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An Open Trial of Emotion Regulation Therapy For Generalized Anxiety Disorder and Co-Occurring Depression

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Abstract

Background—Although CBT is efficacious for a wide variety of psychiatric conditions, relatively fewer GAD patients achieve high endstate functioning as compared to patients receiving CBTs for other disorders. Moreover, GAD trials that utilized patient samples without prominent depression have tended to report that effect sizes for depressive outcomes were small or diminished to pre-treatment levels in the follow-up period. Emotion Regulation Therapy (ERT) integrates principles from traditional and contemporary cognitive behavioral treatments with basic and translational findings from affect science to offer a blueprint for improving intervention by focusing on motivational, regulatory, and contextual learning mechanisms.

Method—The purpose of this investigation was to provide initial support for the efficacy of ERT in an open trial of patients with GAD and co-occurring depressive symptoms. Twenty-one patients received a 20-session version of ERT delivered in weekly individual sessions. Standardized clinician ratings and self-report measures were assessed at pre-, mid-, and post-treatment as well as at three- and nine-month follow-ups. Intent-to-treat analyzes were utilized.

Results—GAD patients, half with comorbid major depression, evidenced statistically and clinically meaningful improvements in symptom severity, impairment, quality of life, and in model-related outcomes including emotional/motivational intensity, mindful attending/acceptance, decentering, and cognitive reappraisal. Patients maintained gains across the three and nine month follow-up periods.

Conclusions—These findings, although preliminary, provide additional evidence for the role of emotion dysregulation in the onset, maintenance, and now treatment of conditions such as GAD and co-occurring depressive symptoms.

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Keywords

Generalized Anxiety Disorder; Depression; Mindfulness/Meditation; Treatment; Clinical Trials; Behavior Therapy; Anxiety/Anxiety Disorders; Emotion Regulation

Although often viewed as a mild problem of the "worried well"[1] and, subsequently, under-investigated[2], generalized anxiety disorder (GAD) is a actually a chronic and debilitating condition that is associated with diminished impairment equivalent to major depressive disorder (MDD[3]), poorer life quality than MDD when only non-comorbid cases are compared between the two disorders[4], and independent associations with negative functional outcomes when both disorders are comorbid[5]. Despite the considerable burden it can cause, GAD, and conceptually related conditions (e.g., MDD with anxious apprehensive features) lag behind in treatment efficacy compared to other anxiety and mood disorders[6], makes otherwise efficacious interventions for MDD refractory[7], and are associated with notable difficulties in maintaining durability of comorbid MDD gains over the long term[8].

Much of our understanding of the phenomena underlying GAD (i.e., anticipatory anxiety, worry) has stemmed from the work of Borkovec[9], who demonstrated that worry serves an avoidance function or, as more recently explicated, that worry becomes reinforced by increasing predictability of negative emotional experience, often at the cost of experiencing positive and rewarding states (i.e., diminishing emotional contrasts[10]). A functional perspective of worry has been supported in studies utilizing neurobiological[11], psychophysiological[12–14], behavioral[15], and self-report[16–17] measurement. Further, investigators have built on Borkovec's perspective by emphasizing the role of maladaptive cognitive (i.e., intolerance of uncertainty[18]; meta-worry[19]), emotional (i.e., non-acceptance[17]; dysregulation[16, 20]), and interpersonal[21, 22] processes in increasing worry.

These findings have advanced our understanding of GAD but have also resulted in conceptual heterogeneity. Affect science may provide a comprehensive yet empirically sound framework for integrating these findings, particularly in relation to compensatory functions of worry and other destructive forms of self-referential mentation[23]. Elsewhere, we have developed an emotion regulation theory that posits that dysfunction in distress disorders such as GAD can best be understood by 1) *motivational mechanisms*, reflecting the functional and directional properties of an emotional response tendency; (2) *regulatory mechanisms*, reflecting the alteration of emotional response trajectories utilizing less and more elaborative systems; and (3) *contextual learning consequences*, reflecting, optimally, the promotion of broad and flexible behavioral repertoires. Thus, dysfunction in GAD can be understood by a failure in each of these normative systems of functioning[24].

Using this framework, GAD, especially when comorbid with MDD, can be seen as characterized by subjective emotional intensity/distress[20], reflecting greater temperamental negative affect[25–26] and activation of motivational systems that prize obtaining safety (from perceived threat) over seeking reward[27–29] [motivational mechanisms]. Subsequently, these individuals may respond poorly to this motivational conflict, demonstrating deficits in different systems of regulation[30] encompassing attentional (e.g., attentional flexibility in processing both interoceptive and exteroceptive emotional stimuli[31–33, 11]) and more

cognitive elaborative strategies (e.g., meta-cognitive awareness, cognitive reappraisal[34, 35]). Instead, individuals with GAD often resort to perseverative strategies (e.g., worry, rumination, self-criticism) to compensate for an inability to manage distressing emotions and motivations [regulatory mechanisms]. The net result of these regulatory failures is a paradoxical maintenance of these negative states[10] and reduced clarity in understanding and optimally responding to them[36]. Indeed, worry results in increased threat conditionability, greater stimulus generalization, and diminished ability to discriminate stimuli and learning contingencies[37–39] and GAD is associated with restrictions in valued actions and goals[40]. Similarly, rumination is associated with decreases in the likelihood of new reward-based learning, weakens instrumental action, and engagement of potentially rewarding social networks[41–43] [contextual learning consequences].

Emotion Regulation Therapy (ERT) was derived from this affect science perspective with the goal of increasing motivational awareness, developing less and more elaborate regulatory capacities, and engaging novel contexts to generate new learning repertoires. ERT draws from traditional[9, 44] and contemporary CBT[45–48] as well as experiential therapy[49] to form an integrated, mechanism-targeted, CBT endeavoring to improve acute and enduring treatment efficacy for GAD especially when comorbid with MDD. The present study represents a preliminary examination of the efficacy of ERT utilizing an open trial design. We hypothesized that ERT would diminish GAD and comorbid depressive symptoms and produce durable gains in functioning/life quality as well as model-related outcome measures including emotional intensity, mindful attention regulation (i.e., mindfulness, acceptance), and more elaborative meta-cognitive regulation (e.g., decentering, reappraisal).

Method

Participants

Participants were 21 treatment-seeking individuals at two PhD training clinic sites in the Northeastern United States (Site 1 was located in Philadelphia, PA, $n = 11$; Site 2 was located in New Haven, CT, $n = 10$). Institutional Review Boards approved procedures for the study at both sites and all patients offered informed consent. The *Anxiety Disorders Interview Schedule, Lifetime version for DSM-IV* (ADIS[50]; site 1) and the *Structured Clinical Interview for DSM-IV* (SCID[51]; site 2) are semi-structured diagnostic interviews that were use to diagnosis GAD and other disorders. Interviewers at both sites were clinical psychologists or doctoral students in clinical psychology trained according to the guidelines of the ADIS or SCID. The intake clinician assessed for all Axis I disorders and provided a rating of severity using the ADIS clinician severity rating (CSR). A score of 4 or greater (range=0–8) is given for diagnoses that meet full DSM-IV criteria and are clinically significant. GAD was expected to be primary or co-primary in all participants. In addition, the GAD module was also administered prior to treatment by the independent assessor. Diagnostic agreement (in diagnosis and CSR within 1 point; ADIS CSR was used at both sites) was necessary for study inclusion.

Additional inclusion criteria consisted of being at least 18 years old; fluent in spoken and written English; and willing and able to give informed written consent for the treatment

protocol. Exclusion criteria consisted of a principal DSM-IV diagnosis other than or in addition to GAD; prominent active suicidal ideation/intent; DSM-IV diagnosis of substance abuse or dependence within the previous 6 months; a current DSM-IV diagnosis of organic mental disorder; schizophrenia, psychotic disorder, bipolar I disorder, or dementia; an unwillingness to terminate or suspend concurrent psychotherapy; and concurrent psychotropic medication not stabilized for at least 3 months.

The sample was mostly women (*n* = 17), aged 35.25 (*SD* = 10.96), primarily Caucasian (*n* =18) and well-educated (16 had at least a college education). Eleven patients were employed full time with the remaining patients reporting working part-time (*n* = 2), being full-time students (*n* = 5), or unemployed (*n* = 3). Median income for the sample was $40000 (Range = $0 to $124000). Eleven patients had a concurrent MDD diagnosis. Sixteen patients had at least one additional current diagnosis (Range: 1– 4) including obsessive compulsive disorder (*n* = 2), panic disorder (*n* = 5), post-traumatic stress disorder (*n* = 2), social phobia (*n* = 6), specific phobia (*n* = 2), dysthymic disorder (*n* = 1), eating disorder (*n* = 1), and an Axis II diagnosis (*n* = 2). Four patients participated while receiving concurrent antidepressant medication; four patients entered the trial receiving a benzodiazepine[1]. Twenty patients completed the acute treatment phase of the trial and entered a no-treatment follow-up (three-months & nine-months follow-up). The one patient who withdrew from the trial did so following an emergent medical crisis unrelated to GAD or any other emotional difficulty.

Findings are based upon an intent-to-treat (ITT) analysis plan using data from all 21 patients. There were no demographic differences across the two sites, and no baseline clinical and demographic differences associated with treatment response (analyses available upon request). Thus, findings are aggregated across sites.

Treatment

ERT consisted of 20 weekly sessions of 60 minute duration except for Sessions 11–16 which lasted 90 minutes to accommodate exposure exercises. In the first half, sessions focus on teaching emotion regulation strategies so that clients respond “counteractively” (instead of “reactively”) via the utilization of mindful attending to somatic and emotional cues (i.e., attention regulation) and more verbally elaborate emotion regulation skills (i.e., meta-cognitive regulation). In the second half of ERT, patients endeavor to become more antecedent[30] or “proactive” in their deployment of regulation skills by engaging contexts that simultaneously invoke both elevated reward and threat motivations via in-session exposure exercises and out-of-session exposure exercises conducted in the ensuing week. In the final sessions, the focus shifts to consolidating gains and preparing for termination[52, 53].

Measures

Patients completed both clinician-assessed and self-report measures of anxiety, depression, worry, disability and quality of life as well as model-related outcome measures. The assessment schedule of these measures was pre-treatment, mid-treatment, post-acute

[1]Outcome analyses reported below utilized the full sample. We reran analyses in either participants who did not have any medication usage (n=14) or who did not have anti-depressant medication usage (n=16). These analyses replicated the findings from the whole sample analyses. Thus, we report the whole sample analyses below.

treatment, and three- and nine-month follow-up. Intake interviewers (pre-treatment) as well as independent assessors (all time points) who were blind to particular details about the patient (beyond receiving ERT) completed the clinician measures.

Clinician and Diagnostic Assessment

Independent assessors administered clinician and diagnostic assessments at all assessment points including the ADIS CSR and a modified version of the *Clinical Global Impression Rating Scales* (CGI[54]). Specifically, a version with anchor points developed specifically for rating impairment and changes in symptoms associated with GAD was utilized. Clients received a rating of 1 (very much improved) or 2 (much improved) were classified as responders.

Self-report Symptom and Severity Measures

The *Penn State Worry Questionnaire* (*PSWQ*[55]) is a widely used 16-item measure of trait worry. The *Beck Depression Inventory-II* (BDI–II[56]) is a 21-item self-report measure that assesses the affective, cognitive, behavioral, and somatic symptoms of depression. The *Mood and Anxiety Symptom Questionnaire-Short Form* (MASQ[57]) is a 62-item instrument comprised of four subscales: General Distress Anxious Symptoms (GDA), General Distress Depressive Symptoms (GDD), Anxious Arousal (AA), and Anhedonic Depression (AD). The 7-item *State-Trait Anxiety Inventory* (STAI[58, 59]) is one of the most frequently used measures of trait anxiety. The Sheehan Disability Scale (SDS[60]) is a commonly utilized measure assessing impairment at work, in social relationships, and in responsibilities at home and with family. The *Quality of Life Inventory* [QOLI[61]] assesses the degree to which an individual is satisfied with 16 areas of his or her life (e.g., health, standard of living, friendships, relationship with family, community, etc.)

Model-Related Outcome Measures

The 10-item Negative Intensity scale from the *Affect Intensity Measure* (AIM[62]) was used to assess emotional intensity. The 11-item Decentering subscale of the *Experiences Questionnaire*[63] was used to assess the construct of decentering (i.e., the meta-cognitive ability to observe items that arise in the mind with distance and perspective). The *Difficulties in Emotion Regulation Scale* (DERS[64]) is a 36-item measure of the acceptance of emotions, ability to engage in goal-directed behavior when distressed, impulse control, awareness of emotions, access to strategies for regulation, and clarity of emotions. The *Emotion Regulation Questionnaire* (ERQ[65]) is a 10-item rationally derived measure of two aspects of emotion regulation: reappraisal and suppression. The *Five Facet Mindfulness Questionnaire* (FFMQ[66]) is a 39-item self report measure assessing trait mindfulness.

Analysis Plan and Assessing Clinical Significance

For the purpose of statistical analysis, conceptually similar measures were grouped into families of analyses: Diagnostic Outcomes, Self-Reported Anxiety Outcomes, Self-Reported Depression Outcomes, Disability/Quality of Life Outcomes, and ERT Model-Related Outcomes. A series of paired samples *t*-tests comparing Pre-Treatment to Mid-Treatment, Post-Treatment, 3-Month Follow-up, and 9-Month Follow-up. Given multiple comparisons

and to address the possibility of Type I errors, we utilized a Bonferroni correction for each family of analysis, which resulted in only reporting significance if p-values were below .0125 (.05/4) for Diagnostic Outcomes, .0125 (.05/4) for Self-Reported Anxiety outcomes, .016 (.05/3) for Self-Reported Depression Outcomes, .025 (.05/2) for Disability/Quality of Life outcomes, and .01 (.05/5) for Model-Related Outcomes. Further, Hedges' *g* effect size estimates of within-subject change are presented in Table 2. Like Cohen's *d*[67], Hedges' *g* is interpreted with conventions of Small = .20, Medium = .50, Large = .80.

Clinically significant improvement was also assessed in four ways (see Table 3). First, simple clinical response was reflected in a CSR score less than 4, which reflects the threshold for a full diagnosis (all current diagnoses including GAD and MDD) or a CGI-Improvement (CGI-I) score less than 3 (only available for GAD). Two more stringent indices of clinical significance were derived from procedures used by[68] and[69]. Specifically, patients were regarded as GAD responders by evidencing a clinically meaningful response on at least 4 of the following 6 GAD indices: (GAD CSR < 4, CGI-I < 3, at least 30% improvement on the PSWQ, STAI-7, MASQ-AA, & MASQ-GDA). Similarly, patients were regarded as MDD responders if they evidenced a clinically meaningful response on at least 3 of 4 MDD indices (MDD CSR < 4,at least 30% improvement on the BDI-II, MASQ-AD, & MASQ-GDD). Also, an index of high endstate functioning was derived by assessing whether patients fell into the normative range (within one standard deviation of healthy norms on published clinical measures[69]) on at least 4 of 6 GAD measures (GAD CSR, CGI-I, PSWQ, STAI-7, MASQ-AA, & MASQ-GDA) and 3 of 4 MDD measures (MDD CSR, BDI-II, MASQ-AD, & MASQ-GDD). These two measures of clinical significance yield dichotomous scores (Responder = 1, Non-Responder = 0). Consistent with an ITT approach, clinical significance during the acute treatment phase of the trial was assessed as a ratio of the number of responders over the total number of patients enrolled in the acute phase (N = 21). In the follow-up phase, clinical significance was assessed based on the number of completers at that time point as well as the total number of patients entering the no-treatment follow-up phase (N = 20). Given that missing data were minimal in the follow-up period, a last observation carried forward approach was employed by carrying forward post-acute treatment data into the three month follow-up assessment point (n = 1) and carrying forward three-month follow-up data into the nine-month follow-up assessment point (n = 3).

Results

Treatment Adherence

Treatment adherence ratings were completed by two independent raters who rated 25% of the therapy recordings. Inter-rater agreement was derived by having raters concurrently code 40% of sessions being reviewed for adherence. Raters were instructed to code the frequency, and degree of skillfulness evidenced by therapists. Raters also tallied the number of times therapists broke protocol. High agreement was established for both frequency (90%) and skillfulness (88%) ratings. In terms of treatment adherence, ERT therapists evidenced high frequency (80% to 100%) and skillfulness (73% to 100%) of treatment component delivery. Further, there were relatively few lapses into non-treatment components per session (0.1 to 1.6).

Effects of treatment on diagnostic outcomes

See Tables 1 (means and standard deviations) and 2 (test statistics, significance levels, and effect sizes). Findings demonstrated improvements from pre- to mid-treatment (g = 1.42) on GAD CSR (other diagnoses were not assessed at mid-treatment). Diagnostic indices for all anxiety and mood disorders significantly improved from pre-treatment to post-treatment and the follow-up periods (g's 0.52 to 3.90). For all measures, effect sizes approximate or exceed conventions for a large effect size.

Effects of treatment on self-reported anxiety outcomes

See Tables 1 and 2. Findings reveal improvements by mid-treatment (*g*'s 0.71 to 0.98) with additional gains by post-treatment (*g*'s 0.85 to 1.67). Relative to pre-treatment, gains in anxiety outcomes were maintained at three-month follow-up (*g*'s 0.94 to 1.59) and nine-month follow-up (*g*'s 1.04 to 1.77). For all measures, effect sizes approximate or exceed conventions for a large effect size.

Effects of treatment on self-reported depression outcome[2]

See Tables 1 and 2. Findings reveal some improvement by mid-treatment for depression outcomes (*g*'s = 0.35 to 0.82) with sizable gains by post-treatment for all measures (*g*'s = 0.76 to 1.25). Relative to pre-treatment, gains in depression outcomes were maintained at three-month follow-up (*g*'s = 0.65 to 1.17) and nine-month follow-up (*g*'s = 0.93 to 1.35). MDD CSR and MASQ-AD effect sizes exceed conventions for a medium effect size (Pre-Post; Pre-three-month) and approach conventions for a large effect size (Pre to nine-month). For all other measures, effect sizes estimates approximate or exceed conventions for a large effect size at all time points.

Effects of treatment on disability/quality of life

As seen in Tables 1 and 2, findings reveal improvement by mid-treatment for the SDS (*g* = 0.45), but not the QOLI (*g* = −0.30) but sizable gains by post-treatment for both measures (*g*'s= −0.90 to 0.94). Relative to pre-treatment, gains in disability and quality of life are maintained at three-month follow-up (*g*'s = −.77 to .79) and nine-month follow-up (*g*'s = −1.05 to 1.11). Effect sizes estimates for pre to post-treatment and the follow-up periods approximate or exceed conventions for a large effect size at all remaining time points.

Effects of treatment on ERT model-related outcomes

Findings reveal no improvements by mid-treatment for candidate mechanism measures (*g*'s = −0.23 to −0.75). However, at post-treatment, all measures show sizable improvements (*g*'s = −0.61 to −1.32). Relative to pre-treatment, gains in all but one model-related outcome (i.e., AIM Negative Intensity) were maintained at three-month follow-up (*g*'s = 0.25 to −1.30). At nine-month follow-up, all measures remain measurably improved (including AIM Negative Intensity) relative to pre-treatment (*g*'s = 0.65 to −1.32). Across time points, effect

[2]All outcome analyses were also conducted within the subgroup of patients diagnosed with MDD (n=11). Baseline mean (SDs) for this group were: GAD CSR = 6.00 (0.71), MDD CSR = 4.67 (1.00), BDI = 23 (7.96), MASQ-AD = 74.8 (13.89), MASQ-GDA = 31.2 (5.63). Anxiety, Depression, Disability/Quality of Life, and Model-Related Outcomes had comparable effect sizes to analyses conducted on the whole sample (Hedge's *g*'s ranged from 1.53 to 3.93).

sizes for model related outcomes exceed conventions for medium effects and in some instances exceed conventions for large effect sizes.

Clinical Significance Analyses for GAD and MDD Outcomes

Open-label ERT was associated with impressive rates of clinician-assessed improvement (CSR < 4, 81.0% or CGI-I < 3, 95.2%) with gains maintained or increased at three months and nine months post-treatment (Range = 85% to 95%). Using a more stringent index where patients must evidence at least 30% improvement on at least 4 or 6 clinician or self-report GAD measures, a considerable majority of patients receiving open-label ERT also evidenced strong gains following treatment (81%) with the gains maintained at three months (90%) and nine months (90%). Finally, patients were assessed against whether their treatment gains resulted in normalization on at least 4 of 6 clinician and self-report measures (i.e., high endstate functioning). Findings indicated that 66.7% of patients achieved high endstate functioning following treatment and that the percent increased at three months (75.0%) and nine months (85.0%).

With respect to MDD improvement, patients receiving ERT evidence considerable gains. For instance, nine of 11 patients entering the trial with comorbid MDD achieved a CSR score of less than four by the end of treatment. Moreover, all 11 of these patients remained or achieved subclinical on the MDD CSR at three months and nine months following the end of treatment. Using a more stringent index where patients must evidence at least 30% improvement on at least 3 of 4 clinician or self-report MDD measures, a majority of patients with comorbid MDD (54.5%) evidenced clinical improvement with the rate increasing at three months (90.0%) but diminishing somewhat at nine months (60.0%). Finally, 45.5% of patients with comorbid MDD achieved high endstate functioning following treatment, and the percentage increased at three months (70%) and nine months (80.0%).

Discussion

Findings from this open trial offer initial support for the efficacy of ERT in the treatment of GAD. In this trial, GAD patients, half meeting MDD diagnostic criteria, evidenced statistically and clinically meaningful improvements in symptom severity, impairment, quality of life, and in model-related outcomes including emotional/motivational intensity, mindful attending/acceptance, decentering, and cognitive reappraisal. Patients were then followed for nine months, and treatment gains were maintained. These findings, although preliminary, provide additional evidence for the role of emotion dysregulation (see[52, 53]) in the onset, maintenance, and now treatment of conditions such as GAD with and without MDD.

Until now, two overarching themes characterized the extant literature on CBT treatments for GAD. First, although CBT is clearly efficacious, relatively fewer GAD patients achieve high endstate functioning as compared to patients receiving CBTs formulated for other mood and anxiety disorders[6]. Second, many of these trials utilized samples of GAD patients without prominent MDD or depression[68–70] and, in some instances, found that effect sizes for depression outcomes were small or diminished to pre-treatment levels in the follow-up period[8]. Similarly, non-CBT treatments such as mindfulness-based interventions consisting

solely of meditative practice[71–73] or bias modification programs[74, 75], which may be seen as requiring less effort to deliver, have shown some promise in reducing GAD severity but often did not enroll GAD patients with prominent levels of depression[72]), restore patients to high endstate functioning[75, 76], or significantly resolve depression when it was present at pre-treatment[74, 75].

One possible explanation for these relatively inferior efficacy findings is that these treatments are often targeting different components of a heterogenous condition. For instance, traditional CBTs largely utilize more cognitively elaborative, verbally mediated, techniques that are focused on cognitively elaborate deficits such as distorted beliefs. In contrast, mindfulness-based meditative interventions and bias modification programs focally target less elaborative, attentional, deficits without relying heavily on verbal, linguistically mediated, exchange. Although speculative, greater efficacy may be achieved for GAD (particularly when comorbid diagnoses or symptoms are present) by treatments that target both more and less elaborative deficits. Elsewhere, we have argued that core intervention principles that address both lesser and greater elaborative processes (i.e., simultaneously increasing attentional flexibility, improving meta-cognitive processing, and promoting the engagement of both threat and reward contexts) may be the conceptual framework that underlies successful CBT interventions[77]. Indeed, recent CBT interventions have integrated both less and more elaborate training components by synthesizing mindfulness and attention training with traditional CBT techniques[78–80], resulting in high endstate functioning which is achieved and maintained through follow-up periods, while also sampling patients evidencing prominent depression features at baseline[78–80]. Although open trial designs have several weaknesses (e.g., lack of a control comparison, ability to address expectancy effects), ERT provided notably high response rates and endstate functioning levels, utilized a sample with prominent baseline depression, and produced large reductions in MDD symptoms and severity that were maintained into the follow-up period. Further, model-related outcomes that span less and more elaborative regulatory capacities (e.g., mindful attention/acceptance, decentering, and reappraisal) were improved and maintained. The magnitude of these findings matches or exceeds findings from the trials noted above. However, a well-powered, randomized control design is necessary to corroborate the significance of these findings for the efficacy of ERT. Further, despite these strong effects, this trial utilized a 20-session version of ERT. It is unclear if this length is necessary and cost-effective. We are currently testing 16 and 8 session versions to determine the proper level of dosing for clinically significant effects.

An important next step, and one that aligns well with NIMH priorities (i.e., Research Domain Criteria [RDoC][24]) is to refine interventions so that treatment components are comprehensive in addressing the scope of dysfunction in GAD but still targeted to specific candidate mechanisms which comprise this condition. Such an approach is consistent with the model posited within ERT where GAD, especially when it co-presents with MDD, is marked by motivational/emotional intensity and a reliance on maladaptive less elaborative (i.e., attentional) and more elaborative (i.e., meta-cognitive) emotion regulation strategies, which result in suboptimal threat- and reward-related contextual learning. Many of these characteristics were assessed and shown to improve and maintain in the follow-up period, suggesting their consideration as candidate mechanisms of change. However, we make this

claim with appropriate caution, given the preliminary nature of this trial, the small sample size, and the reliance on self-report indices.

It will also be important to contextualize our hypothesized mechanisms within other frameworks for emotional phenomena (e.g.,[81–83]) as well as more actively test how our treatment aligns and is distinguished from treatments utilizing similar components (e.g.,[47, 48]), especially given meta-analytic findings demonstrating that common elements such as mindfulness have been shown to contribute to efficacy in a number of intervention approaches (see[84, 85]). Future studies using a randomized control design, with a larger sample size, an assessment schedule that accounts for alternative mechanisms as well as temporal precedence of these mechanisms before symptomatic change, ideally while assessing objective biobehavioral indices, are necessary before stronger mechanism claims can be made. We are currently utilizing cognitive, behavioral, psychophysiological, and neural indices of our proposed mechanisms to examine their relationship to clinical outcomes. Despite these caveats, findings from this trial do support the preliminary efficacy of ERT for GAD and co-occurring depressive symptoms and suggest that affect science may provide a useful framework for integrating efficacious approaches to anxiety and depression.

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Table 1

Means (and Standard Deviations) of study variables

Measure	Pre-Treatment	Mid-Treatment	Post-Treatment	3-Mo. Follow-up	9-Mo. Follow-up
Diagnostic Outcomes					
GAD CSR	5.75 (0.78)	4.25 (1.24)	2.71 (0.91)	2.50 (1.05)	2.25 (1.16)
MDD CSR*	1.69 (2.27)	--	0.69 (1.45)	0.25 (2.19)	0.41 (0.93)
# Anxiety Diagnoses*	0.81 (1.03)	--	0.14 (0.35)	0.19 (0.40)	0.10 (0.30)
# Mood Diagnoses*	0.50 (0.51)	--	0.05 (0.22)	0.00 (0.00)	0.00 (0.00)
Self-Reported Anxiety Outcomes					
CGI Severity	5.05 (0.52)	4.00 (0.75)	2.85 (0.67)	2.32 (1.03)	2.37 (1.17)
PSWQ	66.63 (9.24)	59.26 (8.20)	52.35 (11.33)	50.72 (12.21)	47.31 (12.64)
STAI-7	19.59 (3.99)	14.12 (2.37)	12.78 (3.08)	13.19 (4.94)	13.14 (2.71)
MASQ-AA	28.47 (6.72)	23.89 (5.93)	22.90 (5.44)	22.67 (5.31)	22.88 (3.24)
MASQ-GDA	29.95 (5.87)	24.52 (5.00)	20.15 (4.64)	20.67 (5.58)	20.88 (6.05)
Self-Reported Depression Outcomes					
BDI-II	16.90 (9.97)	9.00 (8.60)	6.20 (6.79)	6.63 (6.91)	5.23 (6.60)
MASQ-AD	68.89 (14.29)	64.05 (13.31)	58.45 (14.38)	59.28 (14.74)	55.69 (13.47)
MASQ-GDD	32.79 (9.90)	27.95 (8.33)	23.55 (7.86)	24.61 (10.85)	22.56 (8.24)
Disability/Quality of Life Outcomes					
Sheehan Disability Scale	12.41 (6.65)	9.53 (6.01)	6.84 (4.93)	7.12 (6.55)	5.67 (5.22)
QOLI	0.25 (1.48)	0.71 (1.55)	1.59 (1.42)	1.40 (1.51)	1.89 (1.58)
Model Related Outcomes					
DERS Total	91.79 (20.67)	85.21 (14.85)	77.35 (19.85)	75.94 (23.30)	73.56 (16.21)
FFMQ Total	115.29 (24.59)	119.94 (14.17)	130.47 (24.20)	121.76 (15.53)	130.75 (15.97)
EQ-Decentering	30.53 (4.71)	32.41 (4.09)	36.11 (4.72)	35.20 (4.52)	36.93 (4.80)
ERQ-Reappraisal	21.68 (5.81)	26.44 (6.65)	28.00 (4.21)	27.94 (3.45)	27.69 (5.57)
AIM-Negative Intensity	38.67 (6.44)	39.11 (5.86)	35.11 (6.61)	37.12 (5.73)	34.75 (5.79)

Note. GAD=generalized anxiety disorder, CSR=Clinician's Severity Rating from the Anxiety Disorders Interview Schedule for DSM-IV, Lifetime Version; CGI=Clinical Global Impression; PSWQ=Penn State Worry Questionnaire; STAI=State Trait Anxiety Inventory; MASQ=Mood and Anxiety Symptom Questionnaire; AA=Anxious Arousal; GDA=General Distress Anxiousness; MDD=Major Depressive Disorder; BDI-II=Beck Depression Inventory-II; QOLI=Quality of Life Inventory; AD=Anhedonic Depression; GDD=General Distress Depression; QOLI=Quality of Life Inventory; DERS=Difficulties in Emotion Regulation Scale; FFMQ=Five Facet Mindfulness Questionnaire; ERQ=Emotion Regulation Questionnaire; EQ=Experiences Questionnaire; AIM=Affective Intensity Scale;

*Clinician assessment not conducted at Mid-Treatment; Patients without MDD coded CSR=0

Table 2

t-statistics and Hedge's g effect size estimates showing clinical change over time

	Pre to Mid		Pre to Post		Pre to 3-mo. Follow-up		Pre to 9-mo. Follow-up	
	t	***g***	***t***	***g***	***t***	***g***	***t***	***g***
Diagnostic Outcomes								
GAD CSR	4.39***	1.42	12.06***	3.90	9.41***	3.39	10.64***	3.41
MDD CSR*	--	--	2.28**	0.52	2.80**	0.63	3.08**	0.72
# Anxiety Diagnoses*	--	--	3.16**	0.84	3.08**	0.78	2.91**	0.91
# Mood Diagnoses*	--	--	3.93**	1.12	4.36***	1.36	4.32***	1.36
Self-Reported Anxiety Outcomes								
PSWQ	5.24***	0.83	7.02***	1.40	6.46***	1.50	5.90***	1.77
STAI-7	6.47***	1.09	9.19***	1.67	5.45***	1.56	6.56***	1.58
MASQ-AA	2.45	0.71	3.26**	0.85	3.00**	0.94	2.34	1.04
MASQ-GDA	3.30**	0.98	7.28***	1.79	5.96***	1.59	3.88***	1.49
Self-Reported Depression Outcomes								
BDI-II	5.28***	0.82	6.04***	1.25	4.83***	1.17	5.21***	1.35
MASQ-AD	1.69	0.35	3.53**	0.76	3.07**	0.65	3.19**	0.93
MASQ-GDD	2.60	0.52	5.85***	1.07	4.36***	0.77	3.00**	1.10
Disability/Quality of Life Outcomes								
Sheehan Disability Scale	2.43*	0.45	4.09***	0.94	4.18***	0.79	3.22**	1.11
QOLI	−1.70	−0.30	6.03***	−0.90	3.22**	−0.77	4.03***	−1.05
Model Related Outcomes								
DERS Total	1.55	0.37	3.45**	1.00	3.05**	0.72	3.98***	1.00
FFMQ Total	−1.16	−0.23	−3.13**	−0.61	−2.23	−0.32	3.12**	−0.73
EQ-Decentering	−1.97	−0.42	−5.26***	−1.30	−4.48***	−1.00	5.18***	−1.32
ERQ-Reappraisal	−2.02	−0.75	−4.26**	−1.32	−2.85**	−1.30	2.93**	−1.04
AIM-Negative Intensity	0.47	−0.07	3.29**	0.66	1.42	0.25	3.48**	0.63

Note. GAD=generalized anxiety disorder, CSR=Clinician's Severity Rating from the Anxiety Disorders Interview Schedule for DSM-IV, Lifetime Version; CGI=Clinical Global Impression; PSWQ=Penn State Worry Questionnaire; STAI=State Trait Anxiety Inventory; MASQ=Mood and Anxiety Symptom Questionnaire; AA=Anxious Arousal; GDA=General Distress Anxiousness; MDD=Major

Depressive Disorder; BDI-II=Beck Depression Inventory-II; QOLI=Quality of Life Inventory; AD=Anhedonic Depression; GDD=General Distress Depression; QOLI=Quality of Life Inventory; DERS=Difficulties in Emotion Regulation Scale; FFMQ=Five Facet Mindfulness Questionnaire; ERQ=Emotion Regulation Questionnaire; EQ=Experiences Questionnaire; AIM=Affective Intensity Scale;

* $p < .05$;

** $p < .01$;

*** $p < .001$;

All Hedge's g's computed as ITT/ITF ($n = 21$)

Table 3

Number of treatment responders (and % ITT sample size) using conventional indices of clinical significance

	Post-Acute Treatment # Responders (% ITT)	3-Month Follow-up # Responders (% ITF)	9-Month Follow-up # Responders (% ITF)
GAD Clinical Response			
ADIS GAD CSR < 4	17/21 (81.0%)	17/20 (85.0%)	18/20 (90.0%)
CGI-Improvement < 3	20/21 (95.2%)	19/20 (95.0%)	19/20 (95.0%)
30% Improvement (4+ of 6 Criteria Met)	17/21 (81.0%)	18/20 (90.0%)	18/20 (90.0%)
High Endstate Functioning (4+ of 6 Criteria Met)	14/21 (66.7%)	15/20 (75.0%)	17/20 (85.0%)
MDD Clinical Response			
ADIS MDD CSR < 4	9/11 (82.0%)	10/10 (100%)	10/10 (100%)
30% Improvement (3+ of 4 Criteria Met)	6/11 (54.5%)	9/10 (90.0%)	6/10 (60.0%)
High Endstate Functioning (3+ of 4 Criteria Met)	5/11 (45.5%)	7/10 (70.0%)	8/10 (80.0%)

Note. All findings based upon ITT analyses at post-acute (N = 21) and ITF in the no-treatment follow-up periods (N = 20)