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THE EFFECTS OF WORRY ON RESPONSE TO AN EXPOSURE INTERVENTION

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Abstract

Worry, or repetitive thought about future threat, is a cardinal feature of generalized anxiety disorder (GAD) and common across anxiety disorders. Worry could maintain pathological anxiety by interfering with emotional dynamics that promote fear extinction learning. Specifically, the contrast avoidance model states that worry heightens subjective and physiological components of fear activation and reduces the probability of subsequent increases in fear activation (Newman & Llera, 2011). The emotional processing theory states that increases in fear activation during encounters with feared scenarios are necessary for fear extinction learning (Foa & Kozak, 1986). Thus, by interfering with increases in fear activation during fear extinction (e.g., during exposure-based therapy), worry could impede the reduction of anxiety over time.

Fifty-two undergraduate students with fear of public speaking ($N = 20$ with GAD) were randomly assigned to worry ($N = 32$) or relax ($N = 20$) before completing each of four public speaking exposure exercises. Levels of subjective and physiological fear activation were assessed during resting baseline, worry and relaxation inductions, and the first minute of each exposure exercise. Levels of public speaking anxiety were assessed at pre-exposure, immediately post-exposure, and one-week follow-up.

Worry, compared to relaxation, led to a significantly greater increase in subjective distress and skin conductance from baseline levels. Moreover, worry was associated with a significantly smaller increase in subjective distress during the first minute of exposure exercises, suggesting that worry interfered with fear activation during exposure. Despite these effects, only participants in the worry condition experienced a significant decline in their public speaking anxiety from pre-exposure to immediately post exposure and one-week follow-up, and participants in the worry condition experienced a significantly greater decline in cognitions related to public speaking

anxiety from pre-exposure to one-week follow-up. The effect of worry on immediate subjective distress was only significant among participants without GAD, and greater increases in fear during exposure predicted nonsignificantly smaller reductions in public speaking over time.

Results generally supported the contrast avoidance model of worry, indicating that worry increases concurrent fear activation while reducing the probability of subsequent increases in fear activation. However, results refuted hypotheses derived from the emotional processing theory, instead suggesting that increases in fear activation during exposure do not promote anxiety reduction and that worry before exposure could promote anxiety reduction. An alternative account of the effects of worry on fear extinction, in which worry enhances the discrepancy between expectations and reality during exposure, is discussed.

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List of abbreviations

GAD: generalized anxiety disorder

SUDS: subjective units of distress scale

BAT: behavioral avoidance test

PRCS: personal report of confidence as a speaker

SSPS: self-statements during public speaking

SCL: skin conductance level

RMSSD: root mean squared successive difference

The Effects of Worry on Response to an Exposure Intervention

Chapter 1. Introduction

Worry is defined as repetitive thinking about future threat and is the central feature of generalized anxiety disorder (GAD; American Psychiatric Association, 2013). GAD is frequently comorbid with other anxiety disorders (Judd et al., 1998), and elevated worry has been documented across all anxiety disorders (Kertz, Bigda-Peyton, Rosmarin, & Björgvinsson, 2012; McEvoy, Watson, Watkins, & Nathan, 2013; Weems, Silverman, & La Greca, 2000). Thus, studying the effects of worry on the long-term maintenance of anxiety could improve models of the nature and treatment of GAD and anxiety disorders more generally. A fruitful line of inquiry could be to study worry's effects on fear extinction learning. Fear extinction involves confrontation with a conditioned stimulus in the absence of an aversive unconditioned stimulus, until a person learns that the conditioned stimulus does not reliably signal the onset of the unconditioned stimulus. Meta-analytic evidence suggests that fear extinction learning is impaired across anxiety disorders (Duits et al., 2015), and extinction learning deficits are thought to contribute to the long-term maintenance of pathological fears (Graham & Milad, 2011; Milad, Rosenbaum, & Simon, 2014). Moreover, fear extinction learning is considered a primary mechanism of exposure therapy, one of the most efficacious interventions for anxiety disorders (Olatunji, Cisler, & Deacon, 2010). Therefore, a critical question for the maintenance and treatment of pathological anxiety is what effect worry has on fear extinction learning.

The contrast avoidance model (Newman & Llera, 2011) points to a possible mechanism by which worry could impede fear extinction and maintain pathological anxiety. First, the theory proposes that worry heightens and prolongs state negative affect (e.g., Llera & Newman, 2010, 2014; Newman et al., 2019) and physiological arousal (e.g., Brosschot, Gerin, & Thayer, 2006;

Skodzik, Zettler, Topper, Blechert, & Ehring, 2016; Steinfurth, Alius, Wendt, & Hamm, 2017). Second, the theory proposes that the emotional state associated with worry reduces the degree of subsequent increases in negative affect and arousal in response to aversive stimuli (e.g., Llera & Newman, 2010, 2014; Newman et al., 2019; Skodzik et al., 2016). Therefore, the theory states that a primary function of worry is the avoidance of sharp negative emotional shifts (i.e., negative emotional contrasts; Newman & Llera, 2011). Importantly, increases in subjective and physiological fear upon confrontation with feared scenarios may be necessary for fear extinction learning. The emotional processing theory proposes that sharp increases in fear during confrontations with feared scenarios, termed *initial fear activation*, indicate that a person has retrieved his or her fear structure from memory, enabling the integration of safety information (Foa & Kozak, 1986; Kozak, Foa, & Steketee, 1988). By worrying and consequently avoiding increases in fear, people with anxiety disorders may render themselves unable to learn from the non-occurrence of anticipated negative events, either during exposure therapy or naturalistic fear encounters.

Basic and clinical research support emotional processing theory's assertion that increases in fear activation enhance corrective learning. Increases in skin conductance and heart rate in response to fearful stimuli co-occur with activity in the parietal and visual cortices, neural structures involved in attention and sensory processing (Low, Lang, Smith, & Bradley, 2008; Moratti, Keil, & Miller, 2006). Increases in emotional arousal upon confrontation with feared stimuli could therefore promote attention toward, and encoding of, extinction learning memories. Arousal also enhances memory consolidation of fear extinction learning (Berlau & McGaugh, 2006), and this effect may only occur if feared stimuli elicit increases in arousal during learning (Roozendaal & Hermans, 2017). For example, post-training administration of epinephrine

selectively enhanced memory for images that elicited increases in emotional arousal during training (Cahill & Alkire, 2003). Increases in emotional arousal during extinction may therefore also promote the consolidation of extinction learning memories, leading to greater long-term fear reduction. Accordingly, clinical studies have associated larger increases in fear during exposure therapy with more favorable outcomes (Kozak et al., 1988; van Minnen & Hagenaars, 2002). Similarly, greater variability in fear during exposure, resulting in more momentary increases in fear (Culver, Stoyanova, & Craske, 2012), and higher levels of self-reported emotional reactivity (Niles, Mesri, Burklund, Lieberman, & Craske, 2013) predicted greater anxiety reduction following exposure-based interventions. By reducing initial fear activation during learning, worry could impede the formation of fear extinction memories and long-term fear reduction.

It bears noting that some research has disputed the effects of initial fear activation on exposure outcome (Craske et al., 2008; Meuret, Seidel, Rosenfield, Hofmann, & Rosenfield, 2012), suggesting that the contrast avoidance effects of worry could be benign in the context of exposure. Furthermore, it is possible that by intensifying thought about potential catastrophes (e.g., Vasey & Borkovec, 1992), worrying before exposure could increase the mismatch between expectations and reality and *improve* fear extinction. Supporting this prediction, the inhibitory learning theory proposes that exposures can be made most effective if exposure leads to strongly violated expectations of aversive unconditioned stimuli (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). Moreover, cognitive models of anxiety construe exposure therapy as a form of behavioral experimentation, deriving its effectiveness from the mismatch between a patient's explicit predictions and observed outcomes (Salkovskis, Clark, & Gelder, 1996; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2007). Larger expectancy violation has accordingly been found to predict greater long-term fear reduction in both conditioned fear

extinction (Brown, LeBeau, Chat, & Craske, 2017) and exposure-based therapy (Guzick, Reid, Balkhi, Geffken, & McNamara, 2018). Therefore, worry before extinction learning could highlight the inaccuracy of worrisome beliefs and promote anxiety reduction.

The effect of worry on fear extinction and exposure response has been examined in a limited body of research. Gazendam and Kindt (2012) found that reading worry-related statements before conditioned fear extinction training impaired the extinction of unconditioned stimulus expectancies. Relatedly, Hazlett-Stevens and Borkovec (2001) found that participants randomly assigned to worry before exposure experienced more subjective distress across exposure trials than participants randomly assigned to relax prior to exposure. Although both of these studies suggest that worry could impede extinction learning and promote the maintenance of pathological fears, neither study used a method robust enough to strongly support this hypothesis. It is unclear to what extent the conditioned fear extinction method and written worry scripts used by Gazendam and Kindt (2012) generalize to naturalistic encounters with feared scenarios, exposure-based therapies, and idiographic worry processes. Additionally, Hazlett-Stevens and Borkovec (2001) did not administer a post-exposure or follow-up outcome measure, instead only measuring change in fear across an exposure session. Given that fear reduction during exposure may not reliably predict outcome (Craske et al., 2008), these results are insufficient to draw conclusions on the effects of worry on long-term anxiety maintenance.

Studying worry's effect on response to an exposure intervention could have implications for the nature and treatment of GAD and other anxiety disorders. Given that exposure is an analogue for naturalistic fear extinction (Foa, Huppert, & Cahill, 2006), the topic could provide insight into the effects of worry on learning from confrontations to feared scenarios in everyday life. Potential clinical implications include helping individuals with anxiety disorders understand

the long-term impact of worry and informing therapists about the optimal practice of exposure-based therapies (e.g., whether worrying before exposure is to be encouraged or discouraged). Given the high comorbidity between GAD and other anxiety disorders (Judd et al., 1998), and the presence of worry across anxiety disorders (McEvoy et al., 2013), testing the effects of worry on response to exposure could help explain the maintenance of pathological anxiety and improve existing treatments transdiagnostically.

The present study tested the effects of worry on fear activation and response to an exposure intervention for public speaking fear. Based on the contrast avoidance theory (Newman & Llera, 2011), I hypothesized (1) that worry would be associated with a greater increase in subjective and physiological fear activation from baseline levels, compared to relaxation. Additionally, based on the contrast avoidance theory (Newman & Llera, 2011), I hypothesized (2) that worry would be followed by a smaller increase in fear activation during exposure, compared to relaxation. Although the emotional processing theory (Foa & Kozak, 1986) and inhibitory learning theory (Craske et al., 2008) suggest different effects of worry on response to exposure, I hypothesized (3) based on the two existing studies documenting adverse effects of worry on fear extinction (Gazendam & Kindt, 2012; Hazlett-Stevens & Borkovec, 2001), that worrying prior to exposure would be associated with a smaller reduction in public speaking fear from pre-exposure to post-exposure and one-week follow-up, compared to relaxation.

I also conducted follow-up analyses to test whether (A) the effects of worry as outlined in Hypotheses 1-3 varied for participants with GAD vs. participants without GAD and (B) initial fear activation, defined as the change in subjective fear from pre-exposure to the first minute of exposure, mediated the effects of worry on change in public speaking fear over time. Given that worry has been found to immediately increase negative emotion and subsequently decrease the

probability of a negative emotional contrast for individuals with and without GAD (Llera & Newman, 2010, 2014), I expected Hypotheses 1 through 3 to be supported regardless of participants' GAD diagnostic status. Such a finding would suggest that the effects of the experimental worry manipulation similarly affect those who engage in frequent, trait-like worry and those who engage in less frequent worry. Additionally, because emotional processing theory considers initial fear activation necessary for response to exposure therapy (Foa & Kozak, 1986), I expected greater initial fear activation during exposure to predict greater reduction in public speaking fear and to mediate the effects of worry on exposure therapy response.

Chapter 2. Method

Participants & Screening Measures

Fifty-two undergraduates were recruited for having severe anxiety and avoidance of public speaking, as indicated by their responses to a questionnaire (Tsao & Craske, 2000) administered in an online screening survey. As an additional selection criterion, one group of participants ($N = 20$) met diagnostic criteria for GAD as outlined in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5; American Psychiatric Association, 2013), based on their responses to the Generalized Anxiety Disorder Questionnaire-IV (GAD-Q-IV; Newman et al., 2002). Another group of participants ($N = 32$) was selected for not having GAD, as indicated by not meeting diagnostic criteria for GAD and scoring below the mean GAD-Q-IV score on the screening survey.

Among all participants in the sample, 43 (82.7%) were female. Thirty-nine participants (75.0%) identified their race as white, with others identifying their race as black ($N = 5$; 9.6%), Asian ($N = 4$; 7.7%), American Indian or Alaskan Native ($N = 1$; 1.9%); or another race not

listed in the demographic questionnaire (N = 2, 3.8%). Seven participants (13.5%) identified their ethnicity as Hispanic. The average age in the sample was 18.64 years (SD = 0.85).

Self-report measures

Public speaking anxiety screen. The public speaking anxiety screen (Tsao & Craske, 2000) includes two questions (1. *How anxious would you feel giving a formal speech in front of a live audience?* 2. *How likely would you be to avoid taking a class that requires an oral presentation?)* rated on eight-point Likert scales. Participants were selected for choosing an answer of at least six on both questions. The same screening criterion has been to recruit public speaking-anxious participants in previous exposure studies (e.g., Culver et al., 2012; Shin & Newman, 2018).

GAD-Q-IV. The GAD-Q-IV assesses the complete diagnostic criteria for GAD as outlined in the DSM-5. The GAD-Q-IV has both a criterion-based scoring method that provides a binary indicator of GAD diagnostic status, as well as a continuous scoring method that provides an indicator of the severity of GAD symptoms. The continuous scoring method of the GAD-Q-IV demonstrates sensitivity and specificity in identifying individuals with GAD (Newman et al., 2002). Therefore, using the criterion-based scoring method to identify participants with GAD and the combined criterion and continuous scoring method to identify participants without GAD, as in the present study, appears to be a valid method of screening for GAD status.

Manipulation check. Participants reported their current levels of relaxation and worry on two scales ranging from 1 (*Not at all*) to 9 (*Extremely*).

Personal Report of Confidence as a Speaker, Short Form (PRCS; Hook, Smith, & Valentiner, 2008). The PRCS is a twelve-item true or false measure of public speaking anxiety.

The item content generally assesses subjective distress (e.g., *I feel disgusted with myself after trying to address a group of people*) and physiological arousal (e.g., *My hands tremble when I am on the platform*) associated with public speaking. Hook et al. (2008) reported strong factorial, convergent, and divergent validity for the PRCS.

Self-Statements During Public Speaking Scale (SSPS; Hofmann & DiBartolo, 2000). The SSPS measures thoughts experienced during public speaking. The measure includes five positive self-statements (e.g., *Even if things don't go well, it isn't a catastrophe*) and five negative self-statements (e.g., *What I say will probably sound stupid*) that correspond to cognitive features of public speaking anxiety. Participants were asked the extent to which they agree with each statement on a six-point Likert scale. The measure has strong convergent and discriminant validity (Hofmann & DiBartolo, 2000).

Subjective Units of Distress Scale (SUDS; Wolpe, 1973). The SUDS is a measure of subjective distress ranging from 0 (complete relaxation) to 100 (worst distress imaginable). SUDS is commonly used to track fear over time in exposure therapy and was used in the present study to assess fear experienced during resting baseline, worry and relaxation inductions, exposure exercises, and behavioral avoidance tests (BATs; see Procedure for details).

Physiological recording

Electrodermal activity. Electrodermal activity was measured using two reusable Ag-AgCl electrodes attached to the index and middle fingers of participants' non-dominant hands. Electrodes were connected to a Biopac MP150 isolated amplifier (Biopac Systems, Inc., Santa Barbara, CA). Average skin conductance level (SCL) in microsiemens was computed as a proxy measure of sympathetic nervous system activity.

Electrocardiogram. Electrocardiogram was measured via the Biopac MP150 isolated amplifier using three reusable Ag-AgCl electrodes attached to participants' torsos. The root mean squared successive difference in milliseconds between R waves (RMSSD) was used to indicate heart rate variability. RMSSD is thought to be a valid indicator of parasympathetic nervous system activity that is less influenced by respiration than other indicators of parasympathetic nervous system activity (Shaffer & Ginsberg, 2017), which is desirable given that the speeches in the study protocol likely affected respiration.

Procedure

In Session I, participants provided informed consent and completed the baseline questionnaires including the PRCS and SSPS. Next, participants were fitted with the physiological recording equipment and asked to sit for a five-minute acclimation period. After the final minute of the acclimation period, participants were introduced to the SUDS and prompted for a current SUDS rating. The physiological measures collected in the final minute of the acclimation period and participants' initial SUDS ratings were used as resting baseline measures.

After the acclimation period, participants completed the pre-exposure BAT. For the BAT, the experimenter told participants that they would be asked to give an impromptu speech to a recording video camera for as long as they could speak, up to three minutes. Participants were told that a member of the research team would later watch and evaluate the videos. Participants were asked to draw two slips of paper from a cup and choose the topic printed on one of the slips of paper for their speech topic (potential topics included capital punishment, gun control, immigration policy, and publicly funded healthcare). These speech topics were chosen because they may evoke strong opinions, and difficulty expressing opinions is often a concern in

treatment for social and public speaking anxiety (e.g., Hope, Heimberg, & Turk, 2010). The experimenter then turned on the camera, asked for a SUDS rating, and asked participants to begin their speeches. Participants provided SUDS ratings after each minute of the BAT, as well as immediately upon finishing the BAT.

Following the pre-exposure BAT, participants were trained in their randomly assigned induction. Inductions were assigned using simple randomization administered via Random.org. For the training period, participants listened to a one-minute voice recording with instructions on how to perform their randomly assigned induction (worry vs. relaxation). Participants in the worry condition were instructed to worry as intensely as they could about giving a speech, and participants in the relaxation condition were given instructions on slow, diaphragmatic breathing. Participants received one minute to practice their assigned induction.

After training in the worry or relaxation induction, participants advanced to the exposure exercises. The experimenter explained that the exercises were designed to help participants face their fears without attempting to avoid the feared aspects of the situation or the emotions evoked in the situation. Participants were told that the goal was to build tolerance for feared situations and strong emotions. Each exposure included four five-minute speeches on several controversial topics not included as topics for the BAT (e.g., abortion, climate change, affirmative action). Exposure exercises were completed in front of the experimenter plus an audience of two research assistants who wore lab-coats, carried clipboards, and maintained neutral expressions.

Participants were given one minute to prepare for each exposure exercise, during which they were permitted to take notes. After the preparation period, participants listened to an abbreviated version of their randomly assigned induction audio tape and then completed their randomly assigned induction for one minute. Immediately afterward, participants rated their

level of worry, relaxation, and average SUDS experienced during the induction and began their speech. Participants rated their SUDS after every minute of the speech. Following the speech, participants were given one minute to rest. They completed the sequence consisting of note-taking, induction, speech, and rest a total of four times. The entire exposure sequence lasted approximately 32 minutes.

At the end of the exposure phase, audience members left the room, and participants completed a post-exposure BAT using a new speech topic. Participants returned to the lab for Session II one week later to complete the SSPS, PRCS, and follow-up BAT using a new speech topic. Participants were then debriefed and received course credit for participation. Please see Figure 1 for a schematic of the study procedure.

Data preparation

Physiological data cleaning. Physiological data files were visually inspected for artifacts. Due to experimenter error or equipment failure, six participants had unusable electrodermal activity data, and 14 participants had unusable electrocardiogram data. Among participants with usable physiological data, files were divided into twenty-four one-minute epochs based on the period of the study (one-minute induction followed by five minutes of exposure, multiplied by four exposures). Twenty-four of 1,196 epochs of electrodermal activity data contained artifacts, and six of 962 epochs of electrocardiogram data contained artifacts. Epochs with artifact data were treated as missing.

Participant drop-out. Four participants dropped out of the study. One participant dropped out of the study immediately after the baseline BAT (i.e., before completing the experimental manipulation) and was excluded from analyses. All other drop-outs occurred

between sessions 1 and 2. These participants were retained in analyses with follow-up data treated as missing.

Worry and relaxation manipulation checks. Four participants (two in the relaxation condition; two in the worry condition) had average manipulation check scores on the target manipulation below 3 out of 9. Analyses were therefore conducted both with and without these participants, and final analyses reported in the main text included these participants. Unless otherwise noted, statistical inferences were the same regardless of whether these participants were excluded.

Data analyses

Missing data. Missing data were handled using multilevel multiple imputation implemented in the *R* package *mice* (van Buuren & Groothuis-Oudshoorn, 2011). Multilevel multiple imputation accommodates repeated-measures data such as the data in the present study (e.g., induction and exposure epochs nested within participants and pre-, post-, and follow-up assessments nested within participants; Grund, Lüdtke, & Robitzsch, 2018). Five imputed datasets were generated.

Distributional assumption checks. All dependent variables were approximately normally distributed (univariate skewness between -1 and 1), except for RMSSD (skewness = 2.53, kurtosis = 8.45). RMSSD values were therefore natural log transformed for use in analyses (resulting in skewness = 0.23, kurtosis = 0.34).

Randomization checks. Fisher's exact tests were run to test whether conditions differed in the proportion of women, the proportion of participants identifying as White, and the proportion of participants meeting diagnostic criteria for GAD. Independent samples *t*-tests were

run to test whether conditions differed in age, resting baseline physiology and SUDS, and each public speaking anxiety measure at baseline.

Manipulation checks. Average manipulation check scores were compared across conditions using independent samples *t*-tests.

Selection of SUDS and physiological measurements. Because SUDS upon completion of the BAT could reflect relief due to the offset of the speech, only the pre-BAT and mid-BAT measurements (after the first and second minutes of speaking, if applicable) were analyzed. That is, if a participant spoke for all three minutes of a BAT, his or her SUDS rating immediately before the BAT and ratings after the first two minutes of speaking were retained for analyses. If a participant stopped speaking mid-BAT, all ratings before the ending SUDS rating were analyzed. Each participant's minimum SUDS ratings from these measurements was used as an outcome measure (termed *minimum SUDS during BAT*).

To capture the immediate emotional effects of worry and relaxation, as well as the subsequent effects of worry and relaxation on initial fear activation during exposure, SUDS ratings and physiological measurements from the one-minute resting baseline period, all four one-minute induction periods, and the first minute of all four exposure exercises were retained for analyses.

Hypotheses 1 and 2: Fear activation across baseline, induction, and exposure. Multilevel models were used to evaluate change in fear activation from baseline to induction and from induction to the first minute of each exposure exercise. Fixed effects included an intercept, condition (worry vs. relaxation), baseline-to-induction time, induction-to-exposure time, and interactions between condition and baseline-to-induction time and condition and induction-to-exposure time. Random effects included a random intercept. The two-way interaction between

condition and baseline-to-induction time indicated the effect of worry (vs. relaxation) on change in fear activation from resting baseline to induction. The two-way interaction between condition and induction-to-exposure time indicated the effect of worry (vs. relaxation) on change in fear activation from induction to the first minute of each exposure exercise.

The analyses described above were conducted first using observations across all four inductions and exposure exercises (i.e., nine observations per participant, including the baseline period) and then repeated for each separate induction and exposure exercise (i.e., three observations per participant, including the baseline period). A Monte Carlo simulation study suggested that the former analysis (including observations from all trials) had 94% power to detect small-to-medium (product-moment $r = .2$) interactions between time and condition given the sample of 51 participants. However, the analyses examining each individual exposure trial had much lower power (36%) and would have required at least 148 participants for power of 80% or greater.

Hypothesis 3: Public speaking anxiety from baseline to post-exposure and follow-up. Multilevel models were used to test the effect of worry (vs. relaxation) on change in public speaking fear from baseline to post-exposure and baseline to follow-up. Fixed effects included an intercept, condition (worry vs. relaxation), time (baseline-to-post-exposure or baseline-to-follow-up), and the interaction between condition and time. Random effects included a random intercept. The power to detect a small-to-medium effect size given the observed sample of 51 participants was 36%, and a much larger sample size of 148 participants would have been required for at least 80% power. Therefore, these results should be interpreted with caution.

Follow-up analyses. ***Moderation by GAD status.*** I modified the models used to test Hypotheses 1 and 2 by adding a fixed effect of GAD status, two-way interactions between GAD

status, condition, baseline-to-induction time, and induction-to-exposure time, a three-way interaction between GAD status, condition, and baseline-to-induction time, and a three-way interaction between GAD status, condition, and induction-to-exposure time. The three-way interactions would indicate whether support for Hypotheses 1 and 2 varied as a function of GAD status. That is, did worry's immediate effect on fear activation or worry's subsequent effect on initial fear activation during exposure vary for persons with vs. without GAD?

I also modified the models used to test Hypothesis 3 by adding a fixed effect of GAD status, a two-way interaction between GAD status and time (pre-exposure to post-exposure or pre-exposure to follow-up), and a three-way interaction between GAD status, condition, and time (pre-exposure to post-exposure or pre-exposure to follow-up). The three-way interaction would indicate whether support for Hypothesis 3 varied as a function of GAD status. That is, did worry exert different effects on public speaking anxiety reduction for participants with vs. without GAD?

Initial fear activation as a mediator of worry's effect on exposure response. To test if initial fear activation mediated the effects of worry on change in public speaking anxiety from baseline to post-exposure or one-week follow-up, I modified any models used to test Hypothesis 3 in which there was a significant interaction between time and condition by adding a fixed effect of initial fear activation (defined as a participant's average change in SUDS from baseline to first minute of exposure) and an interaction between initial fear activation and time. A significant interaction between initial fear activation and time, as well as a non-significant interaction between condition and time, would indicate that initial fear activation predicted change in public speaking anxiety, and that this effect mediated the effect of condition on change in public speaking anxiety.

Effect size calculation. Cohen's *d* was calculated from *t* statistics using the formulas outlined by (Rosenthal & DiMatteo, 2001).

Chapter 3. Results

Randomization checks

Twenty participants were randomized to the relaxation condition, and 32 participants were randomized to the worry condition. There were no significant differences across conditions in the proportion of women (90% of worry condition; 75% of relax condition Fisher's exact p = .407), the proportion of White participants (80.9% of worry condition; 65% of relax condition; Fisher's exact p = .324), or the proportion of participants with GAD (41.9% of worry condition; 30% of relax condition; Fisher's exact p = .554). Conditions also did not differ significantly in age (β = -0.223, SE = 0.251, t(47) = -0.887, p = .380, d = -0.250).

At pre-exposure, participants in the worry condition scored significantly higher than participants in the relaxation condition on the PRCS (β = 1.303, SE = 0.586, t(45.8) = 2.223, p = .031, d = 0.655) and SSPS (β = 4.906, SE = 2.136, t(47.0) = 2.296, p = .026, d = 0.679) and marginally higher on minimum SUDS during the BAT (β = 7.899, SE = 4.712, t(46.0) = 0.671, p = .100, d = 0.483). Participants in the worry condition also had significantly lower resting baseline SCL (β = -1.762, SE = 0.754, t(32.1) = -2.338, p = .026, d = -0.693) and nonsignificantly lower resting RMSSD (β = -0.517, SE = 0.247, t(16.4) = -2.095, p = .052, d = -0.614). There was not a significant difference between conditions in resting baseline SUDS (β = 5.039, SE = 3.917, t(47.1) = 1.286, p = .205, d = 0.366). Thus, the randomization was successful for demographic variables, but not for baseline public speaking anxiety or resting physiology.

Manipulation checks

Descriptive statistics for manipulation checks and all other dependent variables are reported in Table 1. In the full sample, participants in the relaxation condition scored non-significantly higher on levels of relaxation than participants in the worry condition (β = -0.748, SE = 0.525, t(43.9) = -1.425, p = .161, d = -0.407), and participants in the worry condition scored nonsignificantly higher on levels of worry than participants in the relaxation condition (β = 0.883, SE = 0.478, t(46.0) = 1.850, p = .071, d = 0.536). However, as noted, both inductions resulted in medium effect sizes. These differences were statistically significant after removal of the four participants who failed manipulation checks (relaxation: β = -1.309, SE = 0.489, t(38.6) = -2.676, p = .011, d = -0.837; worry: β = 1.300, SE = 0.461, t(36.8) = 2.821, p = .007, d = 0.892).

Hypothesis 1: Effect of worry on change in fear activation from baseline

In the analysis of SUDS from baseline to induction, there was a statistically significant interaction between time and condition (β = 7.215, SE = 3.321, t(446.9) = -2.172, p = .030, d = 0.204). Please see Figure 2 for a depiction of this interaction. Simple slopes analyses revealed that participants in the worry condition experienced a significant increase in SUDS from baseline to worry induction (β = 22.105, SE = 2.076, t(449.0) = 10.647, p < .001, d = 1.145), and participants in the relaxation condition also experienced a significant yet smaller increase in SUDS from baseline to relaxation induction (β = 14.890, SE = 2.592, t(444.9) = 5.744, p < .001, d = 0.557). Moreover, SUDS ratings were significantly higher during the worry induction than during the relaxation induction (β = 12.253, SE = 5.029, t(448.2) = 2.436, p = .015, d = 0.229). Follow-up analyses from individual exposure trials suggested that the increase in SUDS from baseline was statistically significantly greater in the worry condition for the induction before first exposure exercise only, and SUDS levels differed significantly between conditions in the first

two exposure exercises only (see Table 2). Thus, SUDS results generally supported Hypothesis 1, suggesting that worry caused a greater increase in subjective fear activation from baseline to induction than relaxation, and this effect was especially robust during the induction before the first exposure exercise.

There was also a statistically significant interaction between time and condition in the model of SCL from baseline to induction (β = 0.833, SE = 0.226, t(329.8) = -3.689, $p < .001$, d = 0.350), with participants in the worry condition experiencing a significant increase in SCL from baseline to worry induction (β = 2.070, SE = 0.159, t(59.4) = 13.032, $p < .001$, d = 1.533) and participants in the relaxation condition also experiencing a significant yet smaller increase in SCL from baseline to relaxation induction (β = 1.237, SE = 0.204, t(41.2) = 6.075, $p < .001$, d = 0.591). Please see Figure 3 for a depiction of the interaction. Despite this interaction, participants in the worry and relaxation conditions did not have significantly different SCL during the inductions (β = -0.929, SE = 0.758, t(145.0) = -1.225, p = .223, d = -0.115). Follow-up analyses from individual exposure trials suggested that the interaction between time and condition was statistically significant in the second, third, and fourth exposures only (see Table 3). Therefore, SCL results lent partial support for Hypothesis 1, suggesting that worry led to a significantly larger increase in SCL from baseline than relaxation, though not to the extent that SCL during worry surpassed SCL during relaxation. The effect of worry on change in SCL effect was most robust during the latter three trials.

In the analysis of RMSSD from baseline to induction, the interaction between time and condition was not statistically significant (β = -0.109, SE = 0.182, t(17.2) = 0.599, p = .557, d = 0.056). There was not significant change in RMSSD from baseline to induction in either condition (worry: β = 0.030, SE = 0.122, t(13.2) = 0.243, p = .812, d = 0.023; relax: β = 0.138,

SE = 0.122, *t*(43.1) = 1.131, *p* = .264, *d* = 0.106). Participants in the worry condition had significantly lower RMSSD during the inductions than participants in the relaxation condition (*ß* = -0.625, SE = 0.171, *t*(249.6) = -3.654, *p* < .001, *d* = -0.346). Please see Figure 4 for a depiction of RMSSD results. The interaction was nonsignificant across all four exposure trials (see Table 4). Thus, RMSSD results did not support Hypothesis 1, and the observed difference between conditions in RMSSD during inductions could have been driven by baseline differences in RMSSD.

Hypothesis 2: Effect of worry on subsequent change in fear activation

In the analysis of SUDS from induction to the first minute of exposure, there was a significant interaction between time and condition (*ß* = -11.486, *SE* = 2.106, *t*(441.6) = -5.454, *p* < .001, *d* = -0.526). Please see Figure 2 for a SUDS levels across the induction to exposure periods. Participants in the worry condition experienced a significant increase in SUDS from worry induction to the first minute of exposure (*ß* = 2.774, *SE* = 1.313, *t*(449.0) = 2.113, *p* = .035, *d* = 0.198), and participants in the relaxation experienced a significant and larger increase in SUDS from relaxation induction to the first minute of exposure (*ß* = 14.260, *SE* = 1.646, *t*(443.5) = 8.662, *p* < .001, *d* = 0.884). SUDS ratings did not differ significantly across conditions during the first minute of exposure (*ß* = 0.768, *SE* = 5.025, *t*(449.0) = 0.153, *p* = .879, *d* = 0.014), suggesting that worry did not in any way prevent the experience of subjective distress. Follow-up analyses suggested that the increase in SUDS from induction to first minute of exposure was significantly smaller in the worry condition compared to the relaxation condition in the first and second exposure trials only (see Table 2). Thus, in accordance with Hypothesis 2, results suggested that worry lessened the increase in subjective fear activation at the start of exposure, and this finding was most robust in the first two exposure trials.

In the models of SCL and RMSSD from induction to the first minute of exposure, there were no significant interactions between time and condition (SCL: β = 0.030, SE = 0.149, t(147.7) = 0.202, p = .840, d = -0.020; RMSSD: β = 0.151, SE = 0.097, t(53.5) = 1.557, p = .146, d = 0.146). Please see Figures 3 and 4 for SCL and RMSSD levels across the induction to exposure periods. Simple slopes suggested that, from the induction to the first minute of exposure, participants in the worry condition experienced a significant increase in SCL (β = 0.436, SE = 0.090, t(285.2) = 4.831, p < .001, d = 0.463) and a significant decrease in RMSSD (β = -0.268, SE = 0.059, t(68.4) = -4.509, p < .001, d = -0.431). Participants in the relaxation condition also experienced a significant increase in SCL (β = 0.405, SE = 0.114, t(225.6) = 3.710, p < .001, d = 0.352) and a significant decrease in RMSSD (β = -0.420, SE = 0.077, t(202.6) = -5.417, p < .001, d = -0.523) from induction to the first minute of exposure. In the analysis of individual exposure trials, the increase in SCL from induction to exposure was only statistically significant in the second trial for participants in the worry condition (see Table 3). Additionally, the decline in RMSSD from induction to exposure was only statistically significant in the first and third exposure trials for participants in the worry condition and in the first, second, and third exposure trials for participants in the relaxation condition (see Table 4). Thus, the physiological results did not support Hypothesis 2, suggesting that participants in the worry and relaxation conditions experienced similar changes in fear activation from induction to the first minute of exposure.

Hypothesis 3: Effect of induction on exposure outcome

Post-exposure outcomes. In the analysis of minimum SUDS during the BAT from pre-exposure to immediately post-exposure, there was no significant interaction between time and condition (β = -6.789, SE = 4.130, t(90.2) = -1.644, p = .104, d = -0.330). Although this

interaction was nonsignificant, simple slopes analyses suggested that participants in the worry condition experienced a significant decline in minimum SUDS (β = -5.806, SE = 2.552, t(94.1) = -2.275, p = .025, d = -0.462) from pre-exposure to post-exposure, whereas participants in the relaxation condition did not experience a statistically significant decline in minimum SUDS (β = 0.983, SE = 3.247, t(86.6) = -0.303, p = .763, d = -0.060). There was no significant difference across conditions in minimum SUDS during the BAT at post-exposure (β = -1.109, SE = 4.983, t(94.1) = -0.223, p = .825, d = -0.044). Thus, contrary to Hypothesis 3, only participants in the worry condition experienced a significant decline in their public speaking anxiety as assessed during the BAT from pre-exposure to immediately post-exposure. However, this effect was not strong enough to cause a difference between conditions in minimum SUDS during the post-exposure BAT.

One-week follow-up outcomes. In the analysis of minimum SUDS during the BAT from pre-exposure to one-week follow-up, there was no significant interaction between time and condition (β = -5.034, SE = 3.516, t(91.8) = -1.432, p = .155, d = -0.287). Participants in the relaxation condition experienced a marginally significant decline in minimum SUDS during the BAT (β = -4.938, SE = 2.751, t(90.6) = 1.795, p = .076, d = -0.361), whereas participants in the worry condition experienced a significant decline in minimum SUDS during the BAT (β = -9.972, SE = 2.193, t(92.9) = -4.547, p < .001, d = -1.008). Again, there was still not a significant difference between conditions in minimum SUDS during the BAT (β = 2.970, SE = 4.398, t(92.5) = 0.675, p < .001, d = -0.134). Thus, inconsistent with Hypothesis 3, only participants in the worry condition experienced a significant decline in their minimum SUDS during the BAT from pre-exposure to one-week follow-up, but not to the extent that they experienced lower ending levels of minimum SUDS during the BAT.

In the analysis of PRCS scores from pre-exposure to one-week follow-up, there was no significant interaction between condition and time (β = -0.662, SE = 0.505, t(65.1) = -1.310, p = .195, d = -0.262). Participants in the relaxation condition experienced a significant increase in PRCS scores from pre-exposure to follow-up (β = 1.302, SE = 0.555, t(90.4) = 2.352, p = .021, d = 0.479), whereas participants in the worry condition did not significantly change in their PRCS scores from pre-exposure to follow-up (β = 0.194, SE = 0.321, t(57.7) = 0.605, p = .574, d = 0.120). PRCS scores did not differ significantly between conditions at one-week follow-up (β = 0.641, SE = 0.556, t(89.2) = 1.154, p = .252, d = 0.230). Thus, contrary to Hypothesis 3, for those in the relaxation condition, the PRCS results suggested an increase in public speaking anxiety from baseline to one-week follow-up, though the worry and relaxation conditions did not differ in ending PRCS scores.

In the analysis of SSPS scores from pre-exposure to one-week follow-up, there was a significant interaction between time and condition (β = -3.132, SE = 1.411, t(58.6) = -2.220, p = .030, d = -0.451). This interaction is depicted in Figure 5. Simple slopes analysis revealed that participants in the relaxation condition did not experience significant change in SSPS scores from pre-exposure to one-week follow-up (β = 1.371, SE = 1.071, t(73.0) = 1.281, p = .204, d = 0.256), whereas participants in the worry condition experienced a marginal decrease in SSPS scores from pre-exposure to one-week follow-up (β = -1.761, SE = 0.901, t(49.5) = -1.953, p = .056, d = -0.394). The interaction between time and condition was reduced to a trend after excluding participants who failed manipulation checks (β = -2.826, SE = 1.482, t(59.6) = -1.906, p = .061, d = -0.401), and SSPS scores did not differ significantly between conditions at follow-up (β = 1.773, SE = 2.300, t(88.1) = 0.771, p = .443, d = 0.160). Thus, contrary to expectations, participants in the worry condition experienced greater declines in SSPS scores from baseline to

follow-up. However, the absence of significant differences in ending SSPS scores and the sensitivity of this result to participants who failed the manipulation check suggests that this result should be interpreted with extreme caution.

Follow-up analyses.

Moderation by GAD status. When simple effects and interactions for GAD status were added to the model of SUDS across the baseline, induction, and exposure periods, there was a significant interaction between GAD status, baseline-to-induction time, and condition (β = 21.155, SE = 6.988, t(435.7) = 3.027, p = .003, d = -0.285). Simple slopes of this model revealed that, among participants with GAD, there was no significant interaction between condition and baseline-to-induction time (β = -6.730, SE = 5.678, t(429.3) = -1.185, p = .237, d = -0.111), whereas for participants without GAD, the original interaction between condition and baseline-to-induction time remained significant (β = 14.425, SE = 4.073, t(443.0) = 3.541, p < .001, d = 0.335). Among participants with GAD, participants in the worry condition (β = 17.904, SE = 3.167, t(443.0) = 5.653, p < .001, d = 0.547) and the relaxation condition (β = 24.590, SE = 4.678, t(437.6) = 5.256, p < .001, d = 0.506) both experienced significant and comparable increases in SUDS from baseline to induction, and SUDS did not differ significantly during the worry and relaxation inductions (β = 3.103, SE = 8.679, t(439.1) = 0.358, p = .721, d = 0.033).

Additional analyses were conducted to ascertain why the worry and relaxation inductions were not associated with different degrees of change in SUDS from baseline among participants with GAD. Simple slopes analyses indicated that participants with GAD and without GAD did not differ significantly in SUDS during relaxation (β = 7.301, SE = 8.581, t(438.9) = 0.851, p = .395, d = 0.080), or SUDS during worry (β = -6.957, SE = 6.382, t(443.0) = -1.090, p = .276, d = -0.102), suggesting that the effect of the induction on absolute SUDS did not vary as a function

of GAD status. Analysis of baseline data suggested a moderate to large, yet nonsignificant difference across the worry and relaxation conditions in baseline SUDS among participants with GAD (β = 9.833, SE = 7.133, t(15.3) = 1.379, p = .189, d = 0.667). Thus, worry was associated with a statistically greater increase in SUDS from resting baseline to induction than relaxation for participants *without* GAD only. However, it is possible that this finding was driven by baseline differences in SUDS across conditions among participants with GAD.

There were no other statistically significant three-way interactions involving GAD status (all ps > .200) in the models of fear activation across resting baseline, induction, and exposure, suggesting that the effects of worry on SUDS from induction to exposure and the effects of worry on SCL and RMSSD from baseline to induction and induction to exposure did not vary as a function of GAD status. Likewise, there were no significant three-way interactions involving GAD status in the models of public speaking anxiety from pre-exposure to post-exposure and one-week follow-up (all ps > .200), suggesting that the effects of worry on change in public speaking anxiety over time did not vary as a function of GAD status.

Initial fear activation as a mediator of worry's effect on exposure response. When the simple effect and interactions involving initial fear activation were added to the model of SSPS scores from pre-exposure to one-week follow-up, there was a trend-level and positive interaction between time and average initial fear activation (β = 0.166, SE = 0.094, t(55.8) = 1.764, p = .083, d = 0.370), and the original interaction between time and condition became non-significant (β = -1.321, SE = 1.610, t(88.2) = -0.820, p = .414, d = -0.170). Thus, although not statistically significant, experiencing a greater increase in SUDS from induction to the first minute of exposure was unexpectedly associated with somewhat smaller reduction in SSPS

scores from baseline to one-week follow-up. Additionally, this effect may partially explain the effect of worry on change in SSPS scores from baseline to one-week follow-up.

Chapter 4. Discussion

Consistent with the contrast avoidance model (Newman & Llera, 2011), the present study found that worrying, compared to relaxation, generated greater increases in subjective distress and skin conductance level from baseline levels. Moreover, also consistent with the model, worrying prior to public speaking exposure resulted in a smaller increase in subjective distress during exposure compared to relaxation prior to exposure. Notably, worrying did not prevent the experience of subjective distress, given that maximum SUDS during exposure did not differ across conditions. The physiological data, however, did not demonstrate the same pattern, with no significant difference between the worry and relaxation conditions in the degree of change in either physiological measure from induction to exposure. Thus, contrast avoidance effects observed in the present study were limited to subjective distress.

Worry's effects on immediate subjective distress and subsequent emotional reactivity generally converge with the results of previous research. As in the present study, worry has been found to increase subjective distress and reduce the probability of subsequent negative emotional contrasts in both laboratory (Llera & Newman, 2010, 2014; Skodzik et al., 2016) and naturalistic (Newman et al., 2019) studies. Moreover, the present study replicates a prior finding that worry caused increases in skin conductance level (Skodzik et al., 2016), suggesting that worrying could have increased sympathetic nervous system activity. It is somewhat surprising that worry did not lead to statistically greater declines in heart rate variability from baseline levels, given that the effects of worry on heart rate variability have been fairly robust in prior research (e.g., Ottaviani et al., 2016). It is possible, however, that the baseline differences in RMSSD observed across

conditions led to a floor effect, such that worry could not exert a strong effect on heart rate variability relative to relaxation. Another surprising finding was that the differential effects of worry and relaxation on subjective distress appeared to be limited to participants without GAD. The most salient contributor to this interaction was low resting baseline SUDS among participants with GAD in the relaxation condition, which could have enabled larger increases in SUDS over time. Given this baseline difference and the small number of participants with GAD in the relaxation condition ($N = 6$), it is possible that this result was spurious. Notably, however, persons with GAD report using worry as a coping strategy to prepare for the worst (Borkovec & Roemer, 1995) and also report relaxation to be more anxiety-inducing than persons without GAD (Kim & Newman, 2019). Thus, a very tentative substantive interpretation of this finding is that participants with GAD may have found it distressing to prepare for speeches using relaxation in the place of worry.

Contrary to the study hypotheses, participants in the worry condition generally fared better than participants in the relaxation condition in the degree of change in public speaking anxiety from over time. Only participants in the worry condition experienced a significant decline in their SUDS scores during the BAT from pre-exposure to post-exposure and one-week follow-up, and participants in the relaxation condition experienced a significant increase in PRCS scores from pre-exposure to one-week follow-up. Finally, and most notably, participants in the worry condition experienced a significantly greater decline in SSPS scores from pre-exposure to one-week follow-up compared to participants in the relaxation condition. Thus, rather than impede fear reduction over time, worry appeared to promote greater fear reduction relative to relaxation. It should be noted that the ending symptom levels did not differ significantly across conditions, suggesting that differences in symptom change may have been

driven partly by baseline differences, such that participants in the worry condition had more room to decline. Despite this caveat, given that all participants had elevated public speaking anxiety at pre-exposure, there was likely not a floor effect in the relaxation condition. Thus, the symptom change results likely indicate a substantive effect of worry before exposure on long-term anxiety reduction.

The apparently beneficial effects of worry may be best understood in light of cognitive (Salkovskis et al., 1996; Salkovskis et al., 2007) and inhibitory learning (Craske et al., 2014; Craske et al., 2008) models of exposure. Given that worry involves catastrophic thinking about possible negative outcomes (Vasey & Borkovec, 1992), worrying before exposure may have enhanced the salience of the difference between expectations (e.g., a hostile audience) and reality (e.g., a neutral audience). Greater levels of expectancy violation have been found to predict greater fear extinction learning retention in the laboratory (Brown et al., 2017) as well as more favorable response to exposure therapy (Guzick et al., 2018). Additionally, experiencing a greater percentage of untrue worries predicted greater worry reduction in a worry outcome monitoring intervention for GAD (LaFreniere & Newman, in press). Thus, participants who worried in the present study may have benefited from more striking discrepancies between anticipated and experienced outcomes during the exposures. Notably, the outcome measure on which the interaction between time and condition was strongest (SSPS) is a measure of public speaking-related cognitions. Therefore, it is possible that worrying before exposure primarily enabled participants to challenge their catastrophic public speaking-related thoughts.

Another unexpected result was that, rather than promote fear extinction, greater initial fear activation predicted nonsignificantly poorer reduction in public speaking anxiety at follow-up. Notably, recent research suggests that the ideal time course of fear activation during exposure

may not be as straightforward as initially suggested by the emotional processing theory. For example, administration of yohimbine, a drug with immediate excitatory effects on the sympathetic nervous system, one hour prior to exposure has been found to significantly improve exposure therapy outcomes relative to pill placebo (Smits et al., 2014; Tuerk et al., 2018). Relatedly, greater cortisol awakening responses on the day of exposure (Meuret et al., 2015), and greater levels of self-reported anticipatory anxiety (Noda et al., 2007) have also been found to predict superior exposure therapy outcomes. Together, these studies suggest that physiological and subjective threat activation in the minutes and hours before exposure could enhance clinical improvement. On the other hand, some research has documented nonsignificant (Benito et al., 2018) or inverse relationships (Meuret et al., 2012) between increases in fear during exposure and exposure outcome. Plausibly, increases in fear activation before exposure could promote attention to the feared situation, whereas increases in fear activation during exposure could indicate delayed attention. Thus, although basic science suggests that acute increases in emotional activation during learning promote memory encoding and consolidation (Roozendaal & Hermans, 2017), clinical research suggests that the optimal time window of threat activation may extend well before the initiation of exposure.

Several limitations of the present study should be noted. First, the sample was small and consisted entirely of undergraduates, reducing both statistical power and the generalizability of the findings to the broader population. Second, although the single-session exposure design used in the present study appears to be a valid analogue for more intensive exposure-based interventions (Rodebaugh, Levinson, & Lenze, 2013), generalizability would be improved by randomly assigning psychiatric patients to worry or relax before exposure sessions occurring in the course of psychotherapy. Third, failures in randomization (as suggested by higher baseline

public speaking anxiety and different resting physiology levels in the worry condition) and manipulation (as suggested by the presence of participants who failed manipulation check measures) hamper the interpretability of the findings. Replication in a larger clinic-based sample would likely address each of these concerns and is critical to corroborate the present findings.

If replicated, the results of the present study could have implications for both the psychopathology and treatment of anxiety disorders. The results render the chronic worry endorsed by individuals with GAD especially paradoxical, given that these individuals should repeatedly reap the benefits of worry observed in the present study. It is possible that worry in GAD arises not from perceived threat probability, but rather than from intolerance of uncertainty (Dugas, Gagnon, Ladouceur, & Freeston, 1998) or rigid avoidance of a negative emotional contrast (Newman & Llera, 2011). That is, even the slightest the possibility of experiencing an adverse outcome could constitute enough cause for individuals with GAD to use worry to prepare for the worst. In terms of clinical practice, the results may indicate that some amount of worry before exposure could enhance outcomes. This is notable given that some clinicians encourage their anxious clients to relax before or during exposures, with the rationale that such a practice may enhance coping self-efficacy (Parrish, Radomsky, & Dugas, 2008). In contrast to this recommendation, the results of this study suggest that heightened levels of anxiety before exposure may present an opportunity to disconfirm catastrophic expectations and improve anxiety over time.

In conclusion, the results of this study support the contrast avoidance model by demonstrating that worry is associated with increases in subjective distress and skin conductance level, in addition to a smaller increase in subjective distress upon confrontation with feared situations. The results failed to support the proposed adverse effect of worry and refuted the

proposed beneficial effects of initial fear activation during exposure, instead suggesting that worry may in fact improve learning from confrontations with fears. Improved expectancy violation is a possible mechanism for the observed beneficial effects of worry, though this proposition warrants confirmation using direct assessment of expectancy violation. Finally, replication in a larger clinic-based sample is necessary to replicate the present study's findings and translate them to clinical practice.

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APPENDIX A: TABLES

Table 1

Descriptive Statistics by Condition

	Relaxation		Worry	
	M	*SD*	*M*	*SD*
SUDS during resting baseline	14.80	8.32	19.84	16.15
SUDS during induction	29.37	16.71	41.94	20.51
SUDS during first minute of exposure	43.95	20.76	44.72	21.06
RMSSD during resting baseline	4.07	0.75	3.50	0.63
RMSSD during induction	4.20	0.64	3.56	0.64
RMSSD during first minute of exposure	3.79	0.63	3.28	0.61
SCL during resting baseline	6.13	2.64	4.25	2.31
SCL during induction	7.29	2.36	6.32	2.50
SCL during first minute of exposure	7.72	2.39	6.69	2.50
Pre-exposure minimum BAT SUDS	26.84	9.89	34.52	19.21
Post-exposure minimum BAT SUDS	27.60	18.06	28.71	18.64
Follow-up minimum BAT SUDS	21.21	11.67	23.64	15.25
Pre-exposure PRCS	7.30	2.15	8.54	1.95
Follow-up PRCS	8.16	1.61	8.79	1.85
Pre-exposure SSPS	19.84	6.44	25.03	8.02
Follow-up SSPS	21.53	6.68	23.64	9.39

Note. $N = 51$. SUDS = Subjective units of distress. RMSSD = natural log-transformed root mean squared successive difference, measured in milliseconds. SCL = skin conductance level, measured in microsiemens. BAT = Behavioral avoidance test. PRCS = Personal report of confidence as a speaker. SSPS = Self-statements during public speaking.

Table 2

Results from Models of SUDS Across Individual Exposure Exercises

	Exposure 1		Exposure 2		Exposure 3		Exposure 4	
Fixed effect	*ß*	*SE*	*ß*	*SE*	*ß*	*SE*	*ß*	*SE*
Condition X baseline to induction time	**13.53**	4.95	8.20	5.20	3.99	5.27	3.14	5.35
Condition X induction to exposure time	**-15.76**	4.95	**-13.37**	5.20	-7.26	5.27	-9.55	5.35
Baseline to induction (worry)	**29.23**	3.10	**22.16**	3.22	**19.19**	3.30	**17.84**	3.35
Baseline to induction (relaxation)	**15.70**	3.86	**13.96**	4.08	**15.20**	4.11	**14.70**	4.17
Induction to exposure (worry)	1.19	3.10	2.42	3.22	4.39	3.30	3.10	3.35
Induction to exposure (relaxation)	**16.95**	3.86	**15.79**	4.08	**11.65**	4.11	**12.65**	4.17
Worry vs. relaxation (induction)	**18.56**	5.05	**13.24**	5.16	9.03	5.34	8.18	5.40
Worry vs. relaxation (exposure)	2.81	5.05	-0.13	5.09	1.77	5.34	-1.38	5.40

Note. $N = 51$. Estimates printed in bold are statistically significant at $p < .05$.

Table 3

Results from Models of SCL Across Individual Exposure Exercises

	Exposure 1		Exposure 2		Exposure 3		Exposure 4	
Fixed effect	*β*	*SE*	*β*	*SE*	*β*	*SE*	*β*	*SE*
Condition X baseline to induction time	0.64	0.37	**0.76**	0.36	**0.93**	0.37	**1.00**	0.41
Condition X induction to exposure time	0.02	0.40	0.12	0.35	-0.07	0.36	0.08	0.41
Baseline to induction (worry)	**2.06**	0.24	**2.06**	0.24	**2.07**	0.23	**2.09**	0.27
Baseline to induction (relaxation)	**1.42**	0.30	**1.30**	0.29	**1.14**	0.30	**1.09**	0.35
Induction to exposure (worry)	0.39	0.23	**0.53**	0.22	0.33	0.23	0.51	0.26
Induction to exposure (relaxation)	0.41	0.31	0.40	0.28	0.39	0.29	0.42	0.32
Worry vs. relaxation (induction)	-1.12	0.78	-1.00	0.75	-0.83	0.75	-0.76	0.81
Worry vs. relaxation (exposure)	-1.14	0.78	-0.88	0.74	-0.90	0.76	-0.68	0.78

Note. $N = 51$. Estimates printed in bold are statistically significant at $p < .05$. SCL = skin conductance level, measured in microsiemens.

Table 4

Results from Models of RMSSD Across Individual Exposure Exercises

	Exposure 1		Exposure 2		Exposure 3		Exposure 4	
Fixed effect	*ß*	*SE*	*ß*	*SE*	*ß*	*SE*	*ß*	*SE*
Condition X baseline to induction time	-0.26	0.22	-0.15	0.21	-0.01	0.20	-0.04	0.21
Condition X induction to exposure time	0.24	0.19	0.33	0.18	-0.04	0.16	0.08	0.18
Baseline to induction (worry)	-0.18	0.15	0.00	0.14	0.16	0.13	0.14	0.13
Baseline to induction (relaxation)	0.07	0.16	0.15	0.15	0.14	0.13	0.18	0.14
Induction to exposure (worry)	**-0.37**	0.13	-0.20	0.12	**-0.32**	0.10	-0.18	0.11
Induction to exposure (relaxation)	**-0.61**	0.16	**-0.53**	0.14	**-0.27**	0.12	-0.26	0.16
Worry vs. relaxation (induction)	**-0.78**	0.22	**-0.67**	0.20	**-0.50**	0.20	**-0.55**	0.19
Worry vs. relaxation (exposure)	-0.54	0.22	-0.34	0.20	**-0.54**	0.22	**-0.48**	0.22

Note. $N = 51$. Estimates printed in bold are statistically significant at $p < .05$. RMSSD = natural log transformed root mean squared successive difference between R waves, measured in milliseconds.

Table 5

SUDS Across Baseline, Induction, and Exposure Among Participants With and Without GAD

	Baseline		Induction		First minute of exposure	
	M	*SD*	*M*	*SD*	*M*	*SD*
Non-GAD						
Relax	16.79	9.32	27.5	16.4	42.38	21.81
Worry	19.72	15.86	44.86	16.47	49.10	18.79
GAD						
Relax	10.17	0.41	33.91	16.92	47.62	17.95
Worry	20.00	17.20	37.90	24.67	38.65	22.66

Note. $N = 51$. GAD = generalized anxiety disorder. SUDS = subjective units of distress.

APPENDIX B: FIGURES

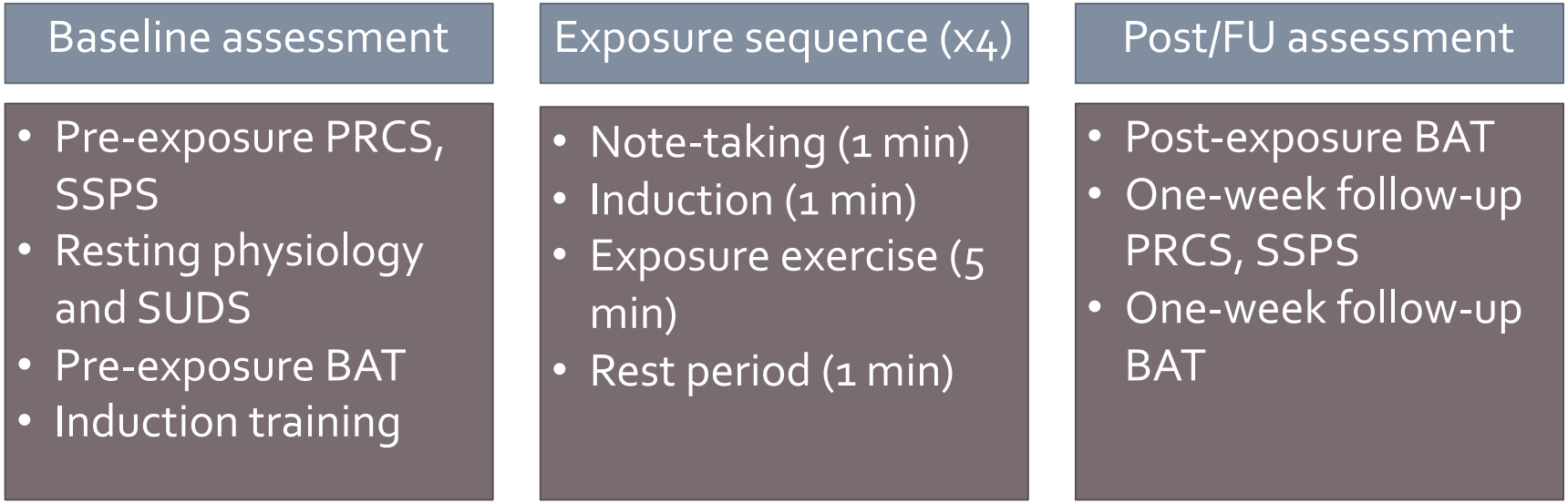

Figure 1. Schematic of study procedure.

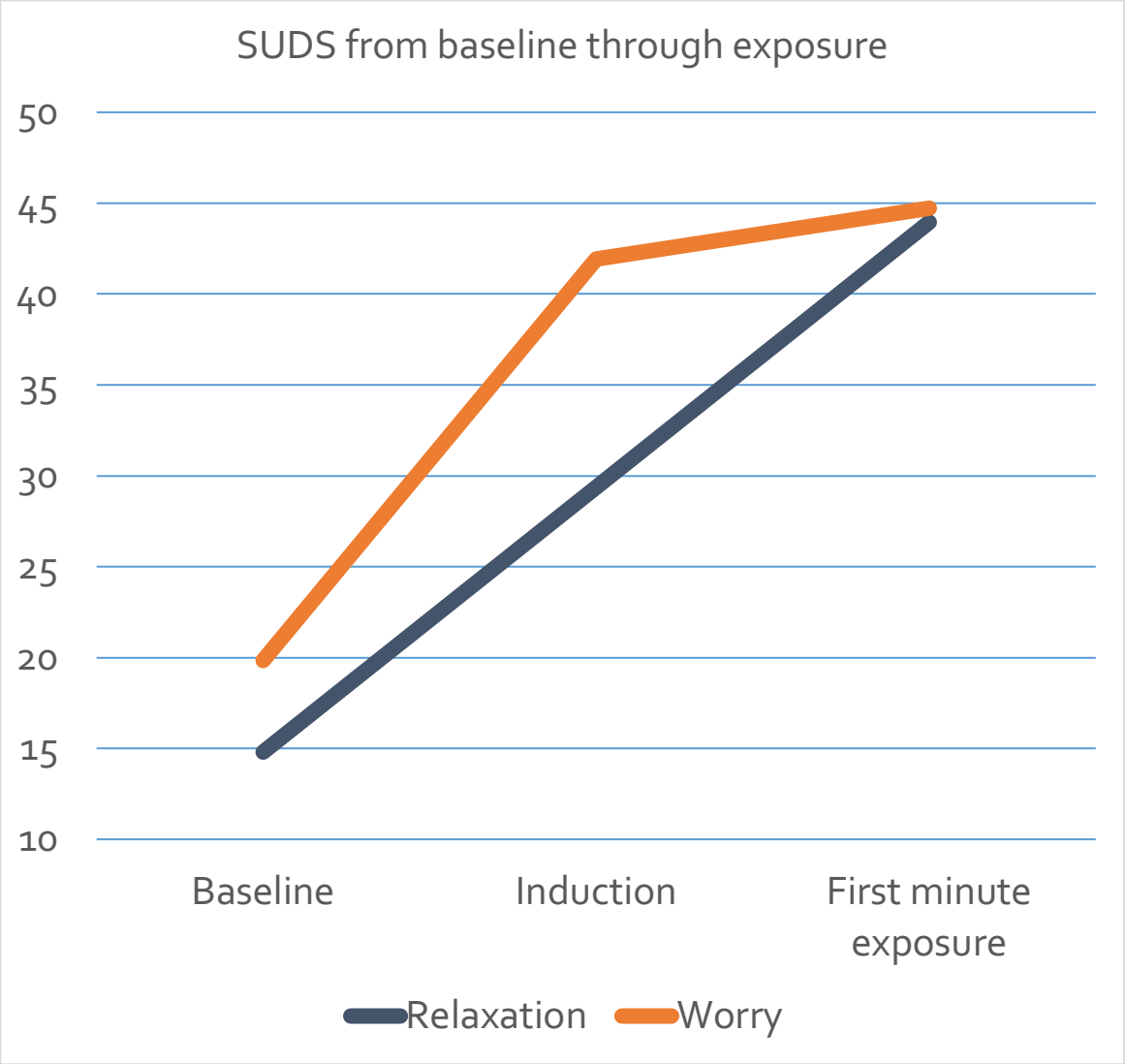

Figure 2. Subjective units of distress from resting baseline through inductions and exposure exercises across the relaxation and worry conditions.

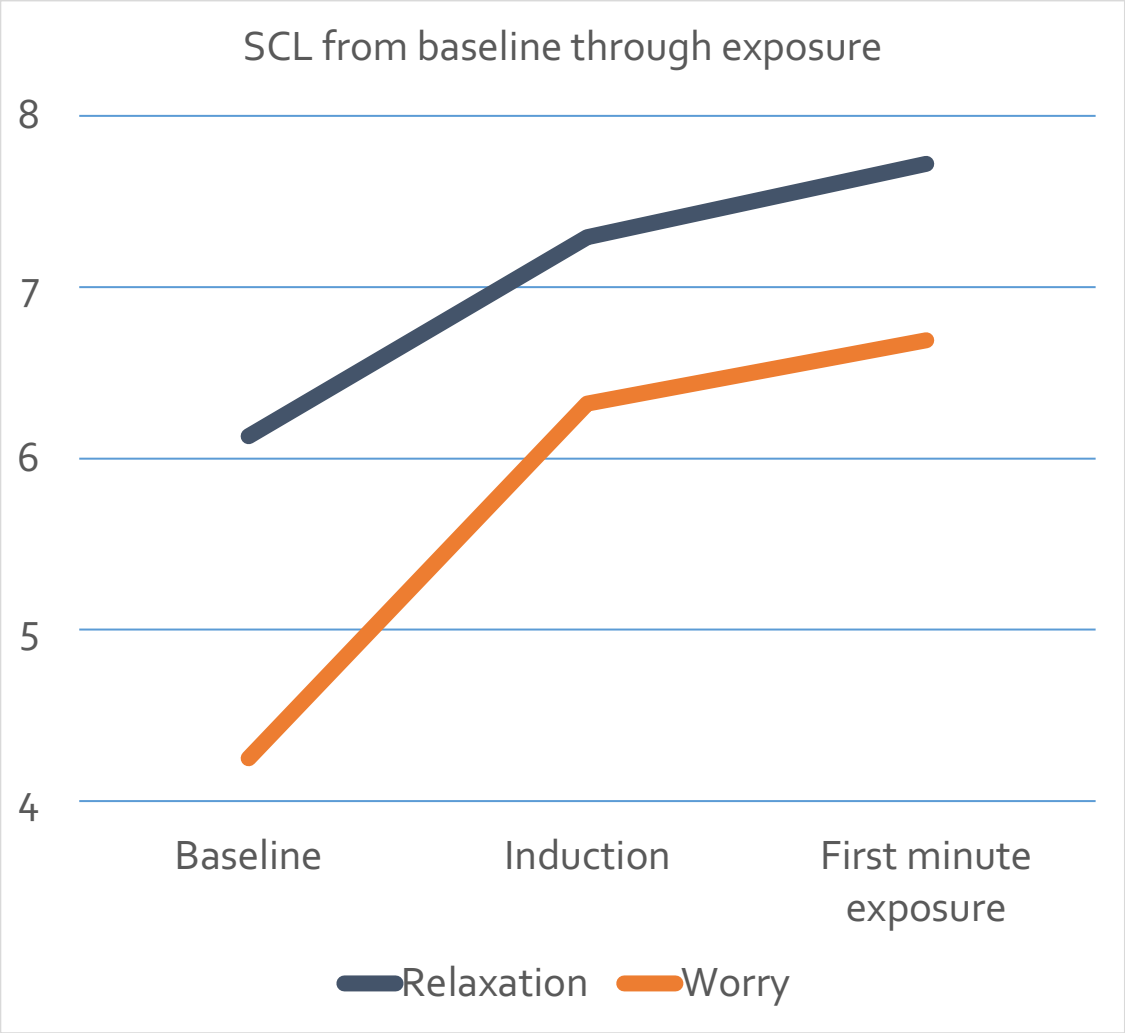

Figure 3. Skin conductance level (in microsiemens) from resting baseline through inductions and exposure exercises across the relaxation and worry conditions.

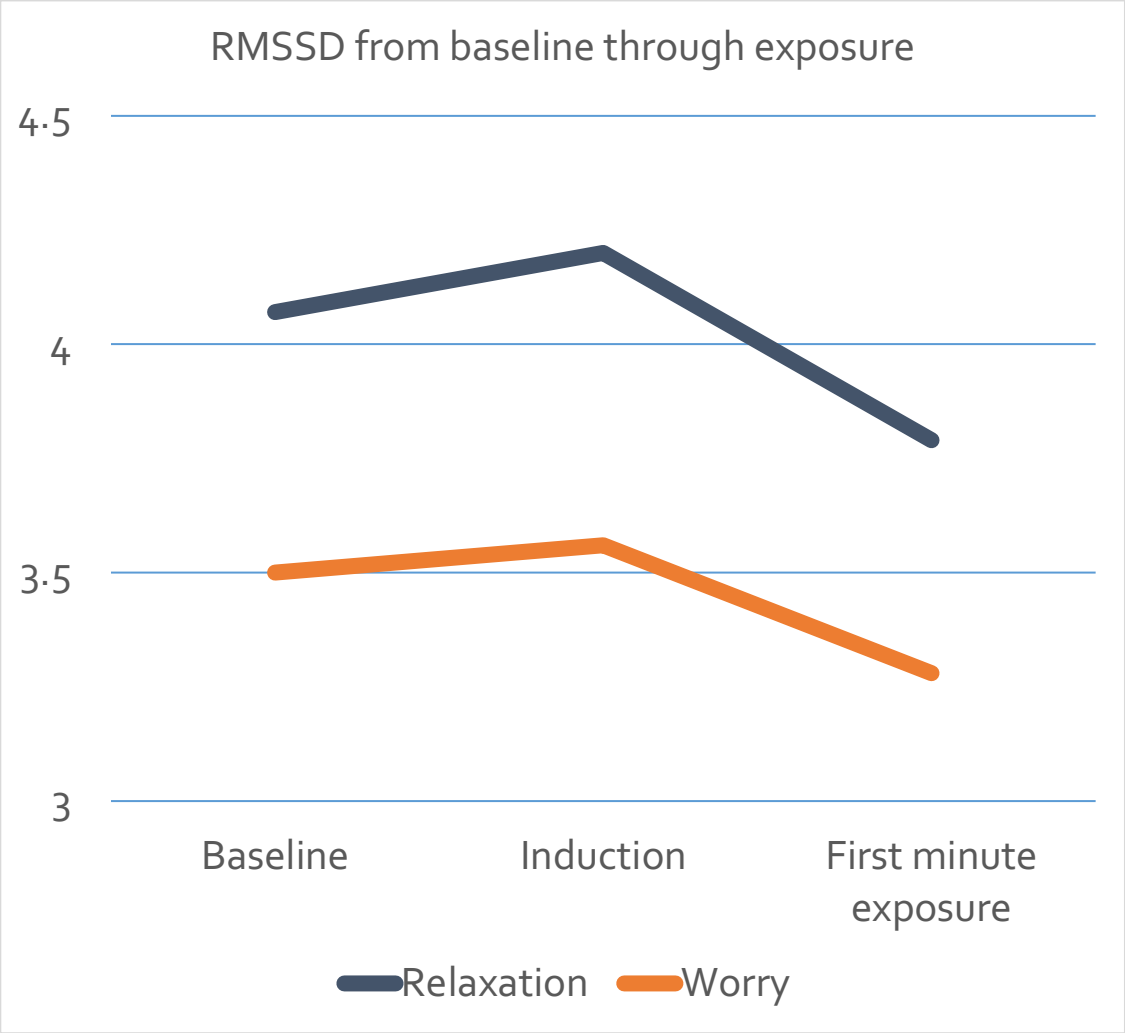

Figure 4. Natural log-transformed root mean squared standard difference in electrocardiogram R waves (in milliseconds) from resting baseline through inductions and exposure exercises across the relaxation and worry conditions.

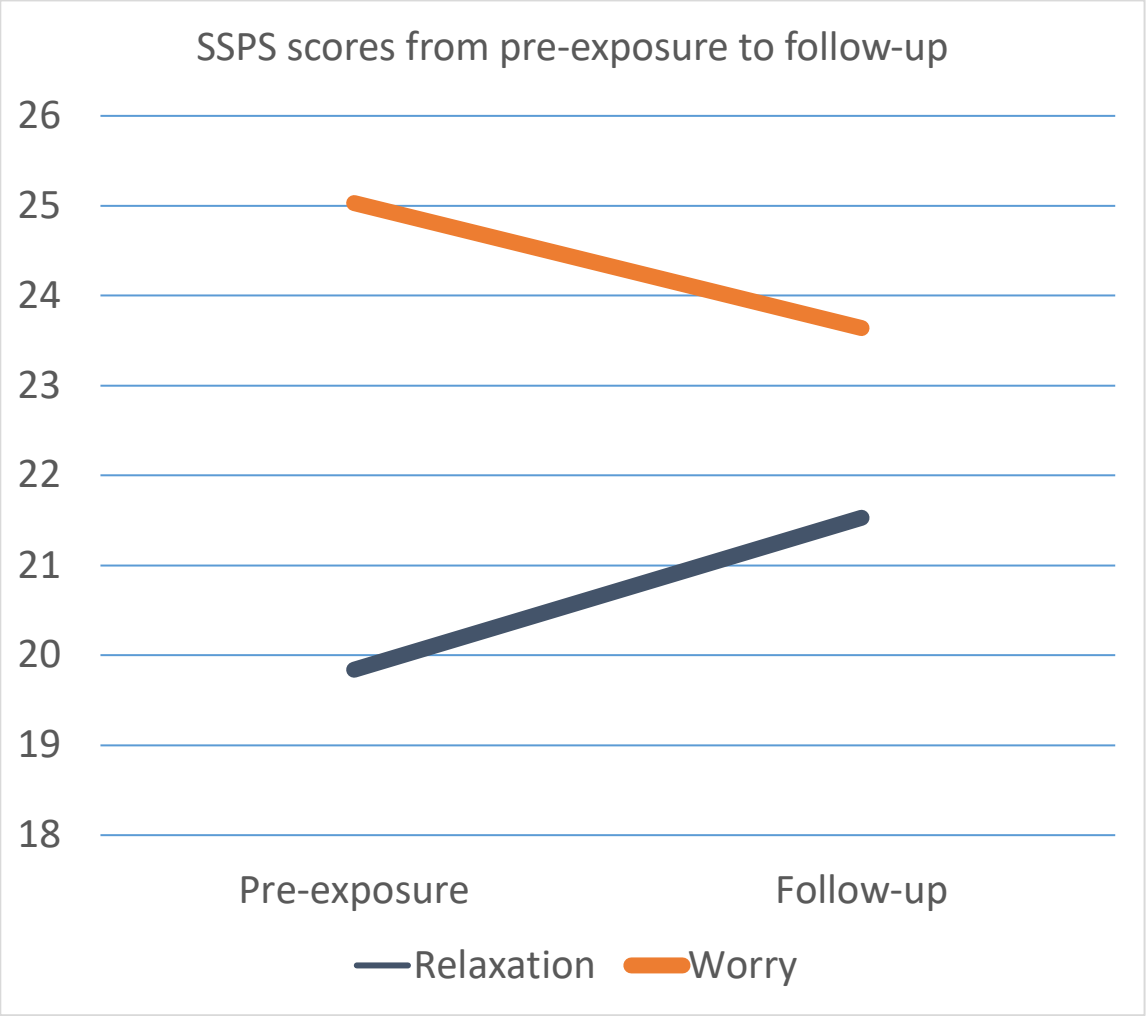

Figure 5. Self-statements during public speaking scores from pre-exposure to one-week follow-up in the relaxation and worry conditions.