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# Relaxation-Induced Anxiety: Effects of Peak and Trajectories of Change on Treatment Outcome for Generalized Anxiety Disorder

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## Abstract

**Objective:** Evidence is mixed regarding whether relaxation-induced anxiety (RIA) impedes relaxation training (RT) efficacy. Unlike past studies that averaged RIA across sessions, we examined peak RIA, change in RIA level across sessions, and timing of peak RIA with outcome.

**Method:** This was a secondary analysis of Borkovec et al. (2002). Forty-one GAD participants were assigned randomly to CBT ($n = 22$) or BT ($n = 19$). Both treatments contained RT and RIA ratings within 13/14 sessions. Analyses used generalized additive mixed models, which accounted for longitudinal nonindependence and examined non-linear trajectories of change.

**Results:** All participants improved significantly regardless of RIA. Average change trajectory of RIA level did not predict outcome. Instead, lower peak RIA predicted fewer GAD symptoms at post-treatment and greater likelihood to continue to improve during follow-up. Also, timing of peak was important. Whereas lower peak early in therapy did not predict outcome, lower peak during the last third of treatment did. Peak RIA's effect was neither accounted for by baseline symptom severity, treatment condition, comorbidity, nor by preceding or concurrent anxiety symptom change.

**Conclusions:** People with consistently low peak RIA and/or who fully habituate to RIA by the end of therapy respond optimally to relaxation-based treatments.

### Keywords

GAD; relaxation training; relaxation-induced anxiety; CBT; behavioral therapy

Generalized anxiety disorder (GAD) is widespread, detrimental, and chronic (Kertz & Woodruff-Borden, 2011; Kessler & Wang, 2008; Newman, Llera, Erickson, Przeworski, & Castonguay, 2013). Although much research has demonstrated that cognitive-behavioral

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therapy (CBT) is efficacious for treating GAD, it is considered the least successfully treated among the anxiety disorders (Newman, Castonguay, Borkovec, Fisher, & Nordberg, 2008). Thus, research on facets of GAD that may account for these insufficient gains is paramount.

Undesirable treatment outcomes may be partially linked to one GAD phenomenon that has received very little research attention: Relaxation-induced anxiety (RIA). RIA is a paradoxical increase in physiological, behavioral, and cognitive aspects of anxiety when an individual engages in efforts to relax (Braith, McCullough, & Bush, 1988; Heide & Borkovec, 1983). Those afflicted with persistent GAD are particularly prone to RIA, as demonstrated by clinical application studies of both progressive muscle relaxation and frontalis EMG biofeedback (Borkovec & Grayson, 1980; Heide & Borkovec, 1983; Raskin, Johnson, & Rondestvedt, 1973). According to etiological theories of RIA, individuals who are especially concerned with maintaining control over physical and psychological processes may find relaxation vulnerable, unpleasant, and activating (Lehrer, 1982). Discomfort and concern with a perceived lack of control during relaxed moments—an inability to “let go”—may result in unsought increases in anxiety during therapeutic attempts at relaxation (Adler, Craske, & Barlow, 1987; Clark, 1989; Smith, 1989). A strong drive to manage personal experience and turning to worries during rest may also provoke feelings that counter relaxation (Heide & Borkovec, 1984; Raskin et al., 1973). These notions are consistent with the Contrast Avoidance Theory, which proposes that clients with GAD engage in worry to elicit and sustain anxiety and tension as a means to avoid being vulnerable to sharp shifts in negative emotion (Newman & Llera, 2011). According to this idea, any degree of successful relaxation would leave people more vulnerable to shifts in anxiety. The threat posed by such shifts may elicit RIA. The drive to remain in control has been posited to serve a primarily defensive or preparatory function for the anxious—attempting to evade or prepare for future threat (e.g. Walsh, 1977). The controlled body is held in a state of chronic tension and hypervigilance by the sympathetic nervous system, mobilizing for sudden action.

Relaxation training (RT), a highly effective group of treatments for GAD, means to counter this defensive stance by slackening muscles, softening hypervigilance toward fear cues, regulating breath, and increasing parasympathetic activation. After training and ongoing practice, clients are equipped to self-induce relaxation in response to identified tension and worry triggers and to maintain a state of lowered tension throughout the day. Many common CBT strategies for anxiety target relaxation, including diaphragmatic breathing, progressive muscle relaxation (PMR), and applied relaxation (AR). RT alone is an empirically supported treatment for GAD (Chambless & Ollendick, 2001). It has demonstrated similar efficacy to mindfulness based cognitive therapy (MBCT), cognitive therapy (CT), and combined CBT packages (Bolognesi, Baldwin, & Ruini, 2014; Hayes-Skelton, Roemer, & Orsillo, 2013; Siev & Chambless, 2007). Achieving relaxed states is vital for RT to work. Yet for those with RIA, when RT causes increased anxiety, it may reverse the process of enabling reduced tension. Treatments relying on relaxation could be victim to this paradoxical effect.

Viewed simply, relaxation techniques can become core coping tools to prevent the spiking of anxiety and worry, eventually reducing anxiety itself in the long-term. According to theory, physiological relaxation is meant to compete with anxiety-promoting behaviors and thoughts, bringing about new learning by muting anxiety (Denny, 1976). This process is

initiated via ongoing and consistent formal relaxation practice—learning how to identify tension and relax it away—until novel patterns are formed (Newman & Borkovec, 2002). At least one animal study suggests that relaxation is a long-latency response that only fosters learning if relaxation lasts until it is fully completed (Denny, 1976). After extended applied and progressive relaxation practice, clients learn to cut off their worry spiral by intervening immediately in response to anxiety cues and creating a competing response. Yet the opposite effect could occur for those with RIA. Theoretically, if relaxation practice breeds anxiety rather than relief, and if that anxiety does not sufficiently dissipate during the intervention, relaxation would be ineffective for conditioning change. Worse, continuing to attempt to guide GAD persons to relax, triggering RIA, and then pairing the experience with anxiety-eliciting cues could not only perpetuate preexisting anxious associations, but also reinforce them over time (Denny, 1976). If those with GAD are both unable to relax during RT and have *increases* in their anxiety, the potential benefits of RT could be curtailed.

Only four psychotherapy trials containing RT have examined whether RIA negatively predicted outcome and have yielded inconclusive results. Whereas some studies found a negative impact of RIA across measures, others have not found a strong relationship. For example, in a single-session administration of PMR (Bernstein & Borkovec, 1973), participants who reported higher RIA during attempted relaxation had significantly worse outcome post-treatment on a composite of 10 measures compared to those with lower RIA (Heide & Borkovec, 1983). Similar results arose from a more extensive 12-session treatment comparing nondirective therapy, cognitive therapy, and self-control desensitization (SCD). In SCD, clients receive progressive muscle relaxation until relaxed, then engage in worry trigger imagery afterward, followed by applied relaxation and positive coping imagery as a means of counterconditioning. All three of these treatment conditions included RT during which RIA was assessed. Higher average in-session RIA predicted poorer outcomes at post-treatment on assessor severity of GAD and a daily diary measure, but not on 5 other measures (Borkovec & Mathews, 1988). Also, in another 12-session trial for GAD including RT plus either cognitive therapy or nondirective therapy, although RIA averaged across in-session practice was negatively associated with assessor-rated severity and the Hamilton Anxiety and Depression scales, there was no relationship of RIA with outcome on six other anxiety-related measures (Borkovec et al., 1987). Similarly, following 12 sessions of either AR or CBT (which included AR and SCD), Borkovec and Costello (1993) observed that greater levels of average RIA during in-session relaxation practice predicted less reduction in anxiety on daily diary measures at 1-year follow up. However, these authors did not find a link between RIA and any other measure of outcome. Furthermore, they found no relationship between RIA and *any* outcome measure immediately after treatment or at 6 months post-treatment. Thus, the actual impact of RIA on GAD treatment remains uncertain.

The inconsistency of findings may be due to prior studies' approach to analyses. Each merely used a treatment-spanning mean of client in-session RIA scores, which is likely a poor measure for capturing its full range of influences. A more intricate understanding of RIA may be achieved through methods of greater detail and sensitivity, including effects of RIA peak, timing of peak, and change in RIA level across time. For instance, with respect to peak, it is possible that a particularly high level of RIA in even one session may restrict potential therapy effects more than consistently low levels. Heide and Borkovec (1984) have

proposed that “sheer intensity” of aversive experiences during RT matters. Clients are taught to ascribe highly focused attention to physical sensations of relaxation in their muscles and viscera, monitoring internal experience. If anxiety surges while clients are attending closely to somatic feelings, such anxiety may not only be more striking, but also more likely to be conditioned to both the internal experience of attempting to relax and the external training procedure. The higher the anxiety response, the greater the likelihood of forming a powerful behavioral association and the lower the likelihood that it will be overcome across time. Since PMR generally gets shorter in duration over treatment due to grouping sets of muscles together (Bernstein & Borkovec, 1973), this form of relaxation may not allow for higher peaks of RIA to habituate despite continued exposure. Sessions late in the treatment may not last long enough to let strong RIA diminish. Thus, it is possible that a higher peak level of RIA, even if infrequent, may prove more interfering to RT than consistently low levels of RIA.

Along with these behavioral ramifications, the pairing of peak RIA levels with somatic sensations may have cognitive consequences. Clients may form beliefs about anxiety during RT that further promote and fortify long-term RIA. Braith et al. (1988) found that all RT subjects who experienced RIA reported an awareness of somatic anxiety cues first, followed by worry about what might be indicated by the anxiety. Thus, high-impact RIA sessions may prompt or create a concern about increased arousal, exacerbating the desire to rely on controlling strategies rather than “letting go” strategies during RT. Higher levels of RIA may also create the belief that relaxation is simply a fruitless endeavor. In addition, Heide and Borkovec (1984) posited that worries about events unrelated to relaxation, such as financial or relational problems, may arise during RT, given a propensity for people with GAD to turn to worry during quiet moments. Since worry increases physiological and subjective anxiety (Newman & Llera, 2011), this cognitive activity may further amplify RIA and be linked to peak RIA levels.

In addition to neglect of peak RIA, prior studies have not examined trends of change in RIA level across treatment. Therapists’ repeated attempts to train relaxation techniques and diminish physiological reactivity might alter clients’ experience of relaxation over time—for better or worse. For instance, Heide and Borkovec (1983) revealed decreases in subjective and physiological RIA (within-session habituation) due to one session of PMR. The logic of exposure treatments may suggest that repeated exposure to anxiety-provoking RT or RT-associated cues might also result in between-session habituation of RIA responses over time. Yet for reasons stated previously, repeated inductions of RIA without allowing for full habituation may reinforce RIA across therapy instead—a potential effect of PMR sessions that grow shorter over treatment.

The current study attempted to address the inconsistency in prior findings by examining effects of peak RIA, timing of the peak, and change in level over time based on 13 consecutive sessions containing RT with individual RIA ratings. Our primary hypothesis was that on average for GAD participants, the higher one’s greatest within-session peak in RIA across the trial, the worse one’s treatment outcome would be. This prediction was based on the idea that higher peak RIA would increase the likelihood of reinforcing anxiety associated with RT, given that RT became shorter as treatment progressed. We also had several

secondary hypotheses. First, the higher one's greatest within-session peak in RIA across the trial, the higher would be their anxious cognitions averaged across the trial, given research that anxious cognitions perpetuate anxiety and impede emotional processing (Newman & Llera, 2011). We also predicted that GAD clients would demonstrate different trends in fluctuation of RIA level across the treatment period (e.g. a linear increase or quadratic rise and fall). Yet these trajectories would show no influence on outcome. Thus, we predicted that participants might show increased levels of RIA across treatments or increase and then decrease, but this pattern would be less important than the final peak levels of RIA that participants reached after repeated exposure to relaxation practice. Given that peak RIA exhibited during the last few sessions of therapy is a proxy for the degree to which participants habituated to their RIA across sessions (Foa & Kozak, 1986), we believed that whereas peak RIA during the first third of treatment would not predict outcome, a higher peak in the last third of treatment would predict worse outcome.

Kraemer, Wilson, Fairburn, and Agras (2002) have suggested that when proposing potential treatment predictors, researchers should avoid those variables that simply reflect change in symptoms in response to treatment. Therefore, an additional goal of the present study was to examine the covariation of peak RIA with concurrent symptom change. If peak RIA significantly covaried with changes in symptom severity, this might suggest that peak RIA was a proxy for such changes. However, if these phenomena were orthogonal, it would substantiate the notion that peak RIA independently predicted symptom reduction following treatment. Related to this, we examined whether preceding symptom reduction predicted peak RIA during either the middle or last third of treatment. If preceding symptom reduction did not predict peak RIA, this would further substantiate peak RIA as independent of symptom reduction during treatment. RIA was collected within 13 of 14 therapy sessions, and anxiety severity data were collected four times daily over the course of the study, allowing for the modeling of within-treatment processes.

## Methods

The present investigation draws on data from Borkovec, Newman, Pincus, & Lytle's (2002) dismantling study on the efficacy of CBT components for GAD. Participants were assigned randomly to three therapy conditions containing 14 sessions: 1) solely cognitive therapy (CT), 2) both applied relaxation and self-control desensitization training (BT), and 3) CT plus both applied relaxation and SCD (CBT). Only conditions 2 and 3 included RT and therefore clients who received 1 were excluded from analyses. In every session that included some form of RT, participants rated their RIA immediately after the relaxation training was received.

### Participants

Of the 46 participants in the two relaxation conditions, five did not receive a sufficient dose of in-session relaxation practice (receiving relaxation training in fewer than 80 percent of therapy sessions). Consequently, there was a total sample of 41 participants who received either combined CBT ($n = 22$) or BT ($n = 19$). The sample was comprised of 27 females and 14 males who were 81% White, 7.3% Hispanic, 4.9% Black, and 4.9% Middle Eastern. All

participants met DSM-III-R criteria (American Psychiatric Association, 1987) for GAD. All but two participants (95.8%) also met DSM-IV criteria (American Psychiatric Association, 1994). Consensus on two independent administrations of the Anxiety Disorders Interview Schedule-III-R (ADIS-III-R; Di Nardo & Barlow, 1988) was required for diagnosis. A trained assessor conducted the first administration, and the therapist who would later treat the client conducted the second. All trained assessors were kept blind to condition. As reported in Borkovec et. al. (2002), any person meeting criteria for panic disorder, severe major depressive disorder, substance abuse, psychosis, or organic brain syndrome was excluded from the study. Regarding the current sample, at baseline 61% had at least one comorbid diagnosis, 43.9% met criteria for social phobia, 9.8% met criteria for simple phobia, 9.8% met criteria for major depressive disorder, 4.9% met criteria for dysthymic disorder, and 2.4% met criteria for post-traumatic stress disorder. Elaborate description of comorbidity in this treatment sample and its influence on treatment outcome can be found in Newman, Przeworski, Fisher, and Borkovec (2010).

## Materials

All participants were administered the following measures prior to treatment, at treatment termination, and at 6-, 12- and 24-month follow-up assessments.

**Penn State Worry Questionnaire—**(PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item self-report measure of pathological worry. Factor analysis indicated the PSWQ assesses a unidimensional construct with internal consistency of .91 (Meyer et al., 1990; .83 in the current sample). High retest reliability (ranging from .74 to .93) was also demonstrated across periods ranging from 2 to 10 weeks (Molina & Borkovec, 1994). Correlations between the PSWQ and measures of anxiety, depression, and emotional control supported convergent and discriminant validity of the measure (Brown, Antony, & Barlow, 1992).

**Hamilton Anxiety Rating Scale—**(HARS; Hamilton, 1959). This 14-item clinician-administered scale provides a rating of severity of each anxiety symptom cluster on a scale from 0 (not present) to 4 (very severe/incapacitating). Internal consistency ranged from adequate to good ($\alpha$ = .77 to .81; Moras, di Nardo, & Barlow, 1992; .82 in the current sample). Retest reliability (intraclass correlation coefficient, or ICC) was .86 across 2 days, and interrater reliability ICCs ranged from .74 to .96 (Bruss, Gruenberg, Goldstein, & Barber, 1994; ICC = .86 in the present sample).

**State-Trait Anxiety Inventory-Trait Anxiety Subscale—**(STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). This 20- item scale measures trait anxiety. Internal consistency is high (in the .80s and .90s; .86 in the current sample), and retest reliability is much higher for the Trait form (high .70s) than the State form (ranging from .27 to .54). Convergent and discriminant validity has also been demonstrated for this questionnaire (Spielberger et al., 1983).

**Clinician's Severity Rating—**(CSR; Di Nardo & Barlow, 1988). As part of the ADIS-R, at each assessment session, trained assessors blind to condition rated the severity of each

participant's symptoms of GAD (0–8 point scale, with 0 = symptoms absent, 4 = moderate symptom severity, and 8 = very severe symptom severity). Diagnostic reliability of CSRs in the current study ranged from an intraclass correlation of .74 to 1.

**Response to Relaxation Session (RRS) questionnaire.—**This measure assesses RIA during relaxation therapy and was used in all prior studies on RIA (e.g., Heide & Borkovec, 1983; Borkovec & Costello, 1993). Immediately after each in-session relaxation practice, clients rated the item "How much did you notice an *increase* in anxiety or nervousness during the relaxation session?" on a 9-point scale (0 = no anxiety, 2 = slight anxiety, 4 = definite anxiety, 6 = marked anxiety, 8 = severe anxiety), providing an ongoing session by session measure of relaxation-induced anxiety (Heide & Borkovec, 1983). They also rated on a 9-point scale their level of distraction by anxious cognitions (0 = not at all distracted, 8 = distracted the entire time) during PMR using the item, "How frequently was your attention distracted by *unpleasant* or *negative* thoughts or images?" (hereafter termed Cognitive RIA). We also examined retest reliability across two of the later therapy sessions (when change from therapy would be likely to level off) held three weeks apart and found good stability for RIA *(r* = .73) and cognitive RIA (*r* = .798).

**Client Daily Diary (CDD).—**Patients recorded anxiety levels four times a day throughout the therapy period (upon arising, end of morning, end of afternoon, and end of evening), rating their overall level of anxiety during the preceding period of the day on a 0–100 scale. Two-week retest reliability was .80 based on weekly averages from the baseline data of the current trial. In addition, convergent and discriminant validity was demonstrated by significantly stronger correlations with the Hamilton Anxiety Scale than with the Hamilton Depression Scale (Newman & Fisher, 2013). The average diary compliance rate in the current study was 95%.

## Procedure

The study procedure is reported within the relevant bounds of the current study's focus. Participants were randomly assigned to one of two treatment conditions (CBT or BT). Each condition consisted of 14 weekly sessions, with one termination session after post-assessment. Also, the first four sessions lasted two hours to account for sufficient psychoeducation on intervention principles, whereas remaining sessions were one and a half hours long. The first session of both conditions did not include relaxation training of any form. Consequently, the RRS was not administered during this session and was not included in the analysis. Every other session did include applied relaxation with or without SCD and therefore included RRS measurement. Follow-up assessment occurred 6-, 12-, and 24-months after termination.

**Behavioral Treatment.—**The first 30 minutes of each behavioral treatment (BT) session involved only supportive listening, based on the manual used in Borkovec and Costello (1993). The methodological purpose of this portion was to hold constant the total amount of treatment time while also holding constant the total amount of time devoted to BT techniques in both BT and CBT. The supportive listening manual instructed therapists to provide an accepting, nonjudgmental, empathic environment.

Clients were informed that treatment would entail learning new coping techniques. Worry and anxiety were described as a habitual, spiraling process and treatment involved self-monitoring of internal reactions and their sequential nature; learning to catch the worry spiral early and to intervene with a variety of relaxation responses to anxious thoughts, feelings, and images to disrupt anxious spirals and to create new coping habits; learning to focus attention on present-moment experience rather than on mentally created past events or future possibilities; and imaginal rehearsal of coping methods to facilitate coping-response-habit acquisition. Relaxation training over the sessions included the full course of progressive relaxation training, cue controlled, and differential relaxation training as described in Bernstein and Borkovec (1973), slowed diaphragmatic breathing, relaxing imagery, and meditational relaxation. The same techniques were used in applied-relaxation training (Öst, 1987), wherein clients learned to deploy their relaxation responses frequently throughout the day and in response to any incipient anxiety cues. Emphasis was placed on formal relaxation practice twice a day to strengthen the relaxation response and frequent application during the day. When RIA occurred, clients were encouraged to continue relaxation practice with the idea that repeated exposure would lead to dissipation of RIA. Practicing these applications occurred within each therapy session as well. Over sessions, discussions increasingly focused on flexible choice of relaxation methods depending on the internal and external circumstances. Participants also received self-control desensitization (SCD). In the second session, clients constructed worry cue hierarchies for use during formal SCD practice, which began in session 4. For each SCD practice, clients first attempted to achieve deep relaxation via progressive muscle relaxation, after which they reported their levels of RIA. Next they imagined themselves in a situation that typically triggered worry until they noted the presence of anxious feelings. They then continued imagining the external situation while imagining that they were deploying coping responses and concurrently used applied relaxation. At the elimination of anxious feelings, they imagined continued coping deployments for 20 s and then turned off all imagery and focused only on their relaxed state for 20 s. Scenes were repeated until clients could no longer generate anxiety or were able to eliminate it rapidly (i.e., within 5–7 s). Homework emphasized frequent applications of applied relaxation, SCD practiced twice/day, and focus on living in the present moment.

**CBT.—**CBT contained all of the treatment techniques described above plus extensive interventions using cognitive restructuring techniques and behavioral experiments. In addition, no supportive listening element was included in CBT.

**Therapists.—**Three doctoral-level therapists and one advanced clinical graduate student therapist conducted the treatment. All therapists saw a nearly equal number of clients in each condition. There were no differences between therapists on any outcome measure.

## Planned analyses

Based on a power calculation, a sample size of 40 was considered adequate (using RMASS2; using power = 0.80 and effect sizes of 0.4) (Hedeker, Gibbons, & Waternaux, 1999). For the outcome measure we formed a composite score of the anxiety measures by summing standardized values of the PSWQ, the HARS, the STAI-T, and CSR. Missing

values were handled using full information maximum-likelihood (FIML). All of the primary fixed effect coefficient's effect sizes were converted to Cohen's *d,* using the following equations for *F*-statistics, Z-statistics, *t*-statistics, and chi-squared statistics:$d = \frac{2 * \sqrt{F}}{\sqrt{(N-1)}}$, $d = \frac{2 * t}{\sqrt{(N-1}}$, $d = Z * 2\backslash\sqrt{N}$ , and $d = ((4\chi^2)/(N - \chi^2))^{1/2}$ (Dunst, Hamby, & Trivette, 2004; Wolf, 1986).

**Primary analyses.—**Peak RIA was defined as the highest level each participant experienced within any of the 13 relaxation practice sessions. Analyses used generalized additive mixed models (GAMMs). GAMMs are extensions of the general linear model (GLM) and include all aspects of a multilevel model, namely: (1) linear fixed effects and (2) linear random effects. Additionally, these models allow the investigation of complex non-linear trajectories over time with coefficients called "smooths" or "splines." These coefficients allow one to investigate the non-linear predictor of a main effect or interaction as highly non-linear over time. Notably, GAMMs have been shown to be robust to small sample sizes (Jiménez-Valverde, Lobo, & Hortal, 2009; Tutz & Binder, 2006).

Reader concerns may exist about the type I error rate in these models, as model complexity may be thought to induce greater possibility of chance findings. Although GAMMs allow the estimation of complex non-linear dynamics, the present models have estimator properties that directly combat these issues. Specifically, we utilized thin-plate regression splines (rather than penalized cubic splines, B-splines) because these splines directly penalize model complexity with model fit (i.e. the model will only introduce a non-linear bend to the data if it would result in substantially better fit).

The primary outcome was tested via the following equations. The outcome composite of $Anxiety_{i,j}\ \mu_i \equiv E(Anxiety_{i,j})$, is linked to a semiparametric predictor, $\eta_i$, expressed as

$$\eta_i = \gamma_{00} + u_{i,o} + f_1(time_{i,j}) + f_2(peakRIA_{i,o}) + f_3(time_{i,j}, peakRIA_{i,o}) + e_{i,j}$$

In this equation, the first two terms are the conventional fixed and random effects in a linear multilevel model. Specifically, $\gamma_{00}$ is a fixed effect and represents the grand intercept across all persons, $u_{i,0}$ is a random effect and represents the intercept for each person. The term $f_1$ is the smooth function for $time_{i,j}$, allowing for non-linear effects of time on anxiety symptoms at outcome (i.e. the treatment effects). Note that the term $time_{i,j}$, includes both pre-treatment, post-treatment, 6-month, 12-month, and 24-month follow-up, meaning that all change (pre-post and post-follow-up) is modeled in this term. The term $f_2$ is the smooth function for $peakRIA_{i,0}$, allowing for non-linear effects of peak RIA on anxiety symptoms at outcome (i.e. non-linear main effect of peak RIA on outcome). The term $f_3$ is for the smooths of tensor products used to approximate the unknown but jointly nonlinear effects of a $time_{i,j}$ and $peakRIA_{i,0}$ on outcome anxiety levels (i.e. the non-linear moderation of peak RIA levels on treatment effects). Lastly, $e_{i,j}$ represents the residual error term for each person at each time period. Lastly, average RIA predicting outcome was analyzed in a GAMM framework to determine if peak RIA would show different results than average RIA, as predicted.

To better understand findings of this analysis, and to ensure that results were not due to one or two outliers, subgroups were formed to examine different trajectories at different levels of peak RIA. Visual examination of Figure 1 revealed that participants with peak RIA of 2 or below appeared to benefit maximally. However, to confirm this, participants were divided into three groups: (1) low peak RIA was defined as less than one standard deviation below the mean peak RIA (i.e. peak RIA between 0 to 2, $n = 8$), (2) moderate peak RIA was defined as between one standard deviation below and one standard deviation above the mean of the peak RIA (i.e. peak RIA between 3 to 6, $n = 22$), and (3) high peak RIA was defined as greater than one standard deviation above the mean of the peak RIA (i.e. peak RIA between 7 to 8, $n = 11$). Changes across discrete periods of time were also examined within these subgroup analyses by examining change between pre-post and post to 24-month follow-up by using the GAMM estimates.

**Secondary Analyses:** Peak cognitive RIA was defined as the highest level of cognitive RIA each participant experienced within any of the 13 relaxation practice sessions. Cognitive RIA was computed as the average across 13 session-level cognitive RIA ratings. The relationship between peak RIA and peak cognitive or cognitive RIA was examined using simple correlations.

We also investigated presence and effect of trajectories of change in RIA level across therapy on treatment outcome. These analyses tested our hypothesis that specific trends of change in RIA level would exist for individuals, but would not impact treatment outcome because the trend in change would not be as important as peak during the last third of treatment. Analyses proceeded in two modeling stages. First, we computed linear mixed models with RIA levels across time as the outcome to examine whether there were significant random effects (i.e. person-specific effects) for linear and quadratic time trends in RIA. Secondly, random effect coefficients for each individual's linear and quadratic slope of RIA were stored for use as predictors. Using GAMMS, we modeled these random effects as predictors by replacing "peak RIA" in equation 1 with random linear trend and random quadratic trend, respectively.

To determine whether peak RIA toward the end of treatment was more likely to predict outcome than RIA during the first part of treatment, we divided therapy into three separate time-blocked peaks and examined: (1) highest level of RIA across sessions 2 to 6, (2) highest level of RIA across sessions 7 to 10, and (3) highest level of RIA from sessions 11 to 14. We divided therapy into thirds to allow analyses to capture highest RIA level across timeframes over which RIA could be expected to meaningfully change (four to five sessions). We then estimated effect of peak RIA within each block using GAMMs by replacing "peak RIA" in equation 1 with each respective blocked peak RIA variable (peak within each third of therapy) for each analysis. We also ran within-subject t-tests to determine whether the means of peak RIA were significantly different between each block. Note that the time effect for all GAMMs secondary analyses included pre, post, 6 month, 1 year, and 2 year follow-up changes within one model.

Lastly, we wanted to ensure that peak RIA was independent of preceding or concurrent reductions in anxiety. Consequently, we studied change in client daily diary (CDD) anxiety

symptoms across three blocks of time. Specifically, we used CDD anxiety symptom change from session 1 to 6 as block 1, 7 to 10 as block 2, and 11 to 14 as block 3. Thus, the time periods reflected the same periods that were used for block peak RIA. To examine the change in this score, we computed linear mixed models with raw CDD levels across time as the outcome and we then stored the slope of the change of the random effects for use as predictors. Next, within GAMMs we used CDD random slope coefficients to predict peak RIA from concurrent time blocks (i.e. block 1 change in CDD predicting block 1 peak RIA) and across all future blocks (i.e. block 1 change in CDD predicting block 2 and 3 peak RIA) for each of the three blocks.

## Results

### Preliminary Analyses

There were no significant differences across therapy conditions in the baseline composite anxiety measure ($F(1, 39) = 0.004$, $p = .951$, $d = .004$). Individuals' peak RIA ranged from 0 to 8 on the RRS. In addition, those with comorbid diagnoses did not differ significantly in peak RIA from those with pure diagnoses ($B = -0.5375$, $SE = 0.7009$, $t = -0.767$, $p = 0.448$, $r = -0.121$). We also tested whether the effects of peak RIA and time were moderated by treatment condition using $f_4(time_{i,j}, peakRIA_{i,0})$ * treatment condition, and the moderation was not significant ($F = 0.492$, $p = .742$, $d = .222$). As such, we proceeded without this term for the primary analyses (see Table 1 for means and standard deviations of outcome measures across time). Peak RIA was also not associated with baseline anxiety symptoms ($t(40) = -0.154$, $p = .866$, $d = -0.049$). On average peak RIA in block one was not significantly different from peak RIA in block two ($t(40) = -0.567$, $p = .574$, d = −0.178), or peak RIA in block three ($t(38) = 0.666$, $p = .509$, $d = 0.216$). Likewise, peak RIA in block two did not differ significantly from peak RIA in block three ($t(38) = 1.213$, $p = .213$, $d = 0.393$). In addition, we tested the non-linear moderation of time with a linear relationship between peak RIA and outcome using $f_5(time_{i,j})$ peak $RIA_{i,0}$, but the term was also not significant ($F = 0.281$, $p = .751$, $d = .168$) and was consequently dropped from the model.

### Primary Results

The non-linear interaction (i.e. the tensor product term: $f_3$) between time and peak RIA significantly predicted outcome ($F = 3.96$, $p < .001$, $d = 0.63$; see Figure 1 for a graphical depiction), showing that level of one's peak RIA influenced treatment effects significantly across all time points. In regard to the subdivided sample, those with low ($\beta = -8.50$, $SE = 0.841$, $Z = -10.104$, $p < .001$, $d = -1.411$), moderate ($\beta = -6.27$, $SE = 0.779$, $Z = -8.045$, $p < .001$, $d = -1.123$), and high peak RIA ($\beta = -6.54$, $SE = 0.779$, $Z = -8.398$, $p < .001$, $d = -1.173$) changed significantly from pre-treatment to post-treatment, and all experienced significant reduction in GAD symptoms. However, those with low peak RIA changed significantly more than those with moderate ($\Delta\beta = -2.23$, $SE = 0.810$, $Z = -2.752$, $p = .006$, $d = 1.162$) and high peak RIA ($\Delta\beta = -1.95$, $SE = 0.810$, $Z = -2.411$, $p = .016$, $d = -0.813$). Differences in results between those with moderate and high peak RIA were not significant ($\Delta\beta = 0.23$, $SE = 0.780$, $Z = 0.354$, $p = .723$, $d = 0.050$). Thus, low peak RIA was associated with significantly greater change from pre-treatment to post-treatment, compared to those with either moderate or high peaks.

Regarding change between post-treatment and 2-year follow-up, dimensionally lower peak RIA was more likely to be associated with continued symptom reduction than higher peak RIA. In addition, when subdivided, those with low peak RIA changed significantly ($\beta$ = −9.61, $SE$ = 2.991, $Z$ = −3.214, $p$ = .001, $d$ = 1.06), but those with moderate ($\beta$ = 1.54, $SE$ = 0.922, $Z$ = 1.665, $p$ = .095, $d$ = 0.232) or high peak RIA ($\beta$ = 0.16, $SE$ = 0.741, $Z$ = 0.210, $p$ = .833, $d$ = 0.029) did not change significantly in simple slopes analyses. Likewise, those with low peak RIA continued to change more from post to 24-month follow-up than those with moderate ($\Delta\beta$ = −11.150, $SE$ = 1.957, $Z$ = −5.697, $p$ < .001, $d$ = 0.796) and high peak RIA ($\Delta\beta$ = −9.769, $SE$ = 1.866, $Z$ = −5.234, $p$ < .001, $d$ = 0.731). As with pre-post changes, there were no significant differences between moderate peak RIA and high peak RIA on post to 24-month follow-up change ($\Delta\beta$ = 1.381, $SE$ = 0.832, $Z$ = 1.660, $p$ = .097, $d$ = 0.231). Thus, although higher peak RIA did not impede individuals from benefiting from treatment, persons with low peak RIA were particularly responsive to treatment and continued to experience symptom reduction from post-treatment to 24-month follow-up, with significantly greater symptom reduction than for those with moderate or high peak RIA (figure 1). Lastly, in contrast to peak RIA results, the tensor product term of average RIA did not predict outcome ($F$ = 0.000, $p$ = 0.897, $d$ = 0.000).

## Secondary Results

As predicted, cognitive RIA was significantly positively associated with peak RIA ($r$ = 0.43, $p$ = .005, $d$ = 0.95), as was peak cognitive RIA ($r$ = 0.48, $p$ = .001, $d$ = 1.09).

There were significant fixed effects for both linear and quadratic change of RIA level over time ($t$(391) = 3.270, $p$ = .001, $d$ = 0.572 and $t$(374) = −.3164, $p$ = .002 $d$ = 0.562, respectively). The combination of these fixed effects suggested that persons tended to increase then subsequently decrease in RIA level over the course of treatment (see Figure 2). Additionally, there was a significant random effect for only the quadratic term ($\chi$2 = 9.373, $p$ = .002, $d$ = 1.089), suggesting that there were person-specific differences in the trajectory of the non-linear change of RIA level over the course of treatment. For some persons, their quadratic term was negligible (meaning that they showed a linear increase in their levels over time), and for others there was a decrease after their rise in RIA levels (Figure 2). Despite person-specific differences in the non-linear change of RIA level over the treatment, these person-specific non-linear quadratic changes did not significantly predict outcome ($F$ = 0.000, $p$ = .729, $d$ = 0.000).

Analyses of time-blocked peak RIA suggested that participants' highest level of RIA (peak RIA) reported across sessions 2 to 6 did not significantly predict outcome ($F$ = 0.362, $p$ = .203, $d$ = 0.142). However, peak RIA across sessions 7 to 10 significantly predicted outcome ($F$ = 10.227, $p$ < .001, $d$ = 1.011), showing that those with lower peak RIA demonstrated greater change than those with higher peak RIA. Likewise, peak RIA in sessions 11 to 14 significantly predicted outcome ($F$ = 9.447, $p$ < .001, $d$ = 0.972), showing that those with lower peak RIA demonstrated greater change than those with higher peak RIA. Thus, whereas peak RIA in the early phase of treatment did not significantly affect change, lower peak scores in mid-to-late treatment were associated with greater change.

Regarding CDD anxiety ratings, there were significant random effects of change in block 1 ($\chi 2 = 152.00$, $p < .001$, $d = 12.649$), block 2 ($\chi 2 = 8.898$, $p = .003$, $d = 1.053$), and block 3 ($\chi 2 = 21.20$, $p < .001$, $d = 2.069$), suggesting person-specific changes in anxiety symptoms in all 3 blocks. This finding allowed us to use person-specific changes in each block to predict peak RIA within each block. Change in block 1 CDD anxiety did not predict peak RIA in block 1 ($F = 1.511$, $p = .220$, $d = 0.389$), block 2 ($F = 0.679$, $p = .220$, $d = 0.261$), or block 3 ($F = 0.013$, $p = .909$, $d = 0.036$). Likewise, change in block 2 CDD anxiety did not predict peak RIA in block 2 ($F = 1.017$, $p = .380$ $d = 0.319$) or block 3 ($F = 0.417$, $p = .680$ $d = 0.204$). Lastly, change in block 3 CDD anxiety did not predict peak RIA in block 3 ($F = 0.678$, $p = .415$ $d = 0.260$). Thus, at no time did change in anxiety symptoms predict peak RIA, suggesting that peak RIA and preceding or concurrent change in anxiety ratings were orthogonal to one another.

## Discussion

Previous inquiries into the effect of RIA on GAD treatment have yielded inconsistent findings using approaches that failed to account for peak RIA, trajectories of RIA change, and the relationship between timing of peak RIA with outcome. In the current study we employed analyses that did account for these factors. Unlike prior papers, we also used models that accounted for the non-independence of repeated measures and allowed for both linear and nonlinear predictors. Results aligned with our predictions. Overall, across assessments those with lower peak RIA responded better to treatments containing RT compared to those with higher peak RIA. Although all participants experienced significant symptom reduction at post-treatment regardless of their peak RIA level, a non-linear interaction of time and peak RIA showed that those with lower peaks in RIA improved significantly more than those with higher peaks in RIA at post-treatment. Furthermore, from post-treatment to 24-month follow up, those with lower peak RIA continued to improve, whereas those with higher peak RIA did not experience further symptom reduction. We also found that peak RIA was independent of baseline GAD symptoms and of preceding and concurrent symptom change, suggesting that peak RIA was not epiphenomenal to symptom severity or the symptom change that occurs during treatment.

Our focus on peak RIA, as well as dividing our sample into subgroups, may have shed light on why prior studies found inconsistent results. We found that it was not so much the case that moderate or severe RIA peaks predicted negative outcome as it was that low peaks of RIA ($\leq 2$) predicted maximal benefit. Those with moderate or severe peaks in RIA still improved significantly and these individuals still maintained their gains from post-treatment to 2-year follow-up. However, the small subgroup of those without appreciable RIA—about 20% of our sample—fell into the group of those who responded ideally to treatment, especially after it had ended. Thus, finding ways to address moderate or greater RIA (a construct distinct from current symptoms and comorbidity) may be important for optimum response to GAD treatment. We also found that cognitive RIA was correlated with peak RIA. Those with greater peak RIA experienced greater levels of unpleasant, negative thoughts and images during relaxation. Thus it is possible that either inability to stop thinking about worries or the defensive use of negative thoughts or images to avoid losing control of tension during the relaxation process contributed to such peaks.

One meta-analysis found that the normative trend for PMR's effectiveness is to *increase* over time after treatment has ended (Carlson & Hoyle, 1993), as was the case with those with lower RIA. One possible interpretation of these findings is that higher peak RIA did in fact hinder participants from the usual post-treatment benefits of PMR, even if gains were maintained. Thus, finding ways to address higher peak RIA may be important for optimum response to GAD treatment. Determining possible factors that differentiate those with lower peak RIA from those with higher peak RIA may also be meaningful. Perhaps those with lower peak RIA are less driven to maintain bodily and mental control, are less vigilant of internal sensations, or less prone to forming negative RIA beliefs. In contrast to peak RIA findings, average RIA level did not predict outcome. As previously suggested, this may indicate that the most important RIA factor is the greatest surge in an individual's anxiety during relaxation. The stronger the RIA in even one session, the more salient the anxiety during internal attentional focus and the more likely it is to become associated with RT. Consistently lower RIA may be optimal. We also found that those with greater peak RIA experienced greater levels of unpleasant, negative thoughts and images during relaxation. Thus, it is possible that either inability to stop thinking about worries or the use of negative thoughts to avoid losing control of tension during relaxation contributed to such peaks.

If peaks were due to an inability to let go of thoughts/images, the well-supported theory of ironic processes of mental control may have been at work (Wegner, 1994). This theory posits that trying to suppress unwanted thoughts and bodily actions often leads to their increase. Conscious efforts to dispel worries and relax away RIA—trying to "force" relaxation—may lead to *greater* RIA. Furthermore, at least two studies have found that intentional relaxation under conditions of stress and mental load produced undesired increases in arousal—even more so than for those *not* asked to relax (Wegner, Broome, & Blumberg, 1997). When clients strive to reduce their heightened anxiety through mental control and it increases as an ironic result, they may either 1) try harder, exacerbating RIA further (i.e., "learned restlessness"; Fogle, 1978), or 2) realize that it is fruitless to attempt to suppress worrisome thoughts during RT and instead give into them (i.e., "learned helplessness"; Seligman, 1975). Seligman's (1975) theory of learned helplessness posits that an inability to volitionally control outcomes leads to a belief that one's efforts are useless. These helplessness beliefs may prevent full habituation to RIA during RT.

On the other hand, RIA has been attributed to discomfort and concern with a perceived lack of control during relaxed moments (Adler et al., 1987; Clark, 1989; Smith, 1989). Thus, some clients may not have been willing to fully let go of tension and/or negative thoughts during PMR. Instead, they may have undermined the effects of RT via negative thoughts or images and/or by not fully engaging with relaxation. According to the Contrast Avoidance Theory, clients with GAD prefer to sustain higher levels of worry, anxiety, and tension over the vulnerability associated with relaxed states (Newman & Llera, 2011). Thus, maintaining a higher level of bodily tension may feel more comfortable to these individuals; they may require direct intervention for contrast avoidance before being willing to engage fully with RT.

If any of these processes occurred, they may explain some of the results of our secondary analysis. As predicted, we found that there were two significant trends in individuals'

change in RIA level across treatment: A positive linear trajectory of increasing RIA across sessions for some and a quadratic rise and fall for others. Despite clear trends, trajectory was unrelated to outcome. Increasing linear trends in RIA can be explained by the reinforcement of RIA and RT pairings via repeated inductions—inductions perhaps shortened too soon for some participants to allow RIA to habituate completely. As noted earlier, manualized PMR sessions generally shorten over time as more sets of muscles are grouped together (i.e., more muscles are tensed and then relaxed at the same time).

The second trend we found in RIA change was an initial increase followed by a later decrease across sessions. This may be due to a response to repeated exposure to RT (i.e. between-session habituation) that those with a linear trend did not experience. However, if such quadratic trends did not lead to complete habituation of RIA, this would explain their failure to predict outcome. We found evidence for this idea with our findings that peak RIA in the first third of treatment failed to predict outcome, whereas lower peak RIA levels toward the end of therapy did. Taken together, these findings suggest that full habituation to RT for those with RIA may be crucial for maximizing treatment gains. Thus, it may not be sufficient to demonstrate a quadratic trend of change in RIA; instead it may be necessary to end treatment only when levels of anxiety are absent to negligible during RT. If RIA entrenches negative beliefs about relaxation (e.g. learned helplessness) or if positive beliefs about tension and/or worry are not directly addressed, even some eventual decreases in RIA may not optimize treatment outcome. Without habituation strong enough to evidence the falsity of such beliefs, clients may not reap the optimal benefits that relaxation can provide.

Our predictions and explanations were based in Emotional Processing Theory (EPT; Foa & Kozak, 1986). EPT posits that in order to change the fear memory, one of the mechanisms of successful exposure occurs via between-session habituation. We recognize that recently an alternative perspective focusing on anxiety variability and sustained arousal—inhibitory learning theory (ILT)—has gained popularity and research attention (Craske et al., 2008). We do not draw from ILT in this study for several reasons, the primary one being that the data we have collected were not sufficient to truly and fairly test whether the central constructs of ILT were met (i.e., we neither collected data on anxiety variability within any one relaxation session nor on final levels of fear at the conclusion of any relaxation session). Thus, we find it appropriate to neither posit nor refute mechanisms related to ILM in the interpretation of our results.

The influences of higher peak RIA and less sufficient habituation of peak RIA have several potential clinical implications. At the very least, RIA in RT for GAD should be assessed after every relaxation session. Once a client's level of RIA and trend of change is known, treatment can be tailored to best fit the individual. Recent theoretical work has underscored the possible value of personalizing interventions to individual client characteristics (DeRubeis et al., 2014; Norcross & Wampold, 2011). In such frameworks a variable that predicts differential response to treatment is first identified; then, following assessment of a client on this variable, treatment is implemented differently (i.e. optimally) based on what would best match the client's profile (DeRubeis et al., 2014). Thus, identifying a GAD client's level of RIA could lead to different treatment choices based on that information. If a GAD client reports higher peak RIA, RT sessions should not be shortened until several

sessions of minimal levels of RIA (i.e., relaxation induces little to no anxiety in the client regardless of their previous levels). Recall findings that behavioral learning via relaxation is a long-latency process (Denny, 1976). These individuals may need longer and more frequent PMR sessions and homework to allow RT to reap its full potential. It may also be helpful to incorporate non-RT techniques (such as CT) to directly target RIA. For example, therapists might question clients about any negative beliefs regarding relaxation, or positive beliefs about worry/tension and challenge these as early as is feasible. Also, therapists should assess for cognitive RIA and distraction during PMR and consider guiding clients in repetitive calming self-statements during practice, potentially making it more difficult to engage in anxious thinking. Yet for clients who show no to little RIA, perhaps repeated sessions of RT alone without CT would be enough for significant improvement. Finally, assessing and intervening on contrast avoidance may also be important. This strategy might entail cognitive restructuring of fear of negative contrast and positive beliefs about tension/worry as well as negative contrast exposure (engaging in long sessions of relaxation prior to imaginal exposure as opposed to pairing applied relaxation at the detection of anxiety from worry trigger imagery). At the same time, those with GAD who experience consistently lower RIA levels may benefit most from purely behavioral and relaxation-intensive therapies, with shortened relaxation over time, as in the current study.

It is important to note several limitations of the study at hand. First, these findings may not generalize well to broader populations. Diversity was constrained, as our sample consisted of a large majority of White and well-educated participants. Sample size was limited due to exclusion of one therapy condition from the original trial. Despite a lower sample size, we still found significant effects with large effect sizes. Nonetheless, findings should be replicated with a larger, more diverse sample. Second, one-item measures such as the one we used to assess RIA have been critiqued as being inherently unstable. However, we believe that our findings of retest reliability across 3 weeks toward the end of treatment, as well as our findings showing that there were two reliable individual change trajectories (either linear or quadratic) in RIA across the entire treatment period suggests that our measure followed patterns that were predictable and reliable across time.

Moreover, the CBT group received additional CT, and the BT group received supportive listening to control for therapist contact. Therefore, it is possible that the presence and treatment effect of RIA could have been influenced by non-RT elements for which this study cannot account. These limitations are mitigated by a lack of difference between treatment conditions in the effect of peak RIA on outcome. However, SCD was present for both compared conditions. Since most in-session progressive relaxation inductions were followed by worry trigger imagery paired with applied relaxation, it might be the case that this sequence would reinforce the association between the worry trigger and RIA as opposed to leading to counterconditioning as intended by SCD. Such reinforcement could enhance the association between peak RIA and negative treatment outcome. At the same time, it should be noted that two of the four prior studies that examined RIA as a predictor of treatment outcome included SCD in half (outcome data was combined across therapy conditions and correlated with RIA; Borkovec & Costello, 1993) or all (Borkovec & Mathews, 1988) of the treatment conditions. Despite this, there was not a specific pattern with respect to whether RIA averaged across sessions with or without SCD predicted outcome differentially. Of the

four studies, Heide and Borkovec (1983) found a relationship between RIA and a composite outcome measure immediately following one session of progressive muscle relaxation without SCD. Of the three full RCTs that examined RIA as a predictor (one with SCD in 50% of the data, one with SCD in all data, and one without SCD; Borkovec & Costello, 1993; Borkovec et al., 1987; Borkovec & Mathews, 1988), all of them found some associations between average RIA and a minority of their total outcome measures that were not very compelling. Given other differences between these studies and the method of using averaged RIA, it is difficult to know whether SCD contributed to these findings. Thus, future research should examine the impact of peak RIA and trajectories of change in RIA over time from a purely relaxation-focused intervention that entails multiple treatment sessions and ongoing homework

Future research would also do well to test whether longer PMR practices result in full habituation of RIA for those with GAD. Similarly, targeting cognitive RIA may also be important. Furthermore, later studies should pursue the reasons that peak RIA had a negative impact on outcome. Perhaps it also would be useful for future studies to test RT treatments that take peak RIA into account by extending relaxation sessions to habituate RIA, incorporating treatments that target contrast avoidance, and addressing cognitions about RIA explicitly. Lastly, exploring the factors associated with lower RIA within GAD populations may be fruitful.

Few things are more frustrating than striving toward one end and reaping the exact opposite. Now that further evidence has surfaced for the importance of RIA in GAD treatment, it may be important to address it directly in both research and practice. For clients caught in relaxation's Catch-22 of peace and peril, freedom from the cycle may make a notable difference.

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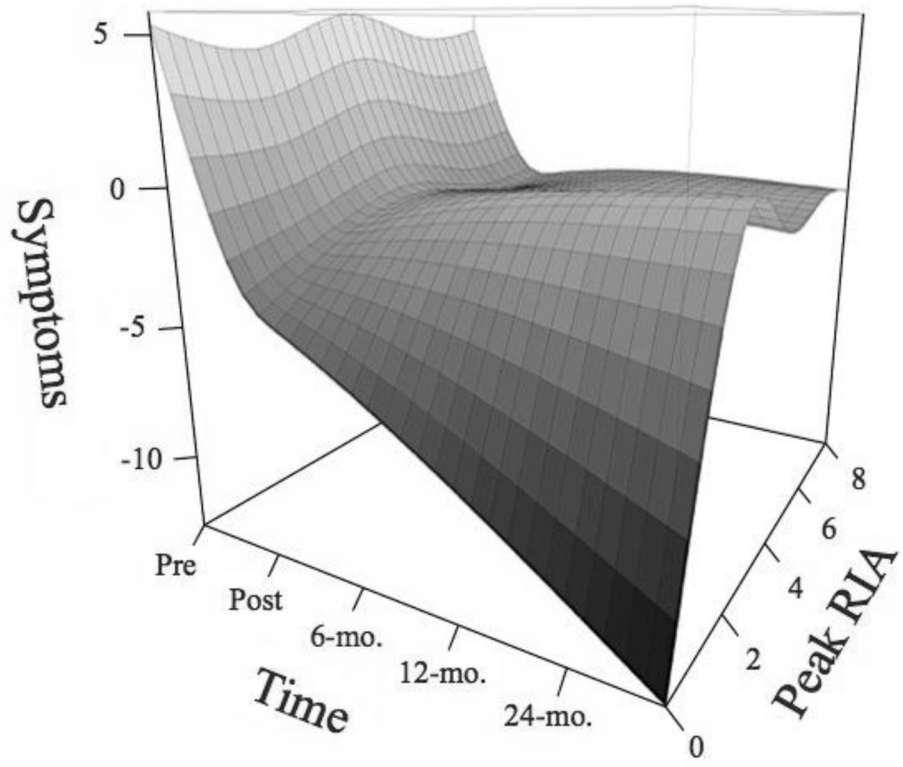


**Figure 1.**

Interaction of peak RIA and time on treatment outcome. This figure depicts the tensor product (i.e. non-linear interaction) between time and peak RIA on the composite outcome. Note that there is both non-linear change over time and non-linear moderation of peak RIA on the outcome.

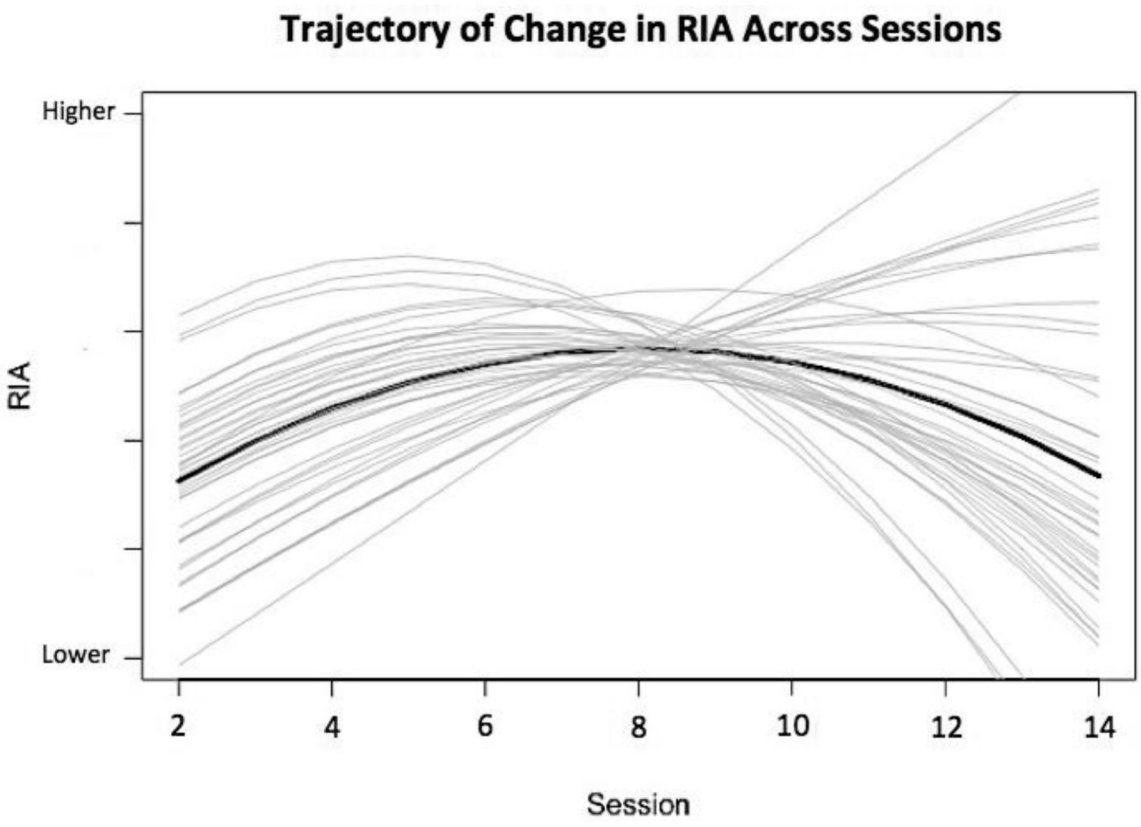


**Figure 2.**

Trajectory of change in RIA over sessions. This figure depicts both linear and quadratic change over time in RIA between sessions 2 and 14. The black line depicts the average trajectory of RIA across persons, whereas the grey lines represent the trajectory of RIA of individual clients.

## Table 1

Means and Standard Deviations of Anxiety Composite and Individual Measures at Pre-Treatment, Post-treatment, and 6-, 12-, and 24-Month Follow-Up

| | Pre-Treatment | Post-Treatment | 6-Month Follow-Up | 12-Month Follow-Up | 24-Month Follow-Up |
|---|---|---|---|---|---|
| | *M* (*SD*) | *M* (*SD*) | *M* (*SD*) | *M* (*SD*) | *M* (*SD*) |
| Composite Anxiety Score | 154.00 (16.11) | 99.91 (24.79) | 99.05 (24.22) | 101.53 (23.66) | 104.82 (25.28) |
| PSWQ | 67.62 (7.05) | 47.63 (12.49) | 46.19 (12.98) | 46.65 (11.34) | 47.84 (12.42) |
| STAI-T | 57.58 (8.09) | 41.67 (10.60) | 41.58 (9.40) | 40.97 (8.94) | 42.42 (9.89) |
| HARS | 23.29 (5.56) | 8.59 (4.97) | 9.14 (5.13) | 11.60 (7.14) | 12.36 (7.33) |
| Clinician-Rated Severity | 5.51 (0.84) | 2.02 (0.92) | 1.90 (0.94) | 2.31 (1.31) | 2.20 (1.34) |

Note. PSWQ= Penn State Worry Questionnaire, STAI-T = State-Trait Anxiety Inventory-Trait Version, HARS = Hamilton Anxiety Rating Scale.