low Risk range, a Low Risk video will play to explain your score. If you scored in the Moderate Risk range, a Moderate Risk video will play, and if you scored in the High Risk range, a High Risk video will play.

[*voice over as graph is displayed*]

The graph you see now shows your results from the depression screening test. Your reaction times were much faster when Me-related words were in the same category with Sad words and Not-Me related words were in the same category with Happy words. As you can see on the graph, your scores fall in the High Risk range. Your results indicate that you have developed a habit of hypersensitive attention toward negative information. Even if you have not consciously noticed mood problems yet, your test results indicate that you are at very high risk of becoming depressed in the near future, because you have learned to process negative emotional information in an over-active way, and this style of thinking has become automatic over time.

In order to help you understand what your scores mean, some information about depression will now be presented.

Many people who have symptoms of depression, or know someone who does, wonder what causes these kinds of problems. The truth is, whether or not a person becomes depressed depends upon a wide variety of factors. Genetics alone can never make someone depressed. In fact, even among people who have an immediate family member with depression, a large majority do not become depressed themselves. Even if a person has a genetically identical twin with depression, most of the time that person will not become depressed.

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. You could think of genes like the heating system in your house, while other factors act like the thermostat. The heating system is always there, but it is not always active—the settings on the thermostat determine whether the heat will be on or not. Similarly, the genes we are born with are always there, but this does not mean all of them will always be active or turned on. The following clip, from the University of Utah's Genetic Science Learning Center, will explain a little bit about how genes get turned on and off.

[*Clip from video at http://learn.genetics.utah.edu/content/epigenetics/twins*]

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. One way to exercise the brain is through learning, which can strengthen or change the activity of cells in the brain. For a depressed person, it can be very helpful to learn new ways of thinking or interacting with others, sometimes with help from a professional therapist, or the kinds of self-help books that are widely available

Brain-imaging studies have looked at changes in the brains of people whose depression improved after learning and practicing these kinds of skills and have found something remarkable. These people's patterns of brain activity were

found to look more like those of people without depression. That is, their brain activity changed for the better, and because of what they had learned, their depression improved.

There are many pathways out of depression, and there are many things that can help people along these paths. For instance, medications are available that can help reduce or control the symptoms of depression, which can make it easier to learn the kinds of skills that allow people to be in control of their mood. Such skills will stay with a person for a long time—just like learning to ride a bicycle, and many people find that learning them can help keep depression away. 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. Whatever might be causing a person's depression, there are many types of support available to help deal with it.

Post-Manipulation Questionnaires

After the causal explanation video ended, the computer automatically presented the remaining self-report measures, with the exception of the demographic and funnel debriefing questionnaires, and instructed participants to respond using the keyboard. See Appendix C for the full text of these measures.

Treatment Referrals

After the self-report measures were completed, the computer automatically presented the treatment referral videos and questions assessing willingness to accept treatment. The order in which referrals were presented was randomized, with participants

viewing either the antidepressant or psychotherapy referral first depending on the referral condition to which they had been randomly assigned.

Debriefing

After participants responded to both treatment referrals, the demographic questionnaire was automatically administered via computer, followed by the funnel debriefing questionnaire. See Appendix C for the full text of these measures. Next, the researcher returned to the room and stated that there would be one last video to watch as part of the session. The researcher opened the debriefing video, and left the room when it began to play. See Appendix B for the full script of the debriefing video.

After the debriefing video ended, the researcher returned to the room and presented the participant with a data use permission form, which they were asked to sign if they granted permission for their data to be used for research purposes. Finally, item 12 of the QIDS-SR was administered on paper to assess for recent thoughts of death or suicide. According to IRB-approved procedures, participants were screened for risk 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. All participants were then given a list of contact information for local and national mental health resources before being dismissed.

CHAPTER III

RESULTS

Participants

Participants were recruited via SONA from the undergraduate subject pool at a large Southern university, and were awarded 1 hour of course credit in exchange for participating. 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 are presented in Table 1.

Table 1

Participant Characteristics (N = 139)

Gender (%)		
Female	77.7	
Male	21.6	
Ethnicity (%)		
White non-Hispanic	63.3	
Black non-Hispanic	32.4	
Hispanic or Latino	1.4	
Asian or Pacific Islander	0.7	
Other	2.2	
Age	*M* = 19.77	*SD* = 1.64

Note. Data on gender was missing for one participant. Data on age was missing for two participants.

Missing Data and Internal Consistency

A total of 35 values were missing from self-report measures assessing depressive symptoms, beliefs about causes of depression, emotion and regulation beliefs, prognostic pessimism, and expectancy and credibility of psychotherapy and antidepressant medication, which amounted to 0.38% of the self-report data. The greatest amount of data to be missing from a single participant was three values, amounting to 4.35% of the data for that individual.

All of the missing self-report data from these measures came from items where participants were asked to use the keyboard to type in percentages: Item 3 and Item 4 of the prognostic pessimism scale (three and four missing values, respectively; 1.3% of data missing from the measure), Item 4 and Item 6 of the credibility and expectancy questionnaire (CEQ) for antidepressant medication (five and 12 missing values, respectively; 2.04% of data missing from the measure), and Item 4 and Item 6 of the CEQ for psychotherapy (four and seven missing values, respectively; 1.32% of data missing

from the measure). In addition to the missing self-report data, four values were missing from the measure of willingness to accept treatment with antidepressant medication, and one value was missing from the measure of willingness to accept psychotherapy.

In order to assess whether the missing values were missing completely at random (MCAR), Little's MCAR test was carried out in SPSS v. 24 on all items to be included in analyses for the present study. Little's test yielded non-significant results, indicating that missing values in the present data set are MCAR. 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 & Fidell, 2013).

The five missing values from the treatment willingness scales 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. Because participants with missing values on these items did respond to the preceding items on the treatment willingness scales, their scores could be identified within a two-point range.[1] Thus, in these five cases, scores were computed by taking the mid-point between the two values that might have been assigned if participants had responded to all of the items validly. For instance, if a participant's score might have been either a 5 or 6 but the percent likelihood item was missing, then a score of 5.5 was entered.

[1] For instance, a participant who responded that they did not wish to make an appointment right away but would "maybe" make an appointment on their own within a week would score either a 5 or a 6 on the relevant treatment willingness measure, depending whether or not they rated the probability of doing so above 50%.

To assess internal consistency, Cronbach's alphas were calculated. Results indicated adequate internal consistency for the QIDS-SR sans item 12 (α = .79), the CEQ for antidepressant medication (α = .89), the CEQ for psychotherapy (α = .85), depression mind set (α = .89), ERBS Emotional Constraint (α = .80), and ERBS Regulation Worth (α = .76). Internal consistency for ERBS Hijack (α = .67) and prognostic pessimism (α = .51) was lower.

Manipulation Checks

After viewing either the fixed biological or malleable explanation video, participants were asked to rate the likelihood that various factors might cause depressed mood. 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).

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. Group means and standard deviations for each of the factors are presented in Table 2. 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 $d = 0.58$.[2] 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.

[2] Given the skewness and kurtosis observed in ratings for some items, Mann-Whitney tests were also conducted comparing causal explanation groups and yielded similar results.

Table 2

Beliefs about the Causes of Depression

	Fixed Biological Explanation (n = 67)		Malleable Explanation (n = 72)			
	M	*SD*	*M*	*SD*	*p*	*Cohen's d*
Genetics	5.46	1.76	2.99	1.93	<.01	1.34
Brain chemistry or biochemical imbalance	5.93	1.51	4.26	1.78	<.01*	1.01
Day to day problems or stress	5.81	1.28	6.04	1.36	.30	.17
Beliefs or style of thinking	5.19	1.61	5.39	1.77	.50	.12
Abnormal brain structure or development	5.87	1.36	4.29	1.67	<.01*	1.04
Brain injury	5.61	1.47	4.71	1.64	<.01	.58
Substance abuse	5.73	1.62	5.97	1.16	.32*	.17
Weakness of character	4.34	2.01	4.75	1.85	.22	.21
Problems from childhood or the way you were raised	5.09	1.87	5.42	1.68	.28	.19
Recent traumatic events	5.97	1.38	6.13	1.33	.50	.12

Note. Mean comparisons presented in Table 2 were conducted via independent samples *t*-tests. Mann-Whitney *U*-tests were also carried out and produced similar results. For items where Levene's test indicated unequal variance, degrees of freedom were adjusted for the significance tests reported in Table 2 and *p*-values are marked with an asterisk.

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. Percentages of participants who expressed doubts within each combination of causal explanation and referral type are presented in Table 3.

Table 3

Participants Grouped by Explanation Condition and Treatment Referral Type

	Fixed Biological Explanation				Malleable Explanation			
Referral type	Medication (n = 34)		Psychotherapy (n = 33)		Medication (n = 31)		Psychotherapy (n = 41)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Treatment willingness	3.97	2.67	4.47	2.64	3.63	2.69	5.02	2.82
Depressive symptoms	7.65	4.68	6.88	5.01	6.48	3.62	8.32	4.16
Treatment history (%)								
Yes	47.1		39.4		16.1		36.6	
No	52.9		60.6		83.9		63.4	
Doubts (%)								
Yes	38.2		45.5		45.2		43.9	
No	61.8		54.5		54.8		56.1	

Note. Treatment willingness = willingness to accept initial treatment referral; Depressive symptoms = Quick Inventory of Depressive Symptomatology – Self Report sans item 12; Treatment history = current or past psychotherapy or use of prescribed medications to treat psychological symptoms; Doubts = response to question "Did you have doubts about any part of the study?"

Of those who did report having doubts, almost all 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" (that is, the IAT) 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, it is possible that participants still accepted the information about depression in general and its causes as accurate, even if they had doubts about the depression screening test and bogus test results. In order to

quantitatively assess this possibility, the independent samples *t*-tests using explanation condition as the IV and ratings about the likelihood of factors causing depression as the DVs were repeated with only those participants (n = 60) who reported doubts about the study. Results still indicated significant differences between explanation conditions, with 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 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, albeit to a lesser degree with regard to biological factors than those who had no doubts. Nonetheless, in order to assess whether credulity belief with regard to the potential cause of depression 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

Preliminary Analyses

Participants were grouped according to explanation condition (i.e., whether a participant viewed the fixed biological or malleable explanation for depression) and initial treatment referral (i.e., whether a participant was initially referred to the student health center for antidepressant medication or the student counseling center for psychotherapy). This yielded four cells: fixed/antidepressant referral (n = 34), fixed/psychotherapy referral (n = 33), malleable/antidepressant referral (n = 31), and malleable/psychotherapy referral (n = 41).

For each cell, skewness and kurtosis statistics for the initial treatment willingness scale[3] were computed. Skewness values ranged from .748 to .989 and kurtosis ranged from .401 to .864, all of which fell within acceptable limits (Kline, 2010). The variance ratio between the largest cell variance and smallest was 1.14, indicating that the variance in treatment willingness was sufficiently similar across cells (Field, 2013).

Main Analyses

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 x 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 are presented in Table 3.

[3] That is, the scale corresponding to whichever treatment option was presented first.

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 $\eta^2 = .01$. Thus, Hypothesis 1 was not supported, as no main effects were predicted and a significant interaction was predicted.

Hypothesis 2

Preliminary Analyses

Participants were grouped according to explanation condition, yielding two groups: fixed biological explanation ($n = 67$), and malleable explanation ($n = 72$). Assumption checks were then carried out on within-subject differences between treatment expectancies for antidepressant medication and for psychotherapy. Skewness (-.786 and .283, respectively) and kurtosis (.245 and -.205, respectively) values all fell within acceptable limits (Kline, 2010). The variance ratio between the largest cell variance and smallest was 1.11, indicating sufficient equality of variance (Field, 2013).

Main Analyses

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 (CEQ for psychotherapy and CEQ for antidepressant medication) as the within-subjects variable. Means and standard deviations are presented in Table 4.

Table 4

Variable Means in Fixed Biological and Malleable Explanation Conditions

	Fixed Biological Explanation (n = 67)		Malleable Explanation (n = 72)			
Variable	*M*	*SD*	*M*	*SD*	*p*	*Cohen's d*
CEQ antidepressant medication	37.54	9.83	33.69	8.40	.01	.42
CEQ psychotherapy	37.33	8.61	36.50	8.34	.56	.10
Prognostic pessimism	18.94	4.71	18.60	4.27	.65	.08
ERBS Emotional Constraint	22.45	6.65	24.26	5.97	.09	.29
Item-level mean	2.49		2.70			
ERBS Regulation Worth	26.85	4.75	27.78	3.92	.21	.21
Item-level mean	3.84		3.97			
ERBS Hijack	15.55	3.42	16.85	3.56	.03	.37
Item-level mean	3.11		3.37			
Depression mind set	9.39	4.51	8.10	4.46	.09	.29

Note. Mean comparisons were carried out via independent samples *t*-tests; ERBS Emotional Constraint = Emotion and Regulation Beliefs Scale, Emotional Constraint subscale; ERBS Regulation Worth = Emotion and Regulation Beliefs Scale, Regulation Worth subscale; ERBS Hijack = Emotion and Regulation Beliefs Scale, Hijack subscale; CEQ antidepressant = Credibility and Expectancy Questionnaire, antidepressant medication; CEQ psychotherapy = Credibility and Expectancy Questionnaire, psychotherapy.

The main effect of causal explanation approached significance, $F(1, 137) = 3.70$, $p = .06$, partial $\eta^2 = .03$. Examination of marginal means revealed that among participants given a fixed biological explanation for depression 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 CEQ ratings for psychotherapy (M = 36.50, SD = 8.34) were higher than those for antidepressant medication (M = 33.69, SD = 8.40). However, the interaction of causal explanation and treatment type was non-significant, $F(1, 137) = 3.00$, $p = .09$, partial $\eta^2 = .02$.

To better understand the main effect, 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. Further, examination of the marginal means showed that 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 (see Table 4 for marginal means, SDs, and results of independent samples t-tests comparing CEQ ratings for the fixed biological and malleable groups). Thus, Hypothesis 2 was partially supported.

Hypothesis 3

Preliminary Analyses

Participants were again grouped into fixed biological explanation (n = 67) and malleable explanation (n = 72) conditions, and assumption checks were carried out on the prognostic pessimism scale. Skewness (.427 and .459, respectively) and kurtosis (1.19 and .178, respectively) values fell within acceptable limits (Kline, 2010). The variance

ratio between the largest cell variance and smallest was 1.26, indicating sufficient equality of variance (Field, 2013).

Main Analyses

Hypothesis 3, that participants given a fixed biological explanation for depression would exhibit greater prognostic pessimism than those given a malleable explanation, was tested using an independent samples *t*-test, with causal explanation (fixed or malleable) as the IV and prognostic pessimism as the DV. Means and standard deviations are presented in Table 4. Results of this test were non-significant, $t(137) = .45$, $p = .65$, Cohen's $d = .08$, indicating no difference in prognostic pessimism between the two causal explanation conditions. Thus, Hypothesis 3 was not supported.

Hypothesis 4

Preliminary Analyses

Assumption checks were carried out on the ERBS Emotion Constraint subscale for the fixed ($n = 67$) and malleable ($n = 72$) explanation conditions. Skewness (.381 and .854, respectively) and kurtosis (-.140 and 1.14, respectively) fell within acceptable limits (Kline, 2010) and the variance ratio was 1.24, indicating sufficient equality of variance.

Main Analyses

Hypothesis 4, that participants given a fixed biological explanation for depression would endorse stronger belief that people are constrained by emotions than those given a malleable explanation, was tested using an independent samples *t*-test, with causal explanation (fixed or malleable) as the independent variable and the Emotion Constraint subscale of the ERBS as the dependent variable. Means and standard deviations are presented in Table 4. Results indicated that the effect of causal explanation on

endorsement of emotion constraint beliefs was not significant, $t(137) = -1.70$. $p = .09$, Cohen's $d = .29$. Thus, Hypothesis 4 was not supported.

Exploratory Analyses

Current Depressive Symptoms and Willingness to Accept Treatment

In order to examine whether current depressive symptoms had an impact on participants' willingness to accept treatment referrals, a 2 x 2 ANCOVA was conducted. Causal explanation (fixed or malleable) and type of treatment referral (antidepressant medication or psychotherapy) were included as IVs, current depressive symptoms (QIDS-SR sans item 12) served as a covariate, and willingness to accept treatment served as the DV.

Results showed a significant main effect of current depressive symptoms, $F(1, 135) = 10.68$, $p < .01$, partial $\eta^2 = .07$, indicating that participants reporting higher levels of depressive symptoms were more willing to accept treatment referrals. In addition, the main effect of referral type approached significance, $F(1, 135) = 3.68$, $p = .06$, partial $\eta^2 = .03$, suggesting that participants may have been more willing to accept referrals for psychotherapy than for medication even when adjusting for current depressive symptoms. No other significant main effects or interactions emerged.

Treatment History and Willingness to Accept Treatment

The demographic questionnaire used in the present study included questions about current or past psychotherapy, as well as current or past use of prescribed medications to treat psychological symptoms such as depression or anxiety. Responses to these questions were used to create a binary treatment history variable, categorizing participants on the

basis of whether or not they had ever received treatment (i.e., psychotherapy or medication) for psychological symptoms. From the full sample, 49 participants reported some history of treatment, whereas 90 participants reported no current or past psychotherapy or medication for psychological symptoms.

In order to examine whether treatment history had an impact on participants' willingness to accept treatment referrals, a 2 x 2 x 2 ANOVA was conducted. Causal explanation (fixed or malleable), type of treatment referral (antidepressant medication or psychotherapy), and treatment history (current or past treatment or no treatment history) were included as IVs, and willingness to accept treatment served as the DV. Results showed a significant main effect of treatment history, $F(1, 131) = 6.83$, $p = .01$, partial $\eta^2 = .05$, indicating that participants with current or previous experience with treatment for psychological symptoms showed greater willingness to accept treatment referrals in the present study. However, the main effect of treatment referral condition was no longer significant, $F(1, 131) = 2.21$, $p = .14$, partial $\eta^2 = .02$. No other significant main effects and no significant interactions emerged.

Credulity and Willingness to Accept Treatment

Given that 43.2% of participants endorsed having doubts about the study—specifically, pertaining to the accuracy or validity of the depression screening test—while the other 56.8% reported no doubts, we wanted to examine whether participants' credulity toward the study moderated the impact of the experimental manipulation on willingness to accept treatment. A 2 x 2 x 2 ANOVA was conducted to test this possibility. 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. Results are presented in Table 5.

Table 5

2 x 2 x 2 ANOVA Comparing Willingness to Accept Treatment across Treatment Referral Type, Causal Explanation Condition, and Binary Doubts Variable

Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Explanation condition	1	1.46	1.46	0.21	<.01	<.01
Referral type	1	34.44	34.44	5.01	.01	.04
Doubts	1	30.23	30.23	4.39	.04	.03
Referral type x explanation condition	1	9.46	9.46	1.37	.24	.01
Referral type x doubts	1	2.36	2.36	0.34	.56	<.01
Explanation condition x doubts	1	17.39	17.39	2.53	.11	.02
Referral type x explanation condition x doubts	1	41.31	41.31	6.00	.02	.04
Error	131	901.39	6.88			
Total	139	3630.00				

Note. Dependent variable: willingness to accept treatment; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question "Did you have doubts about any part of the study?"

Consistent with the results from the original test of Hypothesis 1, there was a significant main effect of type of treatment referral 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 $\eta^2 = .04$.

As a means of probing the significant 3-way interaction, separate 2 x 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. Results are presented in Table 6. 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 $\eta^2 = .10$.

Table 6

Separate 2 x 2 ANOVAs Comparing Willingness to Accept Treatment across Treatment Referral Type and Causal Explanation Condition in Participants With and Without Doubts

	Doubts = No (n = 79)						Doubts = Yes (n = 60)					
Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Explanation condition	1	5.09	5.09	0.68	.41	.01	1	12.72	12.72	2.07	.16	.04
Referral type	1	10.87	10.87	1.46	.23	.02	1	24.12	24.12	3.93	.05	.06
Referral type x explanation condition	1	6.51	6.51	0.88	.35	.01	1	39.72	39.72	6.47	.01	.10
Error	75	557.69	7.44				56	343.71	6.14			
Total	79	2302.50					60	1327.50				

Note. Dependent variable: willingness to accept treatment; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question “Did you have doubts about any part of the study?”

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 (Figure 1). 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, $t(32) = 2.92$, $p = .01$, Cohen's $d = 1.04$.

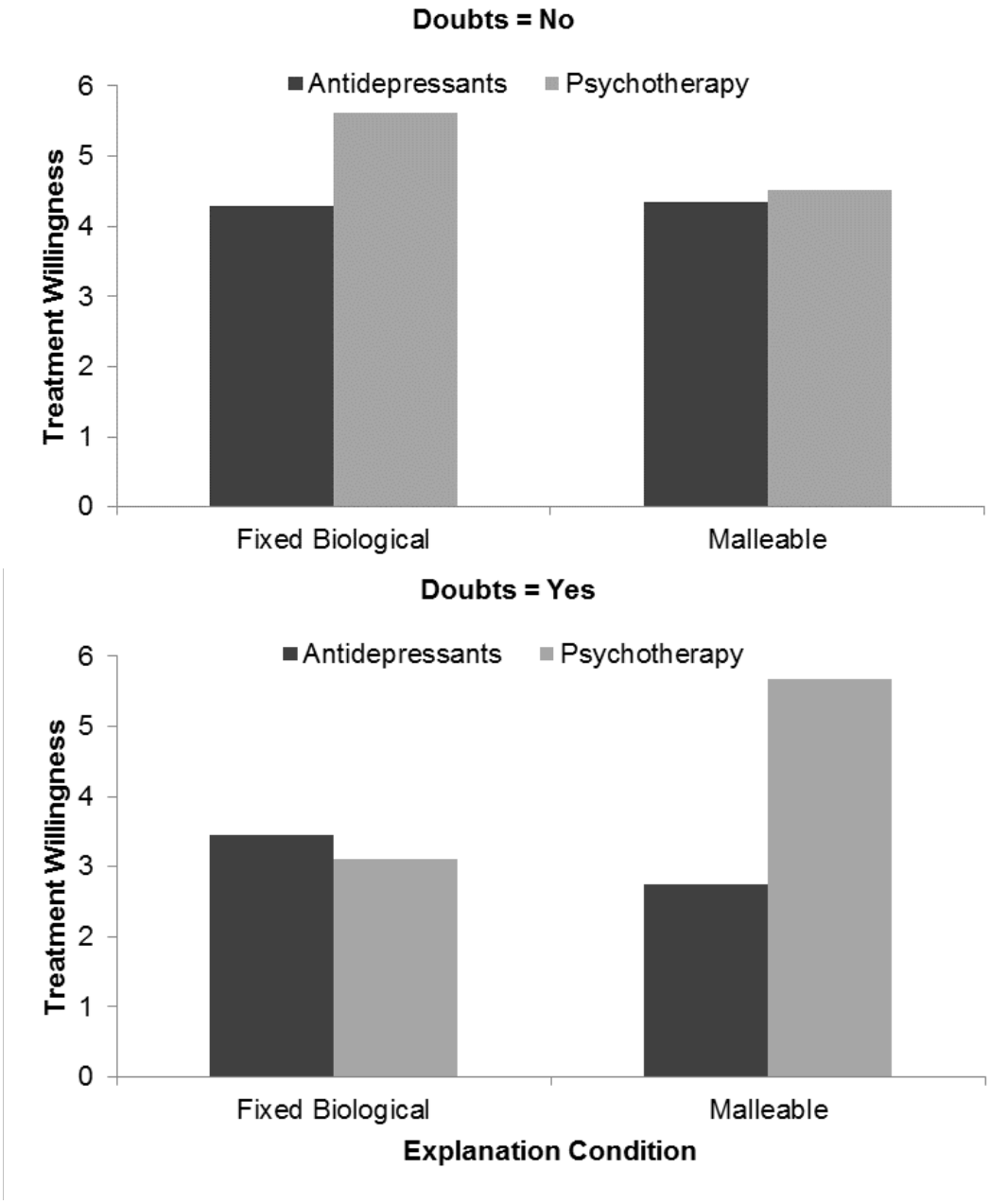

Figure 1. Mean levels of willingness to accept treatment within each combination of causal explanation for depression and treatment referral type, for participants for reported no doubts about the study (top) and those who did report doubts (bottom).

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 x 2 x 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. Results are presented in Table 7. 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 is merely an artifact of differences in depressive symptoms.

Table 7

2 x 2 x 2 ANOVA Comparing Current Depressive Symptoms across Treatment Referral Type, Causal Explanation Condition, and Binary Doubts Variable

Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Explanation condition	1	2.11	2.11	0.11	.74	<.01
Referral type	1	9.81	9.81	0.52	.47	<.01
Doubts	1	115.00	115.00	6.13	.02	.05
Referral type x explanation condition	1	58.07	58.07	3.10	.08	.02
Explanation condition x doubts	1	19.74	19.74	1.05	.31	.01
Referral type x doubts	1	6.43	6.43	0.34	.56	<.01
Referral type x explanation condition x doubts	1	19.29	19.29	1.03	.31	.01
Error	131	2455.84	18.75			
Total	139	10301.00				

Note. Dependent variable: Quick Inventory of Depressive Symptomatology – Self Report sans item 12; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question "Did you have doubts about any part of the study?"

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 explanation at this level, separate 2 x 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. Results are presented in Table 8. The 2-way interaction neared significance among participants who reported having doubts, $n = 60$, $F(1, 56) = 3.97$, $p = .05$, partial $\eta^2 = .07$, and the marginal means of depression within each combination of causal explanation and referral type (Figure 2) bore some similarity to the corresponding values for treatment willingness within each cell (Figure 1). 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.

Table 8

Separate 2 x 2 ANOVAs Comparing Current Depressive Symptoms across Treatment Referral Type and Causal Explanation Condition in Participants With and Without Doubts

	Doubts = No (n = 79)						Doubts = Yes (n = 60)					
Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Explanation condition	1	5.18	5.18	0.25	.62	<.01	1	15.29	15.29	0.96	.33	.02
Referral type	1	18.60	18.60	0.89	.35	.01	1	0.16	0.16	0.01	.92	<.01
Referral type x explanation condition	1	6.04	6.04	0.29	.59	<.01	1	63.46	63.46	3.97	.05	.07
Error	75	1560.50	20.81				56	895.34	15.99			
Total	79	6887.00					60	3414.00				

Note. Dependent variable: Quick Inventory of Depressive Symptomatology – Self Report sans item 12; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question "Did you have doubts about any part of the study?"

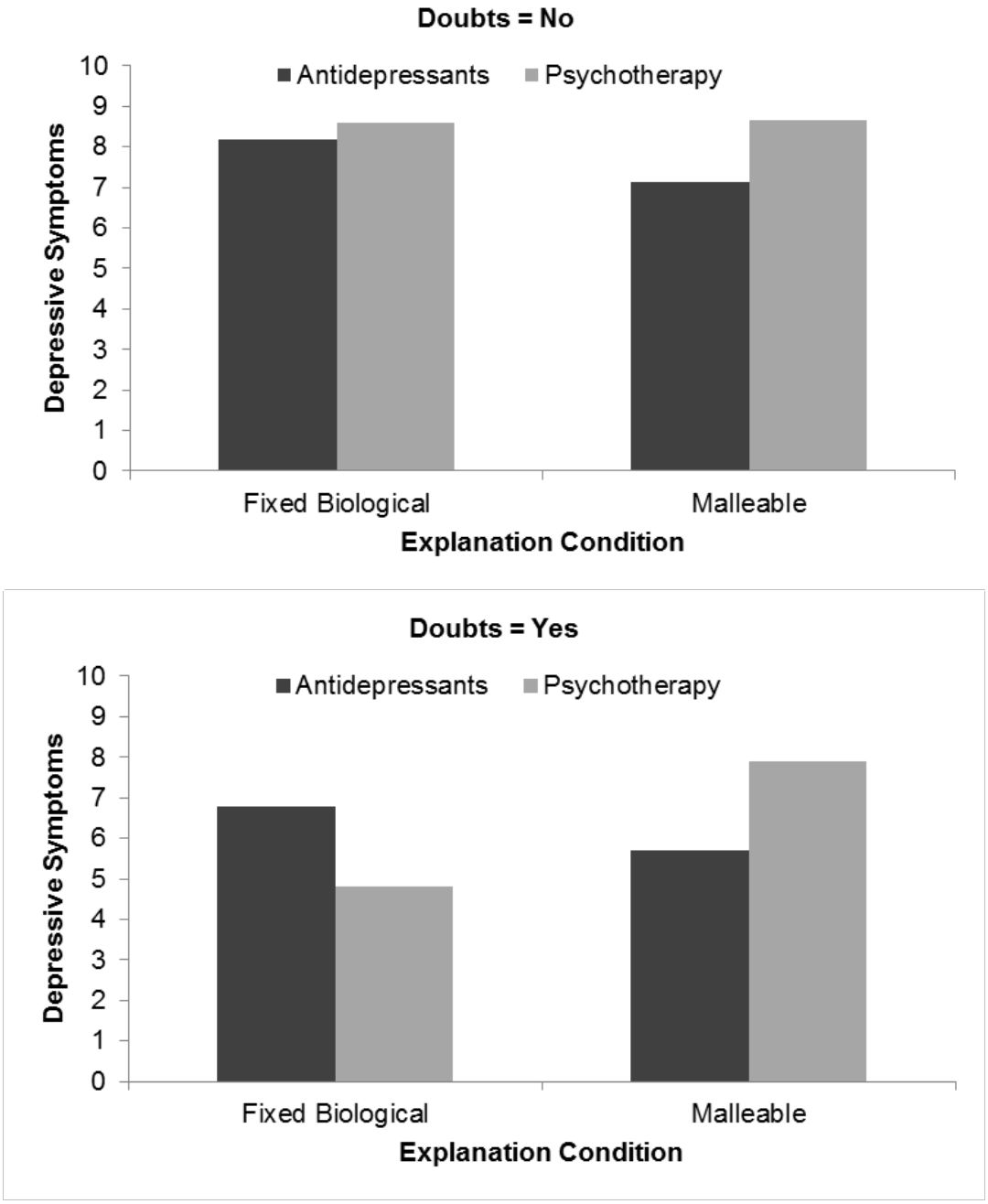

Figure 2. Mean levels of depressive symptoms within each combination of causal explanation for depression and treatment referral type, for participants for reported no doubts about the study (top) and those who did report doubts (bottom).

Finally, the original 2 x 2 x 2 ANOVA and subsequent pair of 2 x 2 ANOVAs within each level of the doubt variable with willingness to accept treatment as the DV were repeated, but this time depression scores were included as a covariate. After adjusting for current depressive symptoms, the 2 x 2 x 2 ANOVA still yielded a significant 3-way interaction of explanation condition, referral type, and doubts, $F(1, 130) = 5.11$, $p = .03$, partial $\eta^2 = .04$ (Table 9). In addition, the 2 x 2 ANOVA 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, $F(1, 55) = 4.57$, $p = .04$, partial $\eta^2 = .08$ (Table 10). Thus, depression was not confounded with this interaction.

Table 9

2 x 2 x 2 ANOVA Comparing Willingness to Accept Treatment across Treatment Referral Type, Causal Explanation Condition, and Binary Doubts Variable when Adjusting for Current Depressive Symptoms

Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Depressive symptoms	1	47.38	47.38	7.21	.01	.05
Explanation condition	1	1.01	1.01	0.15	.70	<.01
Referral type	1	29.41	29.41	4.48	.04	.03
Doubts	1	15.35	15.35	2.34	.13	.02
Referral type x explanation condition	1	3.97	3.97	0.61	.44	.01
Explanation condition x doubts	1	12.52	12.52	1.91	.17	.01
Referral type x doubts	1	3.56	3.56	0.54	.46	<.01
Referral type x explanation condition x doubts	1	33.58	33.58	5.11	.03	.04
Error	130	854.01	6.57			
Total	139	3630.00				

Note. Dependent variable: willingness to accept treatment; Depressive symptoms = Quick Inventory of Depressive Symptomatology – Self Report sans item 12; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question "Did you have doubts about any part of the study?"

Table 10

Separate 2 x 2 ANOVAs Comparing Willingness to Accept Treatment across Treatment Referral Type and Causal Explanation Condition in Participants With and Without Doubts When Adjusting for Current Depressive Symptoms

	Doubts = No (n = 79)						Doubts = Yes (n = 60)					
Source	*df*	*SS*	*MS*	*F*	*p*	partial η^2	*df*	*SS*	*MS*	*F*	*p*	partial η^2
Depressive symptoms	1	38.50	38.50	5.49	.02	.07	1	10.30	10.30	1.70	.20	.03
Explanation condition	1	3.59	3.59	0.51	.48	.01	1	9.74	9.74	1.61	.21	.03
Referral type	1	6.78	6.78	0.97	.33	.01	1	23.71	23.71	3.91	.05	.07
Referral type x explanation condition	1	8.60	8.60	1.23	.27	.02	1	27.72	27.72	4.57	.04	.08
Error	74	519.19	7.02				55					
Total	79	2302.50					60					

Note. Dependent variable: willingness to accept treatment; Depressive symptoms = Quick Inventory of Depressive Symptomatology – Self Report sans item 12; *df* = degrees of freedom; *SS* = type III sum of squares; *MS* = mean square; Doubts = response to question “Did you have doubts about any part of the study?

Credulity and 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 11 for means and *SD*s) 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 $\eta^2 < .01$.

Table 11

Variable Means in Fixed Biological and Malleable Explanation Conditions for Participants With and Without Doubts

	Doubts = No				Doubts = Yes			
	Fixed Biological		Malleable		Fixed Biological		Malleable	
Referral type	(*n* = 39)		(*n* = 40)		(*n* = 28)		(*n* = 32)	
Variable	*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
Prognostic pessimism	19.49	4.51	18.03	4.24	18.18	4.97	19.31	4.26
ERBS Emotional Constraint	23.84	7.16	24.80	6.40	20.50	5.41	23.59	5.40
ERBS Regulation Worth	26.74	4.43	28.23	3.58	27.00	5.24	27.22	4.29
ERBS Hijack	15.80	3.80	17.45	3.10	15.21	2.85	16.09	4.00
Depression mind set	10.74	4.75	8.90	5.05	7.50	3.41	7.09	3.42

Note. ERBS Emotional Constraint = Emotion and Regulation Beliefs Scale, Emotional Constraint subscale; ERBS Regulation Worth = Emotion and Regulation Beliefs Scale, Regulation Worth subscale; ERBS Hijack = Emotion and Regulation Beliefs Scale, Hijack subscale; 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?”

Credulity and Prognostic Pessimism

A 2 x 2 ANOVA was conducted with causal explanation (fixed or malleable) and response to the binary doubts question (yes or no) as IVs and prognostic pessimism as the DV. Neither the main effect of causal explanation, $F(1, 135) = 0.05$, $p = .83$, partial $\eta^2 < .01$ nor the main effect of doubts, $F(1, 135) < 0.01$, $p = .99$, partial $\eta^2 < .01$ approached significance. The 2-way interaction was also non-significant, $F(1, 135) = 2.86$, $p = .09$, partial $\eta^2 = .02$, though examination of marginal means (see Table 11 for means and *SD*s) showed that within the fixed biological explanation condition participants who had no doubts about the study exhibited more prognostic pessimism than those who reported having doubts, whereas in the malleable explanation condition the pattern was reversed, and participants who reported having doubts about the study exhibited more prognostic pessimism than those who had no doubts.

Causal Explanation and ERBS Subscales

Independent samples *t*-tests were carried out to examine whether causal explanation for depression (fixed or malleable) had an impact on the remaining ERBS subscales. The ERBS Regulation Worth subscale measures belief that it is both valuable and possible to learn to regulate one's emotions, whereas the ERBS Hijack subscale measures belief that strong emotions can take over and make a person lose control over his or her actions. Participants were grouped by explanation condition and assumption checks were carried out on ERBS Regulation Worth (skewness = -.60 and kurtosis = -.05 in the fixed biological condition; skewness = -.27 and kurtosis = -.27 in the malleable condition) and on ERBS Hijack (skewness = .23 and kurtosis = -.13 in the fixed

biological condition; skewness = -.18 and kurtosis = 1.10 in the malleable condition). All values fell within acceptable limits (Kline, 2010). The variance ratios between the two conditions were 1.47 for ERBS Regulation Worth and 1.70 for ERBS Hijack, indicating sufficient equality of variance (Field, 2013).

Means and standard deviations for each explanation condition are presented in Table 4. For ERBS Regulation Worth results were not significant, $t(1, 137) = -1.26$, $p = .21$, Cohen's $d = .21$. However, the two conditions did differ significantly on the ERBS Hijack subscale, $t(1, 137) = -2.18$, $p = .03$, Cohen's $d = .37$. Participants who heard the malleable explanation for depression reported stronger emotional hijack beliefs than did participants who heard the fixed biological explanation.

Credulity and ERBS Subscales

To examine whether or not participants' credulity with regard to the study altered the impact of causal explanations for depression on beliefs about emotion and its regulation, a series of 2 x 2 ANOVAs were carried out with explanation condition (fixed biological or malleable) and response to the binary doubts question (yes or no) as IVs and each of the ERBS subscales, in turn, as the DV. For the ERBS Emotional Constraint subscale, the main effect of causal explanation approached significance, $F(1, 135) = 3.59$, $p = .06$, partial $\eta^2 = .03$, with participants who viewed the malleable explanation for depression endorsing stronger emotional constraint beliefs than participants who viewed the fixed biological explanation (see Table 11 for means and *SD*s). In addition, the main effect of doubts was significant, $F(1, 135) = 4.54$, $p = .04$, partial $\eta^2 = .03$, with participants who denied any doubts about the study endorsing stronger emotional constraint beliefs than those who reported having doubts.

For the ERBS Regulation Worth subscale neither the main effect of causal explanation, $F(1, 135) = 1.30$, $p = .26$, partial $\eta^2 = .01$, nor the main effect of doubts, $F(1, 135) = 0.25$, $p = .06$, partial $\eta^2 < .01$, reached significance. The 2-way interaction was also non-significant, $F(1, 135) = 0.72$, $p = .40$, partial $\eta^2 = .01$.

For the ERBS Hijack subscale, the main effect of causal explanation was significant, $F(1, 135) = 4.51$, $p = .04$, partial $\eta^2 = .03$, with participants who viewed the malleable explanation for depression endorsing stronger hijack beliefs than participants who viewed the fixed biological explanation (see Table 11 for means and *SD*s). Neither the main effect of doubts, $F(1, 135) = 2.63$, $p = .12$, partial $\eta^2 = .02$, nor the 2-way interaction, $F(1, 135) = 0.42$, $p = .52$, partial $\eta^2 < .01$, were significant. Thus, the addition of the binary doubts variable as a factor in analyses testing the effects of causal explanation on ERBS subscales yielded results consistent with those that emerged before the doubts variable was included.

Causal Explanation and Depression Mind Set

Next, we tested the impact of causal explanation condition on participants' mind set regarding depression. Higher scores on the Depression Mind Set scale indicate stronger entity beliefs—that is, a belief that people have a set "amount" of depression that cannot be changed.

Skewness (.737 in the fixed biological condition and 1.22 in the malleable condition) and kurtosis (.552 in the fixed biological condition and 1.42 in the malleable condition) fell within acceptable limits (Kline, 2010). The variance ratio was 1.02, indicating equality of variance (Field, 2013). Means and standard deviations are presented in Table 4. An independent samples *t*-test was conducted with causal

explanation as the IV and Depression Mind Set as the DV. Results were non-significant, $t(1, 137) = 1.70$, $p = .09$, Cohen's $d = .29$.

Credulity and Depression Mind Set

A 2 x 2 ANOVA was conducted with causal explanation (fixed or malleable) and response to the binary doubts question (yes or no) as IVs and the Depression Mind Set scale as the DV. Results showed a significant main effect of doubts, $F(1, 135) = 11.59$, $p < .01$, partial $\eta^2 = .08$, with participants who had no doubts about the study endorsing stronger depression entity beliefs than those who reported having doubts. Neither the main effect of causal explanation, $F(1, 135) = 2.30$, $p = .13$, partial $\eta^2 = .02$, nor the 2-way interaction, $F(1, 135) = 0.94$, $p = .33$, partial $\eta^2 < .01$, reached significance. Means and *SD*s are presented in Table 11.

Correlations within Each Explanation Condition

In order to better understand the relationships among variables within each causal explanation condition, we computed separate bivariate correlation matrices for the fixed biological explanation and malleable explanation groups. Results are presented in Table 12. In both the fixed biological and malleable explanation groups, higher levels of current depressive symptoms were associated with stronger depression entity beliefs as measured by the Depression Mind Set scale, $r(65) = .36$, $p < .01$, and $r(70) = .29$, $p = .01$, respectively, as well as poorer perceived prognosis $r(65) = .25$, $p = .04$ and $r(70) = .32$, $p = .01$, respectively. Both groups also evidenced positive associations between ERBS Emotional Constraint and ERBS Hijack beliefs, and between credibility and expectancy ratings for antidepressants and psychotherapy, as measured by the two CEQ scales.

Table 12

Bivariate Correlations between Variables within Each Causal Explanation Condition

Variable	1	2	3	4	5	6	7	8	9	10
1. Treatment willingness	-	.20	.23	.10	.20	.09	.28*	.20	-.02	.31*
2. Depressive symptoms	.38**	-	.36**	.25*	.23	.01	.15	-.10	-.02	.28*
3. Depression mind set	.05	.29*	-	.33**	.50**	-.28*	.49**	-.18	-.26*	.36**
4. Prognostic pessimism	.07	.32*	.06	-	.10	.10	.16	-.17	-.03	.14
5. Emotional Constraint	-.06	.09	.16	.14	-	.10	.56**	.04	.04	.25*
6. Regulation Worth	.07	.06	.10	.04	.21	-	.14	.24	.10	-.03
7. Hijack	-.16	.12	.20	.04	.59**	.40**	-	.001	-.11	.08
8. CEQ antidepressant	.22	.01	-.14	.01	.05	.26*	.02	-	.35**	.09
9. CEQ psychotherapy	.18	-.05	-.08	.11	-.06	.08	-.01	.29*	-	.17
10. Doubts	.01	.13	.20	-.15	.10	.13	.19	.11	.07	-

Note. Pearson's correlations within the fixed biological explanation group (n = 67) appear above the diagonal, and those within the malleable explanation group (n = 72) appear below the diagonal; Treatment willingness = willingness to accept initial treatment referral; Depressive symptoms = Quick Inventory of Depressive Symptoms - Self Report, sans item 12; Emotional Constraint = Emotion and Regulation Beliefs Scale, Emotional Constraint subscale; Regulation Worth = Emotion and Regulation Beliefs Scale, Regulation Worth subscale; Hijack = Emotion and Regulation Beliefs Scale, Hijack subscale; 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?"
*p < .05, **p < .01

In the fixed biological group, stronger entity beliefs about depression were associated with poorer perceived prognosis, $r(65) = .33$, $p = .01$, and lower credibility and expectancy ratings for psychotherapy, $r(65) = -.26$, $p = .04$. These variables were not significantly related within the malleable group. Within the malleable group ERBS Hijack was positively associated with ERBS Regulation Worth, $r(70) = .40$, $p < .001$. These variables were not significantly related within the fixed biological group.

In addition, the two groups exhibited differential relationships between depression entity beliefs and the ERBS subscales. Among participants who heard the fixed biological explanation ($n = 67$), depression entity beliefs were negatively associated with ERBS Regulation Worth, $r(65) = -.28$, $p = .02$, and positively associated with ERBS Emotional Constraint, $r(65) = .50$, $p < .001$, and ERBS Hijack, $r(65) = .49$, $p < .001$. Conversely, among participants who heard the malleable explanation ($n = 72$) depression entity beliefs were not significantly related to ERBS Regulation Worth, $r(70) = .10$, $p = .39$, ERBS Emotional Constraint, $r(70) = .16$, $p = .19$, or ERBS Hijack, $r(70) = .20$, $p = .10$. The difference between correlations when comparing them across the two explanation conditions was statistically significant as determined using Fisher's *Z*-transformation for ERBS Regulation Worth $Z = -2.24$, $p = .03$, ERBS Emotional Constraint $Z = 2.24$, $p = .03$, and ERBS Hijack, $Z = 1.92$, $p = .05$.

When participants were grouped according to both referral type and causal explanation condition (i.e., into four cells), differential relationships emerged between willingness to accept treatment and other variables measured (Table 13). In both cells of participants referred for antidepressant medication, CEQ antidepressant scores were positively related to willingness to accept treatment (both *r*'s = .32). Because these two

cells independently showed medium-sized effects in the same direction, the effect was examined at the level of referral type combining both causal explanation conditions. In this case the relationship between CEQ antidepressant scores and treatment willingness was significant for participants who received medication referrals, $r(63) = .32$, $p = .01$. In both cells of participants given the fixed biological explanation for depression, ERBS Hijack was positively associated with willingness to accept treatment (both rs = .28). When the effect was examined at the level of explanation condition by combining both referral types, the resulting relationship between ERBS Hijack and treatment willingness was significant among participants who heard the fixed biological explanation, $r(65) = .28$, $p = .02$.

Table 13

Bivariate Correlations with Treatment Willingness within Each Combination of Causal Explanation and Referral Condition

	Fixed Biological Explanation				Malleable Explanation			
Referral type	Medication (n = 34)		Psychotherapy (n = 33)		Medication (n = 31)		Psychotherapy (n = 41)	
	Association with treatment willingness							
Variable	r	p	r	p	r	p	r	p
Depressive symptoms	.03	.87	.39	.03	.53	<.01	.23	.16
Depression mind set	.34	.05	.11	.54	.17	.37	-.08	.64
Prognostic pessimism	.13	.46	.06	.74	-.07	.72	.16	.33
Emotional Constraint	.15	.40	.23	.20	-.22	.23	-.03	.83
Regulation Worth	-.01	.98	.18	.31	-.06	.73	.06	.72
Hijack	.28	.11**	.28	.11**	-.22	.24	-.22	.16
CEQ antidepressant	.32	.06*	.01	.94	.32	.08*	.13	.42
CEQ psychotherapy	-.02	.90	-.01	.98	.18	.35	.15	.35
Doubts	.15	.39	.48	.01	.30	.10	-.20	.20

Note. Treatment willingness = willingness to accept initial treatment referral; Depressive symptoms = Quick Inventory of Depressive Symptoms - Self Report, sans item 12; Emotional Constraint = Emotion and Regulation Beliefs Scale, Emotional Constraint subscale; Regulation Worth = Emotion and Regulation Beliefs Scale, Regulation Worth subscale; Hijack = Emotion and Regulation Beliefs Scale, Hijack subscale; 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?"

* Correlation between CEQ antidepressant and treatment willingness when combining across explanation conditions is $r(63)$ = .32, p = .01, for participants who received medication referrals.

** Correlation between ERBS Hijack scores and treatment willingness when combining across referral types is $r(65)$ = .28, p = .02, for participants given the fixed biological explanation.

CHAPTER IV

DISCUSSION

Explanations for Depression, Elaboration Likelihood, and Treatment Willingness

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 study and the depression screening test 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 may partially account for the observed interaction effect on willingness to accept treatment. However, this explanation alone is insufficient, given the size of the interaction effect and the fact that the 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 that this effect only occurred in participants who reported having doubts about the study.

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. The rationale for this interpretation draws upon the elaboration likelihood model (ELM; Petty, Brinol, & Priester, 2009; Petty & Cacioppo, 1986). The ELM describes two potential ways in which one might process and evaluate information: either actively through effortful scrutiny and elaboration on the content of the message (the central route), or passively by applying pre-existing heuristics activated by peripheral or contextual features of the message (the peripheral route).

Participants were told that, based on their performance on a brief reaction time test, they had been identified as being at very high risk for developing depression and should therefore seek treatment immediately, so as to avoid the onset of a full depressive episode. This cover story about the depression screening test may have been inconsistent with participants' prior knowledge and beliefs about how depression and other psychological disorders are assessed and diagnosed. In addition, the bogus feedback provided—that the test results show very high risk for depression—was likely inconsistent with many participants' recent subjective experiences, given that the sample was not preselected for elevated depressive symptoms. Thus, if participants evaluated the cover story and bogus feedback by actively scrutinizing the information presented and

comparing it to their prior knowledge from other sources, then it would arguably be both reasonable and likely for them to have doubts about some part of the study.

Alternatively, participants who processed the cover story and bogus feedback via the peripheral route may have relied on contextual features such as how polished the speaker appeared to be, accompanying visual cues, or the setting in which the information was presented in order to decide whether to accept the message, instead of actively considering the merits of the information itself (Petty et al., 2009; Petty & Cacioppo, 1986). As Petty and colleagues (2009) state, "the source of a message can trigger a relatively simple inference or heuristic such as 'experts are correct' that a person can use to judge the message." Indeed, the video clips used in the present study to convey the cover story and information about depression were designed to include peripheral cues likely to enhance their persuasiveness. For example, the actor appearing in these videos wore professional attire, identified himself as "a researcher and clinician," and described the depression screening test as a tool being developed by "researchers at Mississippi State University." If participants 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, then they might have passively accepted the messages at face value without identifying any doubts about the study.

It is important here to make a distinction between perceived accuracy or validity of the depression screening test and bogus personalized feedback, and perceived accuracy or validity of the information presented about depression in general. Because none of the participants suspected that the study involved deception, it is highly likely that they accepted the general information about the causes of depression even if they doubted that

depression risk could be detected by a brief reaction time test or disagreed with the feedback about their personal level of depression risk. Indeed, among participants who reported doubts about the study, those given the fixed biological explanation still exhibited *significant differences in the predicted directions* from those given the malleable explanation, when asked to rate the likelihood that various factors could cause depressed mood.

If the participants who were able to identify and verbalize doubts about the study were indeed more actively examining and making inferences about the information presented throughout the study, and if they accepted the general information about the causes of depression as valid, then this difference in processing style could potentially account for the role of doubts as a moderator of the interactive effect of causal explanation and referral type on treatment willingness. Participants who viewed the fixed biological explanation for depression were presented with conflicting messages: (a) depression is caused by genetic and physical features that are out of a person's control and can only be altered with medication, and (b) the participant should make an appointment to begin psychotherapy, which will provide behavioral and cognitive strategies aimed at altering depression risk. 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.

However, both of the above messages came from the exact same source and were presented in the exact same context. Therefore, a participant who passively processed the messages might have simply accepted both of them on the grounds that they came from a trustworthy or knowledgeable source, and might not have checked them for logical consistency. Thus, this participant would be just as willing to engage in psychotherapy as a treatment for depression as they would have been if they had viewed the malleable explanation instead. Indeed, this is what we found in the present study. Among credulous participants we saw no difference in treatment willingness when comparing the fixed biological and malleable explanation groups who received psychotherapy referrals.

In summary, some people might engage in active scrutiny and elaboration of messages they hear about mental illness, risk factors, or treatment whereas others might passively accept such messages on the basis of peripheral features such as perceived trustworthiness of the source. Reporting doubts about the study may have been an indicator that participants engaged in effortful processing via the central route. In the present study, effortful thought about the content and implications of causal explanations for depression might have been necessary in order for the interactive effects on treatment willingness to emerge. This would explain why we only found the interaction among participants who reported doubts about the study.

The literature on dual-processing models such as the ELM identifies a range of factors that 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 situation (Heesacker, Petty, & Cacioppo, 1983; Petty et al., 2009).

Need for cognition refers to a preference for effortful cognitive engagement across a range of situations (Cacioppo & Petty, 1982). Individuals high in need for cognition tend to evaluate messages based on active scrutiny of their informational content even when the source of the message is perceived to be credible (Priester & Petty, 1995). Such individuals also tend to be more persistent in their beliefs and skeptical of messages aimed at persuading them to think differently (Haugvedt & Petty, 1992). Participants high in need for cognition would likely have been intrinsically motivated to scrutinize and cognitively elaborate on the information about depression that was provided as part of the present study. In contrast, individuals low in need for cognition try to expend the minimum cognitive effort they deem necessary to reach an acceptable conclusion, and are therefore less likely to scrutinize a message when the source is believed to be honest and knowledgeable (Priester & Petty, 1995). They can, however, be prompted to engage in more effortful scrutiny of messages when situational cues indicate that the information source is not credible.

In the present study it is possible that the provision of inaccurate feedback about depression risk prompted effortful processing. Nonetheless, the observed interaction effect cannot be explained as an experimental artifact, because all of the participants saw the same bogus feedback. The majority of 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 factors other than the experimental cover story played a role in determining whether or not participants engaged in active scrutiny of the

information that was presented, and that such scrutiny is a novel determinant of future action in combination with other previously established factors.

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 credulity of depression assessments.

Factors Correlated with Treatment Beliefs and Willingness to Accept Treatment

In addition to the findings already discussed, the present study shed light on the different factors associated with treatment beliefs and willingness to accept treatment for participants at each level of the independent variables—causal explanation and referral type. First, we found that across both causal explanation conditions participants were more willing to accept referrals for psychotherapy than for antidepressant medication. This effect held up when controlling for current depressive symptoms, as well as when the binary doubts variable was included, though it was reduced somewhat when participants' treatment history was taken into account. Surprisingly, among participants referred for psychotherapy there was no relationship between perceived credibility and effectiveness of psychotherapy and willingness to accept treatment, in either causal

explanation condition. Thus, either participants were not able to accurately report their beliefs about psychotherapy, or their decisions about whether to accept a referral for psychotherapy were not based on explicit beliefs about psychotherapy's effectiveness for treating depression.

In contrast, perceptions of the credibility and effectiveness of antidepressant medication for treating depression were related to willingness to accept treatment among those participants who received medication referrals, across both causal explanation conditions. This finding is notable given that participants in the malleable explanation condition rated antidepressants as significantly less credible and effective than psychotherapy for treating depression. Further, examination of the marginal means showed that 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. Taken together, these findings suggest that providing information about the malleability of biological risk factors for depression may reduce the perceived credibility and effectiveness of antidepressant medication, which in turn may make individuals less willing to accept this form of treatment. We will return to this topic later in the discussion.

Interestingly, for participants who viewed the fixed biological explanation for depression and received a referral for antidepressant medication, current depressive symptoms showed no relationship to willingness to accept treatment. Instead, treatment willingness for this group was moderately positively associated with perceived credibility and effectiveness of antidepressant medication (as already noted), belief that depression is a fixed entity that one cannot alter, and belief in emotional hijack or the power of

strong emotions to take over and cause a person to lose control. This constellation of findings may have implications for individuals who are given a fixed biological causal explanation for depression and who believe themselves to be at elevated risk of becoming depressed—for example, due to a family history of the disorder or having previously experienced a depressive episode themselves. The present study seems to suggest that these individuals may base decisions about taking antidepressant drugs not on the presence of depressive symptoms at the time of the decision, but on a sense of fatalism about their ability to improve depressed moods or regulate their emotions in general, as well as faith in the effectiveness of antidepressant medication.

In contrast, for participants who saw the fixed biological explanation and then received a referral for psychotherapy, current depressive symptoms showed a medium-sized positive relationship with willingness to accept treatment. Again, treatment willingness was also moderately positively associated with emotional hijack beliefs.

Among participants who viewed the malleable explanation for depression and received a referral for medication, current depressive symptoms showed a large positive relationship with willingness to accept treatment, and perceived credibility and expectancy of antidepressant medication was moderately positively associated with treatment willingness. This seems to suggest that individuals who believe themselves to be at risk for developing depression but who are given information about the malleability of biological risk factors base their decisions about whether or not to take antidepressant medication on their actual level of depressive symptoms as well as their faith in the effectiveness of medication as a means of decreasing those symptoms.

Of all four groups, the participants who viewed the malleable explanation for depression and received a referral for psychotherapy proved most difficult to characterize with regard to factors potentially affecting treatment willingness. None of the variables measured in the present study, including current depressive symptoms, were significantly related to willingness to accept treatment.

Causal Explanations for Depression and Beliefs about Emotions

At the group level, participants given a malleable explanation for depression reported greater belief in the power of strong emotions to take over and cause people to lose control of their thoughts and actions. This effect remained significant when the binary doubts variable was included in the analysis. Moreover, with the doubts variable included as a factor, the effect of causal explanations on emotional constraint beliefs also approached significance; again, participants given the malleable explanation for depression endorsed stronger emotional constraint beliefs than those in the fixed biological explanation.

It was initially surprising to see *stronger* emotional constraint and hijack beliefs among participants who were told that emotional processing styles could be altered. However, the direction of these effects may indicate that at a group level, the malleable explanation produced a stronger conceptual link between depression and strong emotions than the fixed biological explanation, thereby conveying the sense that emotions are powerful and capable of constraining and even derailing healthy functioning.

The group-level effects of causal explanation on emotional constraint and hijack beliefs were qualified by the pattern of correlations observed within each explanation condition. Specifically, among participants given the malleable explanation for

depression, beliefs about emotion and its regulation were unrelated to beliefs about depression and the extent to which depressive symptoms can be changed. Moreover, participants in the malleable condition who endorsed stronger emotional hijack beliefs also viewed emotion regulation as more worthwhile and attainable, which differs from previous research with the ERBS wherein hijack beliefs and regulation worth were found to be unrelated (Veilleux et al., 2015).

Conversely, in the fixed biological explanation condition, entity beliefs about depression and about emotions in general tended to cohere. For individuals in this condition, there were strong positive relationships between depression entity beliefs and the beliefs that emotions constrain and hijack a person's functioning. There was also a medium-sized negative relationship between depression entity beliefs and regulation worth, suggesting that persons who hear a fixed biological causal explanation for depression and believe that depression is a fixed feature of individuals that cannot be changed will also tend to see emotion regulation as less attainable and less worthwhile.

The present study demonstrates a link between beliefs about the causes of depression and beliefs about the power and malleability of emotional experiences in general, and shows that experimentally manipulating the former produces effects in the latter. This link is important, as it suggests that messages about the causes and nature of mental illnesses likely impact a broader set of beliefs about one's psychological and emotional functioning.

Implications of the Present Study for Treatment of Depression

It is important to bear in mind the preliminary nature of the present findings, as many of the results discussed here arose from exploratory analyses. Additional studies

will be crucial to determine whether or not the pattern of results observed here replicates. It is with these caveats in mind that we consider the present findings and their potential implications with regard to the treatment of depression.

Implications for Treatment with Psychotherapy

One of the main goals in designing and conducting the present study was to examine whether holding a fixed biological explanation for depression would impact beliefs about or acceptance of psychotherapy. The results of this study did, in part, support our hypothesis, but the predicted effect was limited to individuals who expressed doubts about the study—perhaps because these individuals engaged in more effortful scrutiny and elaboration on information about the causes of depression, and therefore made inferences related to biological essentialism that might undermine willingness to participate in psychotherapy.

This constellation of findings, upon further replication and translation, suggests 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, 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—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, Petty, Feinstein, & Jarvis, 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, Hill, Hennessey, & Tighe, 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 be "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.

Implications for Treatment with Antidepressant Medication

We also found evidence suggesting that information about the malleability of biological risk factors for depression might reduce the perceived credibility and effectiveness of antidepressant medication as a treatment for the disorder, and that such

beliefs may play a role in decisions about whether or not to seek out or accept antidepressant medication. Two alternative interpretations of these findings arise, and conclusions about which interpretation is more plausible hinge on one's view of antidepressant medications. If one starts from the premise that antidepressant medications are an effective and useful means of treating depression, then the present findings seem to indicate that messages downplaying the role of genes and neurobiology in causing depression discourage the public from seeking out and benefitting from effective treatments. From this perspective, one might conclude that clinicians and researchers should promote a disease model of depression, and research findings that run counter to this message should be discussed with care.

Alternatively, one might start from the premise that antidepressant medications are no more effective than placebo for all but the most severely depressed individuals, and they often produce unwanted side effects. From this perspective, the present findings suggest that perceptions of antidepressant medication as a credible and effective treatment for depression depend at least in part on a belief that depression is caused by fixed biological factors, such as faulty genes producing a chemical imbalance in the brain. Therefore, clinicians and researchers should emphasize the malleability of biological risk factors for depression, so that individuals are less likely to take ineffective drugs that produce side effects on the basis of what is at best a vastly oversimplified idea about the causes of depressive symptoms.

The tension between these alternative viewpoints cannot be resolved solely by reference to the present set of findings. It is well beyond the scope of this study to determine the effectiveness or clinical utility of antidepressant medications as a treatment

for depression, nor can the present study determine the comparative validity of fixed biological or malleable models of depression. In any case, it is likely that both of the conclusions outlined above would be too simplistic, given the lack of clear group-level effects in participants referred for medication. Indeed, at the group level individuals who heard the malleable explanation for depression were no less willing to accept a medication referral than those who heard the fixed biological explanation—this was the case regardless of whether participants had doubts about the study. Rather, results from this study suggest that attributing depression to either fixed biological or malleable factors may impact a complex web of relationships among other beliefs about depression, emotion and its regulation, and treatment options.

It is possible that the act of seeking or accepting antidepressant medication may be motivated by different factors and hold different meanings depending upon how one conceptualizes depression. For instance, the present findings suggest that persons who attribute depression to fixed biological causes may be more likely to seek antidepressant medication to the extent that they believe individuals cannot alter depressed moods or regulate emotions on their own. Conversely, persons who believe that depression is caused by malleable factors may be more likely to seek antidepressant medication to the extent that they are actually experiencing depressive symptoms. If these findings prove to be replicable effects, then it will be meaningful to consider whether and how such differences color engagement with and experience of treatment, and ultimately the odds of recovery.

Strengths and Limitations

Use of a Student Population

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 25-32 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 prior to debriefing). 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.

Random Assignment to Treatment Option

In most previous studies examining relationships between causal explanations for depression and beliefs about treatment, effects on beliefs about psychotherapy and beliefs about antidepressant treatment were not measured separately. Therefore, those studies could not evaluate whether the effect of causal explanations for depression on treatment-related beliefs or behaviors is moderated by type of treatment offered. In the present study, random assignment of participants to both a causal explanation condition and a treatment referral condition allowed us to test for the hypothesized interaction. The design of the present study also allowed us to identify the different factors associated with treatment willingness within each combination of causal explanation and referral type.

Decision Not to Include a Control Group

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.

Use of Videos

Video clips were used to give participants the depression screening test results, to present causal explanations for depression, and to provide treatment referrals and pose follow-up questions. Use of pre-recorded videos, rather than scripts presented by experimenters in person, reduced the potential for experimenter error and minimized the chance of experimenters being asked questions that could have altered their interactions with participants during the experiment. Videos also guarded against the possibility of unintended effects owing to the gender, ethnicity, or other individual characteristics of experimenters. In sum, use of videos helped ensure that key aspects of the experiment were delivered in exactly the same way for all participants in a given condition.

Future Directions

Responses from the funnel debriefing questionnaire provided insight into potential ways of strengthening the IAT and cover story in future research. Although participants did take the cover story at face value and accepted that the IAT 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 improved by increasing the number of IAT 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 improved by expanding the explanation of how reaction time tasks can measure individual differences in the processing of emotional information, and how these differences relate to depression risk.

Many of the findings presented in this manuscript arose from exploratory analyses, and therefore follow-up studies will be crucial to determine whether or not the pattern of results observed here replicate, and to test the robustness of those 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. Additional research will be needed to more fully examine the role of effortful versus passive processing as a potential moderator of the relationship between causal explanations for depression and treatment willingness. Further, the literature of cognitive 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 also be needed to assess which factors are most relevant in determining how individuals evaluate messages about the causes of depression.

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. Follow-up studies could also attempt to manipulate beliefs about the credibility and effectiveness of antidepressant medication more directly to see whether changes in these beliefs do indeed impact willingness to accept treatment. 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.

Although examining willingness to accept treatment provided a useful starting point for our research, the present findings suggest that this metric may not capture the full impact of beliefs about the causes of depression. Rather than directly altering treatment willingness, it seems that different causal explanations may lead individuals to seek treatment for different reasons, and may shape their beliefs about the malleability of depressive symptoms and emotions in general. Thus, future studies will be needed to examine whether and how such differences impact engagement with treatment, the ways

in which individuals experience and interpret changes in symptom severity, and ultimately the odds of recovery.

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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APPENDIX A

FUNNEL DEBRIEFING QUESTIONNAIRE PRESENTED VIA COMPUTER

We are interested in assessing participants' perceptions of the study. We want to make sure that our design is sound, and we need your feedback to help us improve this study. Please be as honest as possible in your answers; no feedback we receive, including negative feedback, will result in a loss of research credit. In fact, negative feedback is an important way for us to improve upon our design for future studies. Be as detailed as you feel is necessary to fully answer each question. You may spend as much time on these questions as you want, but we ask that you spend a minimum of 5 min answering these questions.

In your own words, what was the present study about?

Please tell us your opinion of the depression screening test (the computer test that asked you to match words) and your results from the test:

In your opinion, how accurate was the information about depression that was presented?

What do you think was the purpose of the questionnaires you completed and the questions you were asked in the video clips?

Did you have any doubts about any part of the study?

If so, please describe what you thought:

What part of the study made you feel doubtful?

APPENDIX B

DEBRIEFING SCRIPT PRESENTED VIA VIDEO

The true purpose of this study was to test how different messages about the causes of depression affect a person's willingness to accept treatment. Research shows that willingness to get treatment, beliefs about treatment, and expectations for the future significantly affect depressive symptoms and recovery from depression. Therefore, it is very important to understand whether and how messages about the causes of depression influence these factors. Ultimately, this study and others like it can help doctors, psychologists, and counselors understand the best ways of communicating with people about depression, so as to encourage depressed individuals to get treatment and to develop positive expectations about the future.

In order to examine our research question, we needed to tell participants a believable cover story to explain why we would be offering them treatment for depression, and to give them a reason to consider accepting treatment. So, we told you that the computer task you completed was a depression screening test. We told you that we had calculated your score, and that your score showed high risk for depression. In reality, the computer task you completed does measure one aspect of processing of emotional information, but we did not look at your performance on the task at all. Your score has not been calculated, so we have no idea how accurate your responses were. In fact, researchers will not calculate any participant's scores on the task until data collection for this study is finished, which may not be for several months.

In the video that you watched after finishing the computer task, this graph was shown *(graph of bogus test score is displayed again)* and you were told that the numbers on the graph represent your personal score on the computer task. Actually, the scores presented in this graph were totally unrelated to any aspect of your actual performance, or

any other personal characteristic. Every participant in this study is shown the same graph depicting the same results, and every participant is told that their score means they are at high risk for depression.

Next, the video presented information about the causes of depression. This information was intentionally selected to communicate a certain type of cause, and therefore it emphasized some findings from depression research while downplaying or leaving out other findings.

In reality, current research suggests that depression is caused by a combination of genetic, biological, environmental, social, and psychological factors. The exact nature of these causes and the ways in which they interact are not yet fully understood, but risk factors include:

- Personal or family history of depression
- Major life changes, trauma, or stress
- Certain physical illnesses and medications

Depression, even severe cases, can be treated. The earlier that treatment can begin, the more effective it is. Depression is usually treated with medications, psychotherapy, or a combination of the two.

After you saw the video about the causes of depression, you were asked about your beliefs about treatments and coping strategies and your beliefs about how long depression lasts. You also responded to a series of video clips that asked about your willingness to get treatment today or in the future. Again, the purpose of using deception in today's study—for example, telling you that you were at high risk for depression—was to provide a believable cover story to explain why we would be offering treatment for

depression, and to give you a reason to consider treatment. The cover story allowed us to present you with certain information about the causes of depression and then measure your willingness to accept treatment.

Now that you understand the true nature of the study, you have the chance to decline permission for the data that we collected from you to be used for research purposes. After this video, your experimenter will return and present you with the real informed consent form. You are free to ask us not to use your data in our study analysis. If you decline to let us use your data, you will still receive course credit, just as you would if we use your data in our analysis. This is entirely voluntary, but we hope to analyze as much data as possible to better understand the effects of beliefs about the causes of depression on decisions about treatment to eventually help individuals with depression.

In any case, we respectfully request that you do not talk to any other MSU students about this study. If future participants find out the details of the study in advance then our cover story will not be convincing to them, and the responses they provide will not be valid. If this happens, then our study data could lead us to draw the wrong conclusions about how best to help people get treatment for depression.

Finally, the researchers of this study want to thank you for participating. You have made a valuable contribution to this important research. Ultimately, this study and others like it can help doctors, psychologists, and counselors to explain depression in a way that encourages depressed individuals to get treatment, and treatment for depression can save lives.

APPENDIX C

MEASURES

Note: Items measuring prognostic pessimism, beliefs about causes of depression, and willingness to accept treatment are included in full within the Measures section in the body of this document.

Emotion Regulation and Beliefs Scale (ERBS)

1. Emotions operate like a floodgate that is either open or closed. In other words, emotions are either "on" or "off."

   1 2 3 4 5

   Strongly Disagree Strongly Agree

2. Emotions can either be expressed entirely or hidden from others—it isn't possible to share only part of an emotional response.

   1 2 3 4 5

   Strongly Disagree Strongly Agree

3. People can learn to control/regulate their emotions.

   1 2 3 4 5

   Strongly Disagree Strongly Agree

4. People are ruled by their emotions.

1	2	3	4	5
Strongly Disagree				Strongly Agree

5. Putting forth effort to alter emotional experience is valuable.

1	2	3	4	5
Strongly Disagree				Strongly Agree

6. When a person has a strong emotional reaction to another person, they will always feel that way about that other person.

1	2	3	4	5
Strongly Disagree				Strongly Agree

7. When people are feeling down, they have to wait for a better mood to arrive before they can be productive.

1	2	3	4	5
Strongly Disagree				Strongly Agree

8. People would be better off if they took time to figure out where their emotions come from.

1	2	3	4	5
Strongly Disagree				Strongly Agree

9. When strong emotions are present, they dictate what a person says or does.

1	2	3	4	5
Strongly Disagree				Strongly Agree

10. When an emotion comes along, it will continue unless there is a change in the environment.

1	2	3	4	5
Strongly Disagree				Strongly Agree

11. When people acknowledge their emotions, the emotions will completely take them over.

1	2	3	4	5
Strongly Disagree				Strongly Agree

12. Learning how to alter strong emotions is a worthwhile pursuit.

1	2	3	4	5
Strongly Disagree				Strongly Agree

13. It is possible, with effort, to alter strong feelings in any situation.

1	2	3	4	5
Strongly Disagree				Strongly Agree

14. When a person feels really angry, it's virtually impossible to not take the anger out on people or objects nearby.

1	2	3	4	5
Strongly Disagree				Strongly Agree

15. People are slaves to their emotions.

1	2	3	4	5
Strongly Disagree				Strongly Agree

16. People would be better off if they spent more time learning how to control their emotions.

1	2	3	4	5
Strongly Disagree				Strongly Agree

17. Strong emotions will make people do things they wouldn't normally do.

1	2	3	4	5
Strongly Disagree				Strongly Agree

18. When feelings of sadness take over, a person can't really do anything but wallow in the misery.

1	2	3	4	5
Strongly Disagree				Strongly Agree

19. People benefit from learning how to regulate their feelings.

1	2	3	4	5
Strongly Disagree				Strongly Agree

20. It's virtually impossible for people to act opposite to the way they feel.

1	2	3	4	5
Strongly Disagree				Strongly Agree

21. Emotions make people lose control.

1	2	3	4	5
Strongly Disagree				Strongly Agree

Credibility/Expectancy Questionnaire (CEQ)

Set I

1. At this point, how logical does the treatment described above seem?

1 2 3 4 5 6 7 8 9

not at all logical somewhat logical very logical

2. At this point, how useful do you think this treatment would be in reducing depressive symptoms?

1 2 3 4 5 6 7 8 9

not at all useful somewhat useful very useful

3. How confident would you be in recommending this treatment to a friend who experiences depressive symptoms?

1 2 3 4 5 6 7 8 9

not at all confident somewhat confident very confident

4. By the end of the treatment period, how much improvement in depressive symptoms do you think would occur?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

Set II

For this set, close your eyes for a few moments and try to identify what you really *feel* about the treatment and its likelihood of success. Then answer the following questions.

5. At this point, how much do you really *feel* that the treatment would help to reduce depressive symptoms?

1 2 3 4 5 6 7 8 9

not at all somewhat very much

6. By the end of the treatment period, how much improvement in depressive symptoms do you really *feel* would occur?

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

Quick Inventory of Depressive Symptomatology—Self-Report (QIDS-SR)

During the past seven days...

10. Concentration / Decision Making:

- ☐ 0 There is no change in my usual capacity to concentrate or make decisions.
- ☐ 1 I occasionally feel indecisive or find that my attention wanders.
- ☐ 2 Most of the time, I struggle to focus my attention or to make decisions.
- ☐ 3 I cannot concentrate well enough to read or cannot make even minor decisions.

11. View of Myself:

- ☐ 0 I see myself as equally worthwhile and deserving as other people.
- ☐ 1 I am more self-blaming than usual.
- ☐ 2 I largely believe that I cause problems for others.
- ☐ 3 I think almost constantly about major and minor defects in myself.

12. Thoughts of Death or Suicide:

- ☐ 0 I do not think of suicide or death.
- ☐ 1 I feel that life is empty or wonder if it's worth living.
- ☐ 2 I think of suicide or death several times a week for several minutes.
- ☐ 3 I think of suicide or death several times a day in some detail, or I have made specific plans for suicide or have actually tried to take my life.

13. General Interest

- ☐ 0 There is no change from usual in how interested I am in other people or activities.
- ☐ 1 I notice that I am less interested in people or activities.
- ☐ 2 I find I have interest in only one or two of my formerly pursued activities.
- ☐ 3 I have virtually no interest in formerly pursued activities.

During the past seven days...

14. Energy Level:

- ☐ 0 There is no change in my usual level of energy.
- ☐ 1 I get tired more easily than usual.
- ☐ 2 I have to make a big effort to start or finish my usual daily activities (for example, shopping, homework, cooking, or going to work).
- ☐ 3 I really cannot carry out most of my usual daily activities because I just don't have the energy.

15. Feeling Slowed Down:

- ☐ 0 I think, speak, and move at my usual rate of speed.
- ☐ 1 I find that my thinking is slowed down or my voice sounds dull or flat.
- ☐ 2 It takes me several seconds to respond to most questions and I'm sure my thinking is slowed.
- ☐ 3 I am often unable to respond to questions without extreme effort.

16. Feeling Restless:

- ☐ 0 I do not feel restless.
- ☐ 1 I'm often fidgety, wringing my hands, or need to shift how I am sitting.
- ☐ 2 I have impulses to move about and am quite restless.
- ☐ 3 At times, I am unable to stay seated and need to pace around.

APPENDIX D

IRB APPROVAL

From: jbr6@msstate.edu

Sent Date: Wednesday, March 01, 2017 07:30:02 AM

To: tms469@msstate.edu, abw206@msstate.edu, acs715@msstate.edu, ap1189@msstate.edu, cm998@msstate.edu, dgj44@msstate.edu, heh235@msstate.edu, jcs579@msstate.edu, jen144@msstate.edu, jkk99@msstate.edu, knm288@msstate.edu, mgd64@msstate.edu, mn487@msstate.edu, sw1388@msstate.edu, tcj95@msstate.edu, wdt85@msstate.edu

Cc:

Bcc:

Subject: IRB Protocol Approved: IRB-16-682, Taban Salem

Message:

IRB has approved the protocol with the following details.

Protocol ID: IRB-16-682
Principal Investigator: Taban Salem
Department: Psychology
Protocol Title: Computerized Psychoeducational Feedback and Response to Referral
Review Type: EXPEDITED
Approval Date: March 01, 2017
Expiration Date:February 15, 2018

To access your approval documents, log into myProtocol and click on the protocol number to open the approved study. Your official approval letter can be found under the Event History section. For non-exempt approved studies, all stamped documents (e.g., consent, recruitment) can be found in the Attachment section and are labeled accordingly.

If you have any questions that the HRPP can assist you in answering, please do not hesitate to contact us at irb@research.msstate.edu or 662.325.3994.