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Rebuilding trust in psychotherapy for biologically explained depression: A two-part experimental study examining the durability of an online psychoeducational intervention☆

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ABSTRACT

Biological explanations of mental disorders, which are gaining prominence, can decrease trust in psychotherapy. To rebuild trust, this experimental study tests a psychoeducational intervention targeting misconceptions that (1) psychotherapy cannot change the brain; (2) people rarely have agency over biology while psychotherapy requires agency; (3) psychosocial causes, addressed in psychotherapy, are less probable given biological causes. U. S. adults ($N = 602$) rated psychotherapy's effectiveness for depression before and after learning about depression's biological causes. Absent any intervention, control-condition participants rated psychotherapy to be less effective post biological-causes-information. However, participants who viewed an intervention video explaining why the misconceptions are flawed judged psychotherapy as more effective even after learning about depression's biological causes. Active-control-condition participants, who viewed a video about psychotherapy's effectiveness, without directly addressing the misconceptions, also increased psychotherapy ratings, albeit significantly less than the intervention group. Approximately four weeks later, intervention-condition participants maintained their enhanced trust, without any reminder of the video, whereas the two control conditions showed reduced trust. The study offers a practical tool for broader public use with a lasting effect.

Biological explanations of mental disorders, which have become prevalent (Pescosolido et al., 2021), can lead to diminished trust in psychotherapy. Both laypeople (Iselin and Addis, 2003; Kemp et al., 2014; Schroder et al., 2020) and mental health clinicians (Ahn et al., 2009; Perricone et al., 2023) view psychotherapy as less effective when mental disorders are attributed to biological factors. This diminished trust is troubling given that combined treatment with pharmacotherapy and psychotherapy is considered optimal for most mental disorders (Huhn et al., 2014). Thus, if individuals attribute their symptoms to biology, they may resist psychotherapy or have lower treatment expectations, worsening treatment outcomes (Constantino et al., 2018). This experimental study aims to develop and test an enduring intervention to restore trust in psychotherapy's effectiveness for biologically attributed mental disorders.

1. Background

The first order of business is to understand reasons for the reduced trust. Perricone and Ahn (2023c) identified three misconceptions about psychotherapy among a U.S. population, as follows.

Neurodualism. According to classic mind-body dualism (Descartes, 2013/1641), people perceive the mind and the brain as occupying separate domains. Accordingly, people believe that psychosocial causes of mental disorders can be treated solely with psychotherapy, targeting mental processes, while biological causes are treated solely with medication, acting on the brain. Nowadays, however, people understand that the materialistic brain and medications alter mental processes. Nonetheless, people are still reluctant to accept that mental processes, such as those taking place during psychotherapy, change the brain, a belief termed "neurodualism" (Valtonen et al., 2021). Indeed, the extent of endorsing neurodualism was associated with the extent to which people distrust psychotherapy for biologically attributed problems (Perricone and Ahn, 2023c).

Biological Uncontrollability. People view biological processes underlying mental disorders as largely beyond human control (Berent and Platt, 2021; Dar-Nimrod and Heine, 2011). But, psychotherapy

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demands individual effort and agency (e.g., collaborative agenda setting; completing homework; Levitt et al., 2016), therefore making it seem incompatible with treating biological processes. Indeed, the larger the gap people perceived in the controllability of psychotherapy and biological processes, the more people distrusted psychotherapy for biologically explained depression (Perricone and Ahn, 2023c).

Causal Discounting. When people view multiple causes as individually sufficient to produce an outcome (e.g., alcohol use and work stress for insomnia), they discount the likelihood of other causes (e.g., alcohol use) once one cause is discovered (e.g., work stress) (Kelley and Michela, 1980). Even clinicians discount the plausibility of psychosocial causes given biological causes (Ahn et al., 2009). Discounting conflicts with the widely accepted biopsychosocial model (Bolton and Gillett, 2019), and skews treatment choices, as psychotherapy is believed to be for psychological causes. In Perricone and Ahn (2023c), the more participants discounted psychosocial causes when depression was biologically attributed, the more they distrusted psychotherapy.

Recently, studies showed that these misconceptions can be intervened on. When participants learned how psychotherapy changes interactions among brain regions (Perricone et al., 2023), how skills learned during psychotherapy help people gain some control over biological processes (Perricone and Ahn, 2023b), or how biological and psychosocial causes synergistically interplay (Perricone and Ahn, 2023a), they showed significantly greater trust in psychotherapy for biologically attributed depression.

However, there are two crucial gaps to be addressed before publicly disseminating these interventions. First, no single intervention addressing all three misconceptions has been developed and its success is uncertain because of the sheer complexity of the constructs, ranging across philosophy, biology, and psychiatry. Second, no previous studies tested the interventions' durability, leaving their real-life applicability unclear.

2. Study aims and overview

This study aims to develop a *single* intervention useable for the public with long-lasting effects. The new intervention explains that 1) psychotherapy changes the brain, 2) people have agency over biological processes, and 3) biological and psychosocial causes are co-dependent. Addressing these beliefs in tandem may be viable, as one explanation can exploit others (e.g., explaining the interplay between biological and psychosocial causes requires addressing neurodualism).

To overcome the explanations' complexity and enhance their durability, this intervention used a video format, similar to brief YouTube lectures familiar to wider audiences (see https://www.youtube.com/watch?v=XUzF-xI3c9M for the video, and Supplement for the text).

The combined intervention was tested across two timepoints. At T1, participants rated the effectiveness of psychotherapy for a woman with depression (pretest), and again after learning about the biological causes of her depression (post-test). Between learning about biological causes and the post-test, intervention-condition-participants viewed the aforementioned video. Active-control-condition-participants viewed a video that described the effectiveness of psychotherapy for depression, controllability of depression symptoms, and the multiple causes of depression, without addressing the three misconceptions. Control-condition-participants viewed no video. Approximately four weeks later (T2), participants received the same T1 materials describing the woman's symptoms and the biological causes, and rated the effectiveness of psychotherapy for her. Intervention-condition- and active-control-condition-participants received no reminders about the previous videos.

We hypothesized that control-condition-participants would judge psychotherapy to be significantly less effective after learning about depression's biological causes. In contrast, intervention- and active-control-condition-participants would rate psychotherapy as significantly more effective at post-test, relative to baseline, because both videos underscore psychotherapy's benefits. However, intervention-participants would show a significantly greater increase than active-control-participants. Consequently, post-test ratings would be highest in the intervention condition. If the intervention effect is long-lasting, the pattern of results across the three conditions at T2 would remain similar to T1.

3. Methods

3.1. Participants

The target sample size at T1 was 600. Through the online recruitment site, Prolific, 603 U.S. participants were recruited at T1. (See the Supplement for sample size and recruitment details.)

After excluding one participant for failing an attention check, the final T1 sample was 602, 47.2% women; 18-79-years-old ($M_{age} = 41.90$, $SD = 13.81$); 43.5% with a bachelor's degree; 71.3% White; $M_{BDI\text{-}II} = 12.67$, $SD = 12.76$. There were no differences across the conditions in demographics, BDI-II-scores, history of depression, or current treatment with psychotherapy, all p's $> .118$.

Of these participants, 484 completed T2 (no condition differences in attrition, $p = .809$). The T2 sample was demographically similar to the T1 sample with 47.7% women; 19–79 years old ($M_{age} = 42.90$, $SD = 13.81$); 44% with a bachelor's degree; 71.3% White; $M_{BDI\text{-}II} = 11.69$, $SD = 12.34$. There were no differences across the conditions in terms of demographics, BDI-II scores, or history of depression (p's $> .284$), or current treatment with psychotherapy ($p = .087$).

3.2. Design, materials, and procedures

Study procedures (Fig. 1) were approved by the Yale University IRB. All procedures, described in full below, were administered using Qualtrics online survey software. The Supplement provides the verbatim stimuli and measures.

T1. Participants first read about a woman, "Alex" with Major Depressive Disorder symptoms (APA, 2015) and rated the effectiveness of antidepressants and psychotherapy for Alex, using the Credibility Expectancy Questionnaire (CEQ) (Devilly and Borkovec, 2000), which was modified to refer to Alex (Perricone and Ahn, 2023b) (e.g., "How much do you really feel that psychotherapy will help Alex to reduce her symptoms?"). The CEQ was the primary outcome measure throughout the study. Afterwards, participants learned about biological causes of Alex's depression ("A recent brain scan revealed some abnormalities in Alex's brain …") and answered six True/False questions on what they had read so far. All participants met the criteria pre-set for all attention checks (missing three-or-fewer questions on their second attempt).

The Qualtrics program randomly assigned participants to the control ($N = 204$), intervention ($N = 197$), or active control ($N = 201$) conditions. Intervention- and active-control-condition-participants viewed condition-specific videos embedded in the program. To increase engagement, participants were told that questions about the video would follow.

The intervention video, consisting of simple visuals and narratives, addressed neurodualism (e.g., "when we learn healthier thinking patterns and emotion regulation skills in psychotherapy, we are actually rewiring our brains"), explained that biology can be controllable (e.g., "by practicing what we learn in therapy, we're actively taking charge of our biological functions"), and reasons why psychosocial and biological causes are not mutually exclusive (e.g., "Even if someone might be more likely to develop depression because of their biology, often something stressful in life has to happen to kickstart those feelings of depression.").

Even if the intervention is effective, it might be because of its emphasis on psychotherapy's effectiveness for depression rather than psychotherapy's role in biology. Thus, without explaining psychotherapy's biological effects, the active-control video emphasized psychotherapy's effectiveness (e.g., "psychotherapy has been a reliable

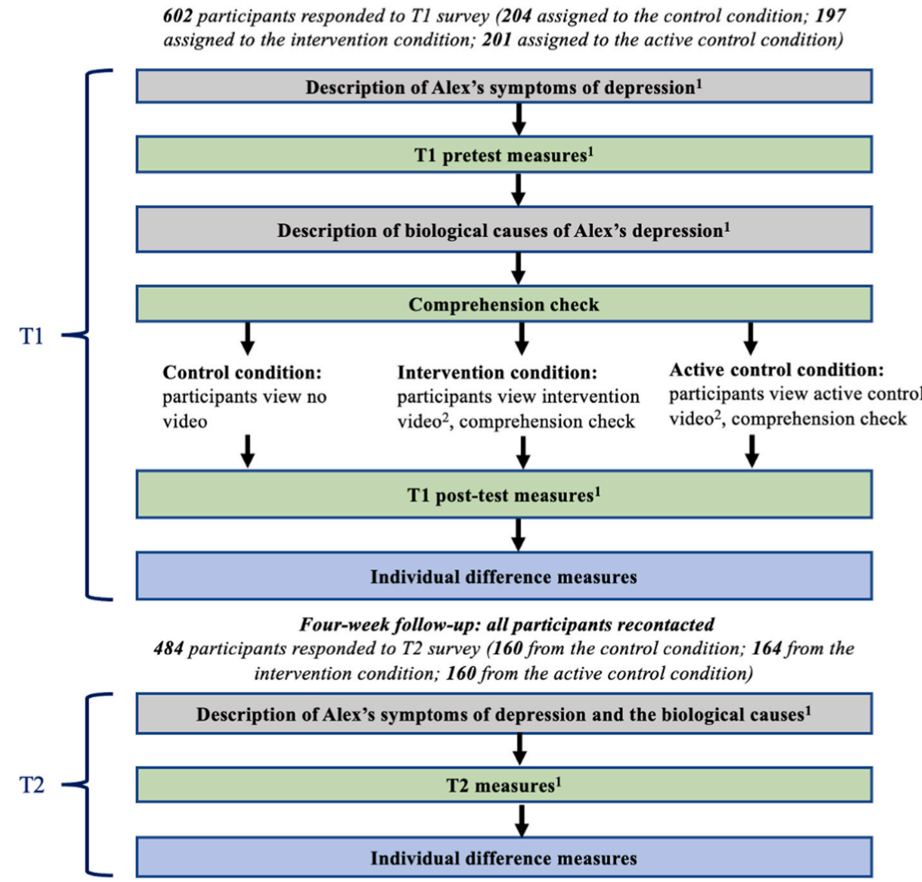

Fig. 1. Study Procedures
Notes: [1]See the supplement. [2]See https://www.youtube.com/watch?v=XUzF-xI3c9M.

treatment option for many people in managing depression"), underscored depression's controllability (e.g., "by choosing to engage in psychotherapy, people with depression can learn and practice the skills that are needed to take control over their own depression") and made psychosocial and biological causes salient (e.g., "… depression can result from various causes, including biological factors (such as brain abnormalities and genetics) and psychosocial factors (such as stressful life events or problems from childhood)"). Both videos used a male narrator (a lab volunteer) and similar visuals and were matched for length (intervention: 7:55 min, 1092 words; active-control: 7:20 min, 1036 words).

Intervention- and active-control-participants then answered four True/False questions on the videos they viewed; participants who missed any had to re-take them. One failed the pre-set criterion.

Then, all participants took the CEQ again (the T1-post-test-measure), followed by the Beck Depression Inventory-II (BDI-II) (Beck et al., 1961) and demographic questions (see the Supplement for analyses involving these secondary measures at T1 and T2). Lastly, participants were reminded about the follow-up survey in four weeks.

T2. After about four weeks participants were recontacted via Prolific to complete the T2 measures online ($M_{response\text{-}time}$ = 29.83 days, SD = 3.64, range = 28–45 days, with no condition differences, p = .177 (see the Supplement for details). All participants read the passages from T1 about Alex's depression and its biological causes and completed the T2-CEQ's "based on how you currently think and feel," followed by the BDI-II and demographic questions.

4. Results

This section reports results on psychotherapy ratings only (see the Supplement for the medication results). See https://osf.io/quj48/for the full data. Data was analyzed using SPSS-V28, excluding any participants with missing data on any outcome measure (N = 26 at T1 and N = 18 at T2).

The T1-pretest-CEQ, T1-post-test-CEQ, and T2-CEQ yielded Cronbach's *alphas* of .93, .96, and .95, respectively, so three CEQ composite scores (T1-pretest, T1-post-test, and T2) were created for each participant.

A 2(timepoint: pretest vs. post-test) x 3(condition) mixed ANOVA over T1-CEQ-scores with timepoint as a within-subject variable revealed no main effect of timepoint, a significant main effect of condition, and a significant interaction effect (Fig. 2, Table 1). A one-way ANOVA on pretest scores showed no significant differences among the conditions, p = .107, indicating that the interaction effect occurred because the pre-test to post-test change depended on condition.

CEQ ratings decreased significantly from pretest to post-test in the control condition, but increased significantly in the intervention condition and in the active control condition (see Table 2). But the increase was significantly greater in the intervention (M_{change} = 1.08, SD = 1.29) than in the active control (M_{change} = .21, SD = 1.46), $t(380) = 6.18$, $p <$ .001, d = .63, 95% CI [.43, .84].

Examining only the post-test CEQ-scores, a one-way ANOVA showed a significant effect of condition, $F(2, 585) = 86.65$, $p <$ .001, η_p^2 = .23, 95% CI for η_p^2 [.18, .27]. Post-hoc analyses using a Bonferroni correction for multiple comparisons showed that T1-post-test-CEQ-scores in the intervention condition were significantly higher than those in the active control condition, $p <$ .001, 95% CI for the mean difference [.33, 1.17], which in turn were significantly higher than those in the control condition, $p <$ .001, 95% CI for the mean difference [1.07, 1.89].

Next, we examined the data from the 484 participants who also

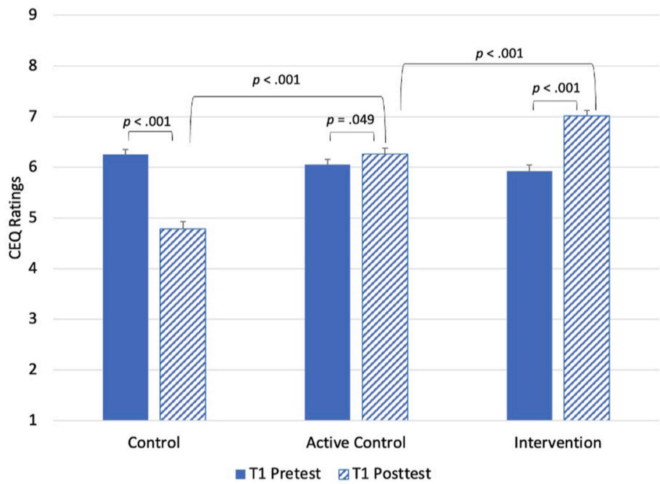

Fig. 2. Mean CEQ-psychotherapy-ratings by participants who completed T1 (N = 602)
Note. Error bars indicate SEs.

completed T2 (Fig. 3, Table 2). As at T1, a one-way ANOVA over T2-CEQ-ratings revealed a significant difference among the conditions, $F(2, 470) = 10.81$, $p < .001$, $\eta_p^2 = .04$, 95% CI for η_p^2 [.01, .08]. Pairwise comparisons using a Bonferroni correction for multiple comparisons showed that T2-CEQ-ratings were significantly higher in the intervention compared to the active control, $p = .012$, 95% CI for the mean difference [.10, 1.06] and control conditions, $p < .001$, 95% CI for the mean difference [.44, 1.39]. However, the two control conditions did not significantly differ, $p = .284$, 95% CI for the mean difference [-.82, .15].

A 2(timepoint: T1 pretest vs. T2) x 3(conditions) mixed ANOVA with timepoint as a within-subject variable revealed a significant main effect of timepoint, but not of condition. Critically, there was a significant interaction effect. See Table 1.

A one-way ANOVA over T1-pretest-CEQ-ratings for this sample showed no effect of condition, $F(2, 474) = 1.38$, $p = .253$, $\eta_p^2 < .01$, 95% CI for η_p^2 [0, .02]. But at T2, both control- and active control-participants showed significantly *less* trust in psychotherapy than at T1-pretest. Though, the decrease was significantly larger in the control ($M_{change} = 1.09$, $SD = 2.06$) versus active control ($M_{change} = .49$, $SD = 1.61$), $t(304) = 2.84$, $p = .006$, $d = .32$, 95% CI [.09, .54], showing some benefit of the active control video. In contrast, intervention-condition-participants showed no decrease, with the means rather in the opposite direction, albeit not significantly (Table 2).

5. Discussion

To summarize, without any intervention, participants exhibited significant distrust in psychotherapy for biologically attributed depression, replicating previous findings (Iselin and Addis, 2003; Schroder et al., 2020). In contrast, those who watched the intervention video on psychotherapy's biological implications increased their trust in psychotherapy and continued to show this benefit even after four weeks. A similar video that did not explain psychotherapy's biological implications was less effective, and its initial small benefit disappeared by follow-up. Together, these findings support our hypotheses that psychotherapy ratings at post-test would be highest in the intervention condition, and that this pattern would persist at follow-up.

The current findings have important clinical, theoretical, and methodological implications. Clinically, discounting psychotherapy for biologically attributed depression is concerning because the American Psychiatric Association recommends psychotherapy even for biologically attributed mental disorders (Tadmon and Olfson, 2022). Such

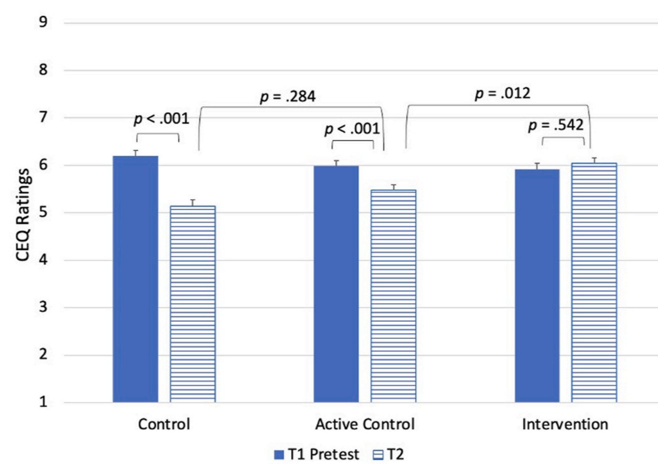

Fig. 3. Mean CEQ-psychotherapy-ratings by participants who completed T2 (N = 484)
Note. Error bars indicate SEs.

Table 1
Results from a 2 (timepoint) x 3 (condition) mixed ANOVA.

Effects	T1 Sample				T2 Sample			
	df	*F*	*p*	Effect size [95% CI]	*df*	*F*	*p*	Effect size [95% CI]
Timepoint	1, 573	1.34	.247	$f = .02$ [0, .13]	1, 463	34.43	<.001	$f = .27$ [.18, .36]
Condition	2, 573	23.49	<.001	$\eta_p^2 = .08$ [.04, .12]	2, 463	2.49	.084	$\eta_p^2 = .01$, [0, .03]
Interaction	2, 573	141.78	<.001	$\eta_p^2 = .33$ [.27, .39]	2, 463	15.97	<.001	$\eta_p^2 = .07$ [.03, .11]

Table 2
Means and paired *t*-test results.

	T1 Sample						T2 Sample					
	Control		Active Control		Intervention		Control		Active Control		Intervention	
	Pretest	Post-test	Pretest	Post-test	Pretest	Post-test	Pretest	T2	Pretest	T2	Pretest	T2
Mean (SD)	6.25 (1.49)	4.74 (2.01)	6.06 (1.43)	6.27 (1.64)	5.94 (1.73)	7.02 (1.43)	6.20 (1.52)	5.11 (1.91)	5.98 (1.41)	5.48 (1.72)	5.95 (1.76)	6.05 (1.68)
t-test	$t(193) = 11.65$, $p <$ .001, $d_z = .84$		$t(191) = -1.98$, $p =$ .049, $d_z = .14$		$t(189) = -11.50$, $p <$ .001, $d_z = .83$		$t(152) = 6.53$, $p <$ .001, $d_z = .53$		$t(152) = 3.84$, $p <$ .001, $d_z = .31$		$t(159) = -.61$, $p = .542$, $d_z = .05$	
95% CI for effect size dz	[.67, 1.00]		[-.29, .00]		[-1.0, −.67]		[.36, .70]		[.15, .47]		[-.20, .11]	

distrust in psychotherapy could also be contributing to the decreasing utilization of psychotherapy (e.g., Olfson and Marcus, 2010) amid an increasing focus on the biology of mental disorders. Yet, the approach tested in the current study could potentially contribute to decelerating or even reversing the trend.

Theoretically, it has been argued that mind-body dualism, from which the targeted misconceptions stem, is universal and developmentally entrenched (Bloom, 2004). However, our findings indicate that it can be enduringly mitigated to some extent.

Methodologically, the current results build on previous investigations into restoring trust in psychotherapy for biologically attributed depression (Perricone and Ahn, 2023a, 2023b; Perricone et al., 2023). First, while these studies addressed each of the three aforementioned misconceptions separately, the current study demonstrated the feasibility of simultaneously correcting these beliefs. Second, whereas the previous studies only tested the immediate effects of the interventions, this study demonstrated the intervention's durability. Third, the immediate benefits observed in their studies might have been influenced by demand characteristics, where participants feel compelled to provide the 'correct' responses as suggested by the intervention. The current participants showed similar responses at T2 in the absence of the intervention, making demand characteristics an unlikely explanation.

However, there are important limitations to this study. Firstly, the intervention should be validated in a symptomatic sample where participants are reasoning about treatment for their own symptoms (see Perricone and Ahn, 2023a; Perricone and Ahn, 2023b; Perricone et al., 2023). Similarly, future studies should examine mental health clinicians who also become skeptical about the efficacy of psychotherapy when mental disorders are biologically attributed (Ahn et al., 2009).

Secondly, this study relied solely on the CEQ as an outcome measure. However, various treatment-related outcomes (e.g., whether the intervention mitigates therapy attrition, encourages greater engagement with homework assignments, or strengthens the therapeutic alliance) should be assessed in clinical settings.

Thirdly, this study utilized a U.S. sample consisting primarily of white individuals. Although forms of mind-body dualism are found across cultures (Chudek et al., 2013), the relevance of the specific misconceptions addressed by this intervention in more ethnically and culturally diverse samples remains to be determined.

Fourth, future research should test whether a similar intervention can rectify misconceptions that people have about other more biologically attributed mental disorders (e.g., schizophrenia, bipolar disorder; see Ahn et al., 2009) and physical health conditions that nevertheless benefit from psychotherapy (e.g., insomnia, chronic pain; see Gilpin et al., 2019).

Upon resolving the scope of the current findings over various samples, measures, and conditions, the brief video intervention can be easily disseminated for broader public health utility, helping those in need of therapy to seek treatment despite biological attributions. The intervention can also be applied in clinical settings, to help patients collaboratively decide on treatment steps with their provider or to enhance patients' confidence in the psychotherapy they are currently receiving.

In conclusion, this study found that trust in psychotherapy for biologically attributed depression can be durably enhanced by explaining that psychotherapy changes the brain, people have agency over biology, and biological and psychological causes interact. The intervention's long-lasting benefits and ease of administration highlight its real-world potential for clinical utility after further examinations of the scope of the findings.

Ethical statement

This study received ethics approval from the Yale University Institutional Review Board.

CRediT authorship contribution statement

Annalise M. Perricone: Writing – review & editing, Writing – original draft, Validation, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Woo-kyoung Ahn:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of generative AI and AI-assisted technologies in the writing process

The authors used ChatGPT 4 to edit the earlier drafts of the Abstract written by the authors for concision and readability. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Data availability

All data used in this manuscript and the supplementary material is publicaly available via the OSF link provided in the manuscript.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2024.117202.

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