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Does affect labeling enhance exposure effectiveness for public speaking anxiety?

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Does Affect Labeling Enhance Exposure Effectiveness for Public Speaking Anxiety?

A dissertation submitted in partial satisfaction of the requirements for the degree of Doctor of Philosophy in Psychology

by

Andrea Nicole Niles

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ABSTRACT OF THE DISSERTATION

Does Affect Labeling Enhance Exposure

Effectiveness for Public Speaking Anxiety?

by

Andrea Nicole Niles

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2016

Professor Michelle Craske, Chair

Fear of public speaking is common and can cause significant impairment in work and educational functioning. Exposure is an effective treatment for public speaking anxiety but approximately half of participants fail to respond fully and are in need of additional strategies. Functional neuroimaging, psychophysiology, and behavioral data provide evidence that affect labeling (labeling one's emotional experience) is a promising approach for enhancing emotion regulation, and evidence from spider fearful subjects suggests that combining exposure with affect labeling may enhance long-term fear reduction. The aim of the current project was to build on existing research by examining whether affect labeling enhances the efficacy of exposure in participants with public speaking anxiety. Participants were randomly assigned to

exposure with or without affect labeling. Physiological arousal, self-reported fear, and avoidance behavior were assessed before and after exposure and were compared between the two groups. Consistent with hypotheses, participants assigned to the affect labeling group showed greater reduction in physiological activation following exposure than did participants in the Control group. Participants who used more labels during exposure showed greater reductions on physiological measures. Hypotheses were not supported for self-report measures on which participants in the affect labeling group showed less benefit than did participants in the control group. Greater incidental emotion regulation deficits at baseline predicted more benefit from exposure combined with affect labeling than from exposure alone. The current research supports the theory that affect labeling can enhance exposure effectiveness for physiological measures of anxiety. These findings provide further evidence that targeting prefrontal-amygdala circuitry in anxiety patients using tasks that activate key regions involved in emotion regulation can improve treatment effectiveness, and that such interventions will be particularly effective for patients who show the greatest incidental emotion regulation deficits.

The dissertation of Andrea Nicole Niles is approved.

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1. Tsai, W., Lau A. S., Niles, A. N., Coello, J., Lieberman, M. D., Ko, A. C., Hur, C., Stanton, A. L. (In Press). Cultural differences in self-enhancement and self-improvement motivations during expressive writing.
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7. Niles, A. N., Lebeau, R. T., Liao, B., Glenn, D. E., & Craske, M. G., (2012). Dimensional indicators of generalized anxiety disorder severity for DSM-V. *Journal of Anxiety Disorders, 26*(2), 279-286.

Chapter 1

Introduction

Although behavioral treatments for anxiety disorders are highly effective in reducing symptoms of anxiety (Butler, Chapman, Forman, & Beck, 2006; Hofmann & Smits, 2008; Tolin, 2010), many patients do not improve, drop out of treatment, or relapse (Loerinc, Meuret, Twohig, Rosenfield, & Craske, Submitted for Publication). Exposure in particular is thought to be the most potent treatment for anxiety disorders and is believed to be the clinical proxy of extinction training. However, findings from basic science research on fear conditioning and enhancing extinction of fear in laboratory studies have not made their way into mainstream anxiety disorder treatment. Given the need to improve treatments, and the major gains made in basic science research on how to enhance fear extinction, the goal of the current project is to bridge the gap between basic science research and intervention research by testing methods for enhancing the effectiveness of exposure therapy in public speaking anxiety. Specifically, this project will compare exposure alone to exposure plus affect labeling on anxiety during public speaking.

Public speaking anxiety is one of the most common psychological disorders in the United States with prevalence estimates ranging from 11% to 30% of the population (Pollard & Henderson, 1988; Stein, Walker, & Forde, 1996; Wittchen, Stein, & Kessler, 1999). For some people, public speaking anxiety is debilitating, particularly in environments in which giving presentations in front of an audience is essential to academic or occupational success. In a community sample, public speaking anxiety was associated with lower income, less education and higher unemployment rates (Stein et al., 1996). Current treatments for public speaking

anxiety combine traditional exposure (e.g. practicing public speaking), with cognitive restructuring in which patients are taught to think about the feared situation neutrally or positively rather than negatively (Heimberg, 2002; Hofmann & Smits, 2008; Hope, Heimberg, Juster, & Turk, 2000; Rapee & Heimberg, 1997). Another approach to regulating emotion that has recently garnered interest from researchers is affect labeling, or labeling one's emotions in the face of a feared stimulus (Lieberman et al., 2007). There is some evidence to suggest that affect labeling can improve exposure effectiveness (Kircanski, Lieberman, & Craske, 2012). Although exposure alone appears to be an effective treatment for social anxiety disorder (Feske & Chambless, 1995), no studies have evaluated whether affect labeling augments the effectiveness of exposure for public speaking anxiety.

1.1. Fear and Social Anxiety Disorder

The experience of fear is an adaptive mechanism found in many species and helps an organism respond to threats in the environment. Fear is characterized by autonomic activity, subjective experience of distress, and behavioral responses. While one of the most common experiences of fear occurs in the context of threat to the physical self (e.g. possibility of pain or injury), humans can also experience an anxiety response during social threat (e.g. possibility of rejection) (Dickerson, Gruenewald, & Kemeny, 2004; Dickerson & Kemeny, 2004). In fact, the same neural regions activated by physical pain are activated following social rejection (Eisenberger & Lieberman, 2004). Maintaining social connections with others is one of the strongest human motivations (Baumeister & Leary, 1995), which suggests that doing so may have been adaptive for survival in early humans. Rejection by one's group may have posed a significant threat to survival in an evolutionary context, and therefore, humans likely evolved mechanisms to avoid being rejected from social groups. Therefore, the experience of anxiety,

fear, or shame in situations during which rejection is possible is an adaptive response to social threat.

For many people, anxiety associated with social threat is excessive and interferes with the ability to interact with others and perform in social contexts. Anxiety of this magnitude is diagnosed as social anxiety disorder. Excessive anxiety that occurs only during public speaking or performance situations is categorized as non-generalized social anxiety and is far more common than the generalized subtype in which anxiety occurs in multiple social contexts (Pollard & Henderson, 1988). Speaking in front of an audience is the epitome of social threat because the attention of the audience is focused solely on the speaker, and there is the possibility that the audience will dislike or disagree with what the speaker presents. Frequently, those viewing the speech are peers, colleagues or superiors who may be important to one's social network, future career, or educational success. The high prevalence of public speaking fears may be better understood given the potential for negative evaluation inherent in public speaking.

1.2. Indices of Fear Responding

The defensive motivational system (Masterson & Crawford, 2010) is a system of behaviors activated in response to environmental threat. Defensive responding includes autonomic activation, subjective discomfort, and avoidance behavior. The amygdala is a key region in the experience of and response to threat and has been identified as the mediator between detection of and response to threat (LeDoux, 2000). In addition, the amygdala plays an important role in fear learning, memory and extinction (Knight, Smith, Cheng, Stein, & Helmstetter, 2004; Öhman & Mineka, 2001). Patients with social anxiety disorder show greater amygdala activity in response to social threat (e.g. angry faces) (Stein, Goldin, Sareen, Zorrilla,

& Brown, 2002), as well as non-social negative stimuli (Brühl et al., 2011; Shah, Klumpp, Angstadt, Nathan, & Phan, 2009).

1.2.1. Autonomic. The experience of threat in the environment activates the sympathetic nervous system, which prepares the body and mind to respond to threat. Sympathetic activation is generally thought of as the "fight or flight" response, and includes changes in heart rate (HR) and sweat gland activity or galvanic skin response (GSR). HR and GSR have been shown to increase in response to an unconditioned threat stimulus (Deane, 1969) and during public speaking (Beatty & Behnke, 1991; Myers, 1974); however decreases in HR (fear brachycardia) also occur in response to threat due to increased attentional focus (Bradley, Codispoti, Cuthbert, & Lang, 2001). Autonomic activity during laboratory stressors has also been shown to decrease following treatment for panic disorder (Craske, Lang, Aikins, & Mystkowski, 2005). GSR in particular is a measure of sympathetic activation as sweat gland activity is stimulated by the sympathetic nervous system. HR receives input from the sympathetic and parasympathetic nervous systems, and therefore, while increases in HR indicate sympathetic activity, HR is not a pure measure of sympathetic nervous system activation (Hugdahl, 1995).

1.2.2. Self report. Fear responding also includes an individual's subjective response such as distress and negative cognitions in feared situations. Individuals with social anxiety report higher levels of anxiety and more negative cognitions during public speaking than non-anxious controls (Hofmann & DiBartolo, 2000; Levin et al., 1993). In addition, self-reported anxiety and negative cognitions decrease after completion of treatment (Craske et al., In Press) and over the course of treatment (Niles, Mesri, Burklund, Lieberman, & Craske, 2013). Given that subjective experiences of anxiety and distress are one of the primary targets in treatment, self-report is an essential indicator of fear responding.

1.2.3. Behavior. Although physiological and cognitive experiences during threat help an individual respond appropriately (e.g. fight or flight), anxiety disorders are characterized by maladaptive avoidance of feared stimuli and situations including escape, retreat, caution or thought suppression (Craske, 2003). Avoidance can negatively impact work or academic performance, social relationships, or other important domains, and immediate decreases in autonomic arousal and subjective discomfort as a result of avoidance reinforce avoidance over the long term.

1.3. Importance of Enhancing Exposure Effectiveness

One of the key ingredients in treatment for social anxiety disorder is repeated exposure to feared and avoided social situations (Feske & Chambless, 1995; Rodebaugh, Holaway, & Heimberg, 2004). Exposure treatment is thought to be analogous to fear extinction in laboratory studies in which subjects are conditioned to fear a cue that signals the onset of a threatening stimulus, then subsequently exposed to the cue in the absence of the threat (Bouton, 1993). With repeated exposure to the cue, subjects show a reduction in fear over time (Watson, 1970). However, while exposure therapy is highly effective in the treatment of social anxiety disorder, many patients do not complete treatment, do not benefit from exposure, or show a return of fear after completion of behavioral treatment (Loerinc et al., Submitted for Publication). Therefore, there is a need to enhance the effects of exposure with the ultimate goal of improving treatment outcomes and preventing relapse.

Laboratory studies of fear extinction in animals and humans have also shown a resurgence of fear after fear has been extinguished (Craske & Mystkowski, 2006; Ricker & Bouton, 1996). This can occur simply after a period of time (Bouton, Woods, Moody, Sunsay, & García-Gutiérrez, 2006), after re-experiencing of the feared stimulus or reinstatement

(Rescorla & Heth, 1975) and exposure to a different context or renewal (Bouton, 1993). These phenomena as observed in laboratory studies likely mirror relapse in humans and highlight a need to find ways of enhancing exposure effectiveness to ultimately reduce relapse rates (Craske & Mystkowski, 2006).

1.4. Using Language to Enhance Exposure Effectiveness

Although studies of extinction in laboratory animals may simulate exposure for anxiety disorders, humans have the ability to use language to regulate emotional experiences in ways that other animals do not. Language is generally used alongside exposure in an attempt to help patients better understand why the situation is feared and to create more realistic interpretations of the situation. Although behavioral treatments generally include a cognitive component, whether this linguistic strategy actually improves the effectiveness of extinction has not been experimentally assessed in controlled laboratory trials.

Lieberman's disruption theory of language and emotion (Lieberman, 2003, 2011) posits that labeling one's emotional state can disrupt the experience of the emotion. Research in functional neuroimaging, psychophysiology, and behavior provide evidence that affect labeling is a promising approach for enhancing emotion regulation during exposure. Linguistic processes activate areas of the prefrontal cortex, such as the right ventrolateral prefrontal cortex (RVLPFC) and the medial prefrontal cortex (MPFC), which corresponds to decreases in activation of limbic emotional response regions such as the amygdala. Activation of the amygdala in turn affects physiological arousal and subjective experiences of emotion (Kim et al., 2011), and therefore reductions in amygdala activation should correspond with decreases in autonomic arousal and subjective experience of distress.

Studies of the neural circuitry underlying extinction suggest that the extent to which prefrontal regions are activated, and the strength of connectivity between prefrontal regions and the amygdala affect the success of extinction training. Electrical stimulation of the mPFC led to reduction of conditioned responding after fear conditioning in rats (Milad & Quirk, 2002). Greater cortical thickness in the mPFC is associated with better extinction effects in humans (Hartley, Fischl, & Phelps, 2011; Milad et al., 2005), and greater mPFC activity is associated with better extinction effects of learned US-CS associations (Delgado, Nearing, LeDoux, & Phelps, 2008; Milad et al., 2005; Phelps, Delgado, Nearing, & LeDoux, 2004). These findings suggest that down regulation of amygdala activity by mPFC is responsible for successful extinction. In addition, evidence suggests that patients with social anxiety disorder have weaker connectivity between the mPFC and the amygdala (Hahn et al., 2011; Kim et al., 2011). Therefore, treatments that strengthen connectivity between these regions may prove beneficial in the treatment of social anxiety. The principle of neural plasticity states that repetition of a process can increase efficiency and efficacy of that process through changes in neuron function, chemical profile, and structure (Anderson, 2010; Kandel & Schwartz, 1982). Therefore, verbal processing may enhance connectivity in PFC-Amygdala pathways therefore improving patients' ability to regulate emotional responses.

1.4.1. Methods of verbalization to activate the RVLPFC. Verbalization such as cognitive reappraisal is emphasized in treatments for anxiety disorders (Craske, Antony, & Barlow, 2006; Novalis, Rojcewicz, & Peele, 1993). Reappraisal is a deliberate attempt to reduce emotional responding by generating neutral or positive (as opposed to negative) interpretations of a situation. Reappraisal is consistent with cognitive restructuring in cognitive behavioral therapies for anxiety disorders in which patients are taught to think more flexibly about feared

stimuli. Affect labeling, another method of verbalization, is the process of identifying and labeling emotional experiences (Lieberman, 2011; Pennebaker, 1997), and has been shown to reduce affective responding to negative stimuli (Lieberman, Inagaki, Tabibnia, & Crockett, 2011). It can be argued that negative thoughts and emotions must first be identified before they can be modified, so affect labeling is also the first step to cognitive reappraisal (Arch & Craske, 2008). Affect labeling has been categorized as an incidental emotion regulation strategy because affective change is a consequence of labeling but not an explicit goal (Burklund, Creswell, Irwin, & Lieberman, 2014). Although both these methods of emotion regulation have been shown to reduce affective responding to unpleasant stimuli (Lieberman et al., 2011), only one study to date has examined whether using affect labeling and reappraisal during exposure results in greater fear reduction at retest compared to exposure alone.

Kircanski, Lieberman & Craske (2012) compared the effects of exposure to a live spider with and without linguistic processing in spider fearful subjects. Participants were randomly assigned to use reappraisal or affect labeling during exposure or to complete exposure without verbalization. At re-test the group that completed exposure with affect-labeling had lower autonomic activity while viewing a spider and moved closer to the spider compared to the reappraisal and exposure alone groups. In addition, those who used the greatest number of anxiety and fear related words during affect labeling showed the greatest reductions in fear responding. This study provides evidence that affect labeling rather than reappraisal may be a more promising approach to enhancing the effectiveness of exposure. Therefore, the current project compared exposure plus affect labeling to exposure alone to specifically test affect labeling as an augmentation strategy for exposure therapy.

1.4.2. Behavioral research on affect labeling. Many studies have demonstrated that expressing emotions through language is beneficial for psychological wellbeing, physical health, and cognitive performance. Pennebaker and Beall (1986) were some of the first researchers to demonstrate through a controlled study that writing about stressful experiences is beneficial for physical health. Subsequently, hundreds of researchers have assessed the effects of expressive writing about a wide variety of experiences and within many different populations. Cognitive processing therapy (Resick & Schnicke, 1993), an effective treatment for post-traumatic stress disorder, involves systematic written exposure about one's traumatic experience and the evoked emotions. Studies that have examined the effect of written or verbal processing of trauma suggest that linguistic processing of traumatic experiences is beneficial for depression and anxiety (Hemenover, 2003; Lepore, Silver, Wortman, & Wayment, 1996) and reduces stress responses to trauma related memories (Smyth, Hockemeyer, & Tulloch, 2008). Studies have also demonstrated that recording worries about an upcoming exam in writing increases exam performance (Frattaroli, Thomas, & Lyubomirsky, 2011; Ramirez & Beilock, 2011), and that writing about negative events decreases intrusive thoughts and increases working memory (Klein & Boals, 2001). Finally, Lieberman and colleagues (2011) compared participants' distress while viewing negative IAPS images with and without affect labeling and found lower reported distress in the labeling condition despite participants' predictions that affect labeling would increase distress.

1.4.3. Neuroimaging and physiological research on affect labeling. Lieberman and colleagues (2007), demonstrated that when participants label the emotional expression of a face (e.g. "angry"), they show reduced amygdala activation, and increased activation in the RVLPFC compared to viewing faces without labeling, or labeling the gender of the face. A significant

number of neuroimaging studies have now demonstrated that labeling one's emotional experience activates areas of the prefrontal cortex, and reduces activation in the amygdala (Gorno-Tempini et al., 2001; Hariri, Bookheimer, & Mazziotta, 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Narumoto et al., 2000). These findings suggest that downregulation of amygdala activation occurs through projections from prefrontal regions.

Tabibnia and colleagues (2008) examined the effect of repeated exposure to evocative images with and without negative affective labels. In study 1 in a non-clinical sample, repeated presentation of emotionally evocative images paired with an affect label resulted in greater attenuation of GSR upon presentation of the images without a label at 1-week re-test. In study 2, the effects were replicated in a spider-fearful sample undergoing exposure to images of spiders. Participants who went through exposure paired with negative labels showed greater attenuation of GSR at 1-week re-test compared to those who saw no labels or those who saw neutral labels.

1.5. Moderators of Response to Labeling Versus Exposure Alone

Better matching of treatments to individuals can improve therapy outcomes. However, researchers have yet to identify whether patients who have deficits in a particular skill are more likely to benefit from treatments that target that skill (deficit model), or whether those who already gravitate towards a coping approach will benefit most from a treatment that matches that approach (matching model). Evidence from the expressive writing literature supports both models with some studies showing that participants high in trait emotional expressivity benefit most from expressive writing (Austenfeld, Paolo, & Stanton, 2006; Niles, Haltom, Mulvenna, Lieberman, & Stanton, 2014; Stanton, Kirk, Cameron, & Danoff-Burg, 2000), while others show that participants with deficits in emotional expression benefit most from expressive writing (Lu & Stanton, 2009; Páez, Velasco, & González, 1999). The current study will examine whether

the matching model can be used to predict who will benefit most from exposure combined with affect labeling.

1.6. Summary

Fear of public speaking is common and in some cases can cause significant impairment in functioning. Exposure is an effective treatment method for public speaking anxiety, and is often combined with cognitive restructuring or other methods of verbalization, but few studies have examined whether verbalization can enhance the effectiveness of exposure. Evidence from neuroscience suggests that in anxiety disorders, there are deficits in connectivity between regions of the prefrontal cortex and the amygdala, which may explain deficits in regulating anxious responding in these individuals. Affect labeling reduces self-reported distress while viewing negative images, and when combined with exposure in spider fearful subjects, produces greater fear attenuation for physiological measures of arousal compared to exposure alone. Affect labeling activates pathways between prefrontal regions and the amygdala, and therefore, combining exposure with affect labeling may strengthen prefrontal-amygdala connections thereby enhancing long-term fear reduction.

1.7. Specific Aims and Hypotheses

The first aim of the study will be to assess whether affect labeling enhances the effectiveness of exposure compared to exposure alone. We hypothesize that participants who use affect labeling during exposure will show greater attenuation of fear of public speaking compared to those who undergo exposure alone. The second aim of the study will be to assess whether the number of anxiety or fear related words used during exposure predicts greater attenuation of fear responding at re-test. We hypothesize that participants who use more anxiety or fear related words compared to other emotion words will show the greatest fear reduction at

re-test. The third aim of the study will be to assess whether individual differences in incidental emotion regulation (i.e. the extent to which affect labeling reduces distress) at baseline moderate response to exposure with affect labeling versus exposure alone. Consistent with the matching model, we hypothesize that participants who show strengths in incidental emotion regulation at baseline will show greater fear attenuation in the labeling condition than in the exposure alone condition.

CHAPTER 2

Method

2.1 Overview of Design, Independent Variables, and Procedure

This study used a 2 (Group) × 3 (Time) mixed design with speech fearful participants. Groups included an exposure combined with affect labeling condition, and an exposure alone condition, and Time was three assessment time-points at baseline (Time 1), following exposure (Time 2) and at one-week follow-up (Time 3). To test Aim 1, the labeling condition was compared to the exposure alone condition on fear attenuation immediately following exposure (Time 2) and at one-week follow-up (Time 3). To test Aim 2, the number of anxiety related emotion words used by participants during exposure was used to predict fear responding following exposure (Time 2) at one-week follow-up (Time 3). To test Aim 3, incidental emotion regulation at baseline was examined as a moderator of response to exposure plus affect labeling versus exposure alone.

For a flowchart of study procedures, see Figure 1. On day one (Time 1), participants first completed a series of questionnaires followed by a behavioral approach task (BAT-1) during which autonomic arousal, self-reported affect, cognitions, self-rated performance, and behavioral avoidance was assessed. Participants next completed an affect labeling task to assess incidental emotion regulation. Participants were then assigned to the labeling or exposure alone conditions. Participants were moved to a different room, and completed 10 exposure trials (Exp-1) either with or without labeling depending on group assignment. Time 2 was three days later, and participants again underwent 10 exposure trials (Exp-2) with or without labeling depending on group assignment. After the exposures, participants moved to the room in which the original

BAT was conducted, and underwent a second BAT using the same protocol as Time 1 (BAT-2). Time 3 was eight days later, and participants again completed the BAT using the same method as at Time 1 and Time 2 (BAT-3).

2.2. Participants

One hundred two participants (AL = 52; Control = 50) were recruited to participate. Two participants assigned to the control group were not included in analyses. Due to experimenter error, one participant received the affect labeling exposure protocol rather than the control protocol on the second day of exposure. The other participant appeared to be answering randomly on questionnaires and fell asleep during the experiment. Therefore, the final sample included in analyses was 100. See Figure 2 for a consort diagram of flow through study procedures. Eligible participants reported a six or higher on anxiety and a five or higher on avoidance of public speaking on a zero to eight scale (see Appendix A). Participants were recruited from the UCLA Psychology Subject Pool and using flyers posted around campus. Participants were given 1 hour of research credit per day of participation or were paid $10.00 per day if they were not enrolled in a course that required research credit. Participants therefore received 3 research credits or $30.00 for completing all three days.

Participants were over 18 years of age, fluent in English, free of heart, neurological, or respiratory conditions, hearing impairment, physician recommendation to avoid stressful situations, current treatment for public speaking anxiety, or psychotropic medication prescription for an emotional problem. These participants were excluded due to potential interference with psychophysiological measurement and for safety precautions.

2.3. Materials

A summary of study measures is included in Table 1. Questionnaires are included in Appendix A.

2.3.1 Indices of fear responding.

2.3.1.1. Physiological activity. Physiological activity recording through the Biopac system was facilitated using an IBM Pentium II and AcqKnowledge software (AcqKnowledge 4.1 for Windows; BIOPAC Systems, inc). Galvanic skin response (GSR) was recorded from electrodes attached to the distal phalanges of the second and third finger of a participant's non-preferred hand. Heart rate (HR) was recorded from electrodes attached below the right collarbone and bottom left rib. Physiological data from the final one-minute of the two-minute baseline (baseline), the one-minute anticipation period prior to the speech (anticipation), and the one-minute recovery period after completion of the speech (recovery) were analyzed.

All physiological data were first visually inspected to ensure proper measurement. For HR, one participant at Time 1, and two participants at Time 2 were excluded from analysis due to recording error. For GSR, nine participants at Time 1, 10 participants at Time 2, and 6 participants at Time 3 were excluded from analysis because no variations in GSR signal were observed. These numbers are consistent with estimates that approximately 10% (or more in clinical samples) of individuals do not show a reliable GSR response (Braithwaite, Watson, Jones, & Rowe, 2013). To assess HR, electrocardiogram signals were collected from two electrodes, one on the participant's right clavicle, and one below the bottom left front rib. HR was defined as the number of heart beats per minute. A band pass filter with a low cutoff of 1.00Hz and a high cutoff of 35.00Hz was applied prior to analysis to limit the effect of signal noise on the data. GSR was measured using two indices: Skin conductance level (SCL) and non-specific skin conductance response (SCR-NS). Skin conductance level was defined as the

average skin conductance level over each one-minute assessment period and was measured to the nearest microsiemen (μs). SCR-NS was assessed by calculating the frequency of non-specific skin conductance responses during each one-minute period. A skin conductance response was defined by a minimum increase of .02 μs. Data were analyzed using built in analysis tools in AcqKnowledge software for HR, SCL, and SCR-NS.

2.3.1.2 Personal Report of Public Speaking Anxiety (PRPSA; McCroskey, 1970). See Appendix A. PRPSA is a 34-item measure that assesses fear of public speaking. Responders rate their degree of agreement with each statement on a 5-point Likert scale (1=strongly disagree to 5=strongly agree). The scale has excellent reliability (α=.90) (McCroskey, 1970). Scores of 131 or higher indicate high public speaking anxiety, scores of 98 – 130 indicate moderate anxiety and scores below 98 indicate low public speaking anxiety. In the current study, α = .97 at Time 1.

2.3.1.3. Subject Units of Distress Scale (SUDS). SUDS is a single item measure used to assess state anxiety. Participants were shown a 0 to 8 Likert scale with 0 indicating no anxiety and 8 indicating extreme anxiety. Participants were then asked to report their SUDS ratings at various points in the study. SUDS ratings were taken directly prior to and following speech tasks, and when reported, SUDS ratings are the average of the ratings before and after the speech.

2.3.1.4. Self Statements During Public Speaking (SSPS; Hofmann & DiBartolo, 2000). See Appendix A. The 10-item SSPS assesses negative and positive cognitions and was completed immediately after the public speaking task; participants rated the extent to which they experienced five negative and five positive thoughts during the speaking task. The SSPS shows good internal consistency (α = .86) and test-retest reliability (r = .80) (Hofmann & DiBartolo,

2000). In the current study $\alpha = .78$ for positive cognitions and .84 for negative cognitions at Time 1.

2.3.1.5. Optional Speech. At each BAT, participants had the option of speaking on an additional speech topic. Participants received $5.00 for completing the additional speech.

2.3.2. Other Measures.

2.3.2.1. Performance Rating Form (PRF; Rapee & Lim, 1992). See Appendix A. This 17-item questionnaire assesses self-reported public speaking performance. The scale includes 12 specific items to represent individual behaviors or reactions necessary to good public speaking (e.g. kept eye contact with audience, had a clear voice), and 5 global items to assess overall evaluations of performance (e.g. generally spoke well). Participants rate items on a 5-point Likert scale from 0 (not at all) to 4 (very much) and higher scores indicate better rated performance. In the current study, $\alpha = .91$ at Time 1.

2.3.2.2. Patient Health Questionnaire (PHQ; Spitzer, Kroenke, & Williams, 1999). See Appendix A. The PHQ is a widely used 9-item questionnaire used to assess symptoms of depression. The PHQ is a valid measure of depressive symptoms (Löwe, Spitzer, et al., 2004) and is sensitive to change (Löwe, Kroenke, Herzog, & Gräfe, 2004). Patients report the frequency of experiencing each symptom on a four-point Likert scale. In the current study, $\alpha = .84$ at Time 1.

2.3.2.3. Exposure Credibility Questionnaire. See Appendix A. This four-item measure includes 0-8 Likert scale ratings assessing participants' perceived credibility of the exposure task. This measure has been used in previous exposure studies as a potential predictor of study drop out (Craske, Street, Jayaraman, & Barlow, 1991). In the current study, $\alpha = .83$ at Time 1.

2.3.2.4. Word use during exposure. During exposures, participants assigned to the verbalization group chose from a set of emotion words displayed on the computer screen using the keyboard. Their responses were recorded, and the number of anxiety related emotions was identified for each participant.

2.3.2.5 Mini Social Phobia Inventory (SPIN; Connor, Kobak, Churchill, Katzelnick, & Davidson, 2001). See Appendix A. This three-item measure assesses symptoms of social anxiety disorder and is based on a longer 17-item version of the scale. Participants respond to items on a 0 (not at all) to 4 (extremely) Likert scale. Scores of 6 or higher indicate possible problems with social anxiety disorder. The scale demonstrates 90% accuracy in identifying a diagnosis of generalized social anxiety disorder. In the current study $\alpha = .86$ at Time 1.

2.3.2.6. Mini International Neuropsychiatric Interview (MINI; Lecrubier et al., 1997). The MINI is a brief fully structured diagnostic interview that can be administered by non-specialized interviewers. Kappa, specificity, and sensitivity for the MINI compared to diagnoses obtained on the Composite International Diagnostic Interview are good or very good for all but three diagnoses (generalized anxiety disorder, agoraphobia, and bulimia), and inter-rater and test-retest reliability are good (Lecrubier et al., 1997). The current project used only the social anxiety disorder section of the MINI to determine whether participants met diagnostic criteria for social anxiety disorder.

2.3.2.7. Incidental Emotion Regulation. Incidental emotion regulation was assessed using the Affect Labeling Task (Lieberman et al., 2011), which assessed how effective participants are at decreasing distress using affect labeling. Incidental emotion regulation was calculated by subtracting participants' average level of distress when labeling negative images from their average level of distress when viewing negative images without labeling. Scores

ranged from -1.4 to 2.0, and higher incidental emotion regulation scores indicated more effective emotion regulation. For more details on the Affect Labeling Task, see procedure below.

2.4. Procedure

Participants who were eligible to participate were scheduled for three appointment times on days 1, 4 and 9. Appointment times were in the afternoon between 4:00 and 6:00 pm. Participants were scheduled at the same time on each of the three days. On day 1, participants completed the Affect Labeling Task, the first BAT (BAT-1), and the first exposure session (Exp-1). On day 4, participants completed the second exposure session (Exp-2) and the second BAT (BAT-2). On day 9, participants completed the third BAT (BAT-3). See Figure 1 for a diagram of study procedures.

2.4.1. Time 1.

2.4.1.1. BAT-1 (30 minutes). On day 1, participants were located in a small room on the A Level of Franz Hall. When participants arrived, they were consented. After being consented, participants were interviewed using the Social Anxiety Disorder section of the MINI Neuropsychiatric Interview by trained research assistants. Following completion of the interview, electrodes were attached to participants to begin recording by the Biopac physiological measurement system. Physiological measurement included HR and GSR. Although physiological activity was recorded throughout the BAT, only measurements taken during the baseline, anticipation, and recovery periods were analyzed.

Participants then reported on demographic characteristics and completed the PRPSA, PHQ, and SPIN while acclimating to the environment for approximately 10 minutes. After completion of the questionnaires, the two-minute baseline period began. Participants were then trained on how to use the SUDS to report anxiety level and practiced entering their SUDS ratings

using the computer. The research assistant then provided the participant with instructions for completing the speech BAT. Once the participant was clear about how the task would proceed, a one-minute anticipation period began during which the participant sat behind a screen. During the anticipation period, the three confederates sat in chairs facing the area where the participant would stand during his/her speech. At the end of the one-minute anticipation, the participant provided a SUDS rating, and stood in front of the audience. The beginning of the speech task was signaled by a tone played from the computer, and the first speech topic was displayed on the computer screen. The participant spoke for one minute on the first topic. The participant then entered a SUDS rating into the computer and, using the computer, indicated whether or not she/he was willing to speak on an additional topic for $5.00. If the participant was willing to continue, a second anticipation period occurred, another SUDS rating was taken, and the next topic was displayed. After one-minute, a final SUDS rating was taken. Regardless of whether or not the participant gave the additional speech, a one-minute recovery period followed the final speech. After completion of the speeches, the participant completed the SSPS, and rated his/her performance on the PRF. The participant was compensated $5.00 if he/she opted to give the additional speech.

2.4.1.2. Affect Labeling Task (10 minutes). When the participant was finished speaking, physiological equipment was removed, and the participant was taken to another location in the building. The participant then completed a computer task that involved affect labeling and viewing negative images from the international affective picture system (IAPS; Lang, Bradley, & Cuthbert, 1999). The Affect Labeling Task followed the procedure of study 1 by Lieberman, Inagi, Tabibnia, and Crockett (2011). Participants viewed neutral and negative images presented in two blocks of four images. Each block contained two moderately negative, and two extremely

negative images. Prior to each block, participants were prompted by cues that said either "scene description" (labeling) or "look and let yourself respond naturally" (watching). Pictures appeared for 5 seconds. For labeling blocks, participants were asked to choose from three labels that appeared at the bottom of the screen. One label was relevant to the image, and the other two were not. Two of the labels were negative, and one was neutral. Participants chose a word by pressing a key on the keyboard that corresponded to the position of the word on the screen. For watching blocks, participants simply viewed each image for 5 seconds. Following the presentation of each image, regardless of block condition, participants were asked "How distressed did you feel while looking at the picture?" and responded on a 9-point Likert scale with 0 being not distressed, and 8 being very distressed.

2.4.1.3. Exp-1 (30 minutes). The participant was then randomly assigned to complete exposure alone, or exposure with affect labeling. The participant completed 10 one-minute repeated exposure speech trials interspersed with 30-second inter-trial intervals between each exposure. This protocol was chosen based on previous research findings that this duration is effective in exposure for phobias (Kircanski et al., 2012; Mineka, Mystkowski, Hladek, & Rodriguez, 1999; Rowe & Craske, 1998). Participants were provided with instructions by a new experimenter. Once the participant understood the instructions and practiced responding to questions using the computer, the participant sat behind a folding screen. Three confederates, including the experimenter, were seated facing the area where the participant gave the speech. A tone was played from the computer, which signaled to the participant to step in front of the audience. All participants were prompted by the computer to provide a SUDS rating. Participants in the affect labeling condition were then prompted by the computer to choose an emotion and a feared outcome from four options presented on the screen. At the top of the

screen, the phrase, "I feel _____" was presented followed by three possible emotions and "other." All three emotions were negative, and one was always anxiety related (see Appendix B for list of emotions). The phrase "The audience will _____" was then presented followed by three possible feared outcomes related to the audience's response to the participant (see Appendix B for list of feared outcomes). An "other" option was also available. Participants used the keyboard to respond and had up to 15 seconds to make each selection. Participants were presented with ten different sets of emotion labels and feared outcomes. Participants in the exposure alone condition completed shape matching. Participants were first presented with a large black shape at the top of the screen, then were asked to match the shape with one of three options at the bottom of the screen. If the shape at the top did not match any of the three shapes at the bottom, the participants were asked to choose "other." Participants then did the same shape matching exercise a second time with blue instead of black shapes. Then, a speech topic appeared on the screen. Participants spoke for 1 minute then were prompted to step behind the screen for a 30 second inter-trial interval. The speech exposures were repeated 10 times. After completion of the exposure, participants completed the ECQ.

2.4.2. Time 2.

2.4.2.1. Exp-2 (30 Minutes). The second exposure session was completed three days after Exp-1. Two exposure sessions were used to parallel multiple exposures as done in therapy. Participants received the same instructions as at the previous exposure session. Procedures were identical to those for Exp-1 on Day 1.

2.4.2.2. BAT-2 (30 Minutes). The second BAT was completed directly following Exp-2. The protocol for BAT-2 followed the exact protocol for BAT-1, however the speech topics were different. The same audience that was present for BAT-1 was present for BAT-2.

2.4.3. Time 3.

2.4.3.1. BAT-3 (30 minutes). The third and final BAT was completed 8 days after BAT-1. One-week retest assessed long-term fear reduction and inhibitory learning (Craske et al., 2008). The protocol for BAT-3 followed the exact protocol for BAT-1 and BAT-2, however the speech topics were different. The same audience that was present for BAT-1 and BAT-2 was present for BAT-3.

2.4.4. Speech topics. Speech topics are listed in Appendix C. Across all three BATs, participants spoke on a total of three to six speech topics depending on how many additional speeches they were willing to complete. The 6 topics for the BATs were divided into three sets of two and were counterbalanced across the three time points using a Latin square. Across both exposure sessions, participants spoke on 20 speech topics. The 20 speech topics for the exposures sessions were the same across all participants and were administered in the same order.

2.4.5. Exposure instructions. Copies of exposure instructions are presented in Appendix D. All participants received the same set of instructions to keep research assistants blind to condition. Participants were informed that they would be giving 10 speeches in front of a small audience and that they may be asked to label the emotion that they were feeling. They were asked to choose the emotion from a list of four emotions that best matched how they were feeling at that moment. They were encouraged to choose one of the emotions listed, but if they really felt that the words did not match their experience, they were allowed to choose "other." They were also instructed that they may be asked to choose how they thought the audience might respond to them while they were speaking, and were asked to choose an outcome from the list that best matched their expectation of how the audience would respond. They were again

encouraged to choose one of the options listed, but could also select other. Finally, they were informed that they may be asked to match shapes, and were asked to choose the shape from a list of four possible shapes that matched a large target shape at the top of the screen. If none of the shapes listed matched the target shape, they were instructed to choose other. Finally, they were informed that they might see the same shaping matching screen with blue instead of black shapes.

2.5. Data Analysis

Analyses were conducted using Stata 12. Dependent measures assessed for all three study aims were HR, SCL, SCR-NS, PRPSA, SUDS, SSPS, PRF, and optional speech. For analyses including HR, HR^2 at baseline was used as a covariate in the models to account for the non-linear relationship between heart rate and activity in the sympathetic and parasympathetic nervous systems (Cacioppo, Tassinary, & Berntson, 2007). SCL was log transformed in accordance with recommendations by Venables and Christie (as cited in Cacioppo et al., 2007). For models including SCR-NS (a count variable) as the dependent measure, Poisson regression was used to account for non-normality. For models including optional speech (a dichotomous variable) as the dependent variable, logistic regression was used. For analyses of the recovery period (following participants' decision to complete or avoid the optional speech), optional speech was tested as a covariate and included in the model when significant. All tests were two-tailed with an α level of .05.

All three study aims were tested using multi-level modeling (MLM) because measurement was repeated at three time-points for each participant. MLM accounts for within and between participant variance. In addition, MLM effectively handles missing data by including all participants in the model regardless of missing data points. Time was modeled at

level 1, and participant level variables (e.g. Group) were modeled at level two. Time was modeled using two segments. Once segment modeled change from Time 1 to Time 2, and the second modeled change from Time 2 to Time 3. This approach was chosen based on a pattern of results typically observed in intervention studies characterized by an initial steep change in symptoms from pre to post intervention and a leveling off of change through follow-up. For each dependent variable, random effects of the intercept and slope and their covariance were first included in the model. Non-significant random effects (tested using likelihood ratio tests) were removed from the model. For each dependent variable, the model with the fewest random effect parameters necessary to achieve optimal model fit was chosen.

For Aim 1, predictors in the model were Time, Group and the Time × Group interaction, and the Time × Group fixed effect was examined for significance. For Aim 2, predictors in the model were Time, number of anxiety labels chosen, and the Time × number of anxiety labels interaction, and the Time × number of anxiety labels fixed effect was examined for significance. Analyses for aim 2 were only conducted within the AL group. For Aim 3, predictors in the model were Time, Group, incidental emotion regulation, Time × Group, Time × incidental emotion regulation, Group × incidental emotion regulation, and Time × Group × incidental emotion regulation, and the Time × Group × incidental emotion regulation fixed effect was examined for significance. If the three way interaction (test of moderation) was not significant, it was dropped from the model and the Time × incidental emotion regulation (test of prediction) fixed effect was examined for significance.

2.6. Effect Size

For study Aim 1, effect sizes reported are Cohen's *d*, and were calculated using an approach described by Feingold (2009) for estimating group differences in randomized clinical

trials with repeated measures. For study Aims 2 and 3, effect sizes reported are Cohen's f^2 and were estimated using an approach outlined by Selya and colleagues (2012). Cohen's f^2 uses residual variance from the model to estimate effect size. However, for multi-level models, effect sizes calculated using residual variance and proportion of variance explained should be interpreted with caution because the addition of variables to the model can, in some cases, increase residual variance resulting in negative estimates of explained variance and even of effect size (Snijders & Bosker, 1994) In addition, this method cannot be used for non-continuous dependent measures. As a result, effect sizes are not reported for analyses with SCR-NS (count variable) or optional speech (dichotomous variable) as the outcome.

2.7. Missing Data

There were missing data on a number of demographic, predictor, and dependent measures. Reasons for missing data included participant non-response, experimenter error, inadvertent deletion, and computer program failure. The numbers of missing data points for all study variables are shown in Table 2.

2.8. Power Analyses

Effect sizes for group comparisons in the study by Kircanski and colleagues (2012), in which verbalization during exposure was compared to exposure alone in spider phobic participants, ranged from .58 to .99 depending on which outcome measure was assessed. To achieve power of 0.8, for an effect size of .60, the goal sample size was 72 participants.

CHAPTER 3

Results

3.1. Preliminary Analyses

3.1.1. Sample characteristics. Groups did not significantly differ on any demographic or clinical characteristics at baseline (*p*s > .05). Table 3 includes baseline descriptive statistics and statistical tests for group differences. At baseline, 54% of the sample fell in the "high" public speaking anxiety range on the PRPSA (scores above 131), 46% of the sample fell in the moderate range (scores between 97 and 131), and 0% of the sample fell in the "low" public speaking anxiety range. The current sample had a mean PRPSA score of 133.0, which is approximately one standard deviation above the mean of 114.6 (SD = 17.2) observed in a college sample (McCroskey, 1970). On the mini-SPIN, 65% of the sample fell at or above the clinical cut-off of 6, indicating possible problems with social anxiety, and 36% met criteria for social anxiety disorder based on the MINI diagnostic interview. On the PHQ, 49% of the sample fell in the minimal range, 39% in the mild range, 7% in the moderate range, 4% in the moderately severe range, and 1% in the severe range for depressive symptoms.

3.1.2. Descriptive statistics and correlations among study variables. Table 4 includes raw means and standard deviations for all dependent measures at all three time points by group. Table 5 includes proportions of affect labels and feared outcomes chosen at Exp1 and Exp2 (AL group only), mean SUDS ratings at the beginning and end of Exp1 and Exp2 by group, and ECQ ratings at Exp1 by group. Table 6 includes correlations among dependent measures, affect labeling variables collected during exposure, and incidental emotion regulation.

3.1.3. Comparison of completers vs. dropout. Table 7 includes means and standard deviations for dependent measures and exposure variables for completers and participants who dropped from the study as well as statistical tests comparing the two groups. Participants who dropped reported significantly fewer positive self statements during the public speaking task, rated their performance significantly lower, had significantly higher HR during anticipation of giving a speech, had marginally significantly higher HR when recovering after giving a speech, had marginally significantly higher SCR-NS during anticipation of giving a speech, and were marginally significantly less likely to give the optional speech compared to participants who completed the study. Dropout did not differ between the AL group (N=11) and the Control group (N=8) $\chi^2 = .33$, $p = .568$. Given that significant differences were found between completers and those who dropped from the study, dropout was tested as a covariate in each model, and when significant, was included in the final model.

3.2. Primary Analyses Aim 1. Does Affect Labeling Enhance Exposure Effectiveness Compared to Exposure Alone?

Figures are provided for each dependent variable assessed. For each model tested, interaction effects from Time 1 to Time 2, Time 2 to Time 3, and Time 1 to Time 3 are reported. For significant and marginally significant interactions, the following simple effects are tested. (1) Group differences at Time points 2 and 3; (2) Simple slopes (whether slopes differ from zero) from Time 1 to Time 2, Time 2 to Time 3, and/or Time 1 to Time 3 (depending on significant interactions).

3.2.1. Heart rate.

3.2.1.1. Anticipation. Results are displayed in Figure 3. For HR during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .934), Time 2 to Time 3 (p = .191), and from Time 1 to Time 3 (p = .152) were not significant.

3.2.1.2. Recovery. Results are displayed in Figure 4. For HR during recovery following the speech, the Time × Group interactions from Time 1 to Time 2 (p = .654) and from Time 1 to Time 3 (p = .153) were not significant. The Time × Group interaction from Time 2 to Time 3 was significant (b = -3.79, 95% Confidence Interval (CI) = -7.4 to -.2, p = .041, d = .33) such that participants in the AL group showed a steeper decrease in HR from Time 2 to Time 3 than did participants in the Control group. Tests of group differences revealed no significant differences at Time 2 (p =.419) or at Time 3 (.281). Tests of simple slopes from Time 2 to Time 3 revealed a significant increase in HR in the Control group (change = 2.77, p = .036) and no significant change in the AL group (p = .432).

3.2.2. Skin conductance level.

3.2.2.1. *Anticipation*. Results are displayed in Figure 5. For SCL during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .690), Time 2 to Time 3 (p = .947), and Time 1 to Time 3 (p = .743) were not significant.

3.2.2.2. *Recovery*. Results are displayed in Figure 6. For SCL during recovery following the speech, the Time × Group interactions from Time 1 to Time 2 (p = .302), Time 2 to Time 3 (p = .861), and Time 1 to Time 3 (p = .227) were not significant.

3.2.3. Non-specific skin conductance response.

3.2.3.1. *Anticipation*. Results are displayed in Figure 7. For SCR-NS during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .875), Time 2 to Time 3 (p = .778), and Time 1 to Time 3 (p = .878) were not significant.

3.2.3.2. *Recovery*. Results are displayed in Figure 8. For SCR-NS during recovery following the speech, the Time × Group interaction from Time 1 to Time 2 was not significant (p = .587), whereas the interactions from Time 2 to Time 3 (b = -1.14, CI = -2.1 to -.2, p = .023, d = 1.0) and Time 1 to Time 3 (b = -.90, CI = -1.8 to 0, p = .023, d = .79) were significant such that participants in the AL group showed a steeper decrease in SCR-NS from Time 2 to Time 3 and from Time 1 to Time 3 than did participants in the Control group. Tests of group differences revealed no significant group difference at Time 2 (p =.273) and a marginally significant difference at Time 3 (difference = .29, p = .100) such that participants in the AL group had fewer SCR-NS during recovery than did participants in the Control group. Tests of simple slopes from Time 2 to Time 3 revealed a marginally significant reduction in SCR-NS during recovery in the AL group (change = -.61, p = .077), but not in the Control group (p = .146), and no significant changes from Time 1 to Time 3 in either group (ps > .123).

3.2.4. Personal Report of Public Speaking Anxiety. Results are displayed in Figure 9. For PRPSA, the Time × Group interactions from Time 1 to Time 2 (p = .196), Time 2 to Time 3 (p = .875), and Time 1 to Time 3 (p = .394) were not significant.

3.2.5. Subjective Units of Distress. Results are displayed in Figure 10. For SUDS, the Time × Group interactions from Time 1 to Time 2 (p = .385), Time 2 to Time 3 (p = .328), and Time 1 to Time 3 (p = .901) were not significant.

3.2.6. Self Statements During Public Speaking.

***3.2.6.1. Positive*.** Results are displayed in Figure 11A (higher scores indicate more positive cognitions). For SSPS Positive, the Time × Group interactions from Time 1 to Time 2 (p = .837), Time 2 to Time 3 (p = .264), and Time 1 to Time 3 (p = .316) were not significant.

3.2.6.2. *Negative*. Results are displayed in Figure 11B. For SSPS Negative, the Time × Group interactions from Time 1 to Time 2 (p = .952), Time 2 to Time 3 (p = .320), and Time 1 to Time 3 (p = .417) were not significant.

3.2.7. Performance Rating Form. Results are displayed in Figure 13 (higher scores indicate better self-rated performance). For PRF, the Time × Group interactions from Time 1 to Time 2 (p = .544) and from Time 1 to Time 3 (p = .353) were not significant whereas, the interaction from Time 2 to Time 3 was marginally significant (b = -3.06, CI = -6.5 to .34, p = .077, d = .36) such that participants in the Control group showed a steeper increase in PRF from Time 2 to Time 3 than did participants in the AL group. Tests of group differences revealed no significant differences at Time 2 or Time 3 (ps > .210). Tests of simple slopes revealed a significant increase from Time 2 to Time 3 in PRF in the Control group (change = 4.57, p < .001), but not in the AL group (p = .217).

3.2.8. Optional speech. Results are displayed in Figure 12. For optional speech, the model did not converge when all three time points were included. Therefore, the model was run including Time 1 and Time 3 only. The Time × Group interaction from Time 1 to Time 3 was not significant (p = .669).

3.3. Secondary Analyses Aim 1. Does Affect Labeling Enhance Exposure Effectiveness for High Labelers Compared to Exposure Alone?

A number of participants assigned to the affect labeling condition chose no or very few anxiety labels; we evaluated whether participants who more consistently selected anxiety labels during exposures showed greater fear reduction compared to those assigned to exposure alone. Participants were categorized into “low” or “high” labelers based on the median frequency of affect labels chosen, and the high labelers were compared to participants in the control condition.

3.3.1. Heart Rate.

3.3.1.1. Anticipation. Results are displayed in Figure 14. For HR during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .554) and Time 1 to Time 3 (p = .198) were not significant whereas the interaction from Time 2 to Time 3 was marginally significant (b = -4.96, CI = -10.3 to .4, p = .071, d = .39) such that participants in the AL group showed a steeper decrease in HR from Time 2 to Time 3 than did participants in the Control group. Tests of group differences revealed no significant differences at Time 2 or Time 3 (ps > .174). Tests of simple slopes from Time 2 to Time 3 revealed no significant change in either group (ps > .191).

3.3.1.2. Recovery. Results are displayed in Figure 15. For HR during recovery following the speech, the Time × Group interaction from Time 1 to Time 2 was not significant (p = .328). The interaction from Time 2 to Time 3 (b = -6.47, CI = -10.6 to -2.3, p = .002, Cohen's d = .56) was significant and the interaction from Time 1 to Time 3 was marginally significant (b = -4.39, CI = -8.9 to .1, p = .054, Cohen's d = .38) such that high labelers in the AL group had a steeper decline in HR during recovery than did participants in the Control group. Tests of group differences revealed no group differences at Time 2 or Time 3 (ps > .129). Tests of simple slopes from Time 1 to Time 3 revealed a marginally significant increase in HR in the Control group (change = 2.63, p = .057) and no significant change in the AL group (p = .333). Tests of simple slopes from Time 2 to Time 3 revealed a significant increase in HR in the Control group (change = 2.70, p = .033) and a significant decrease in HR in the AL group (change = -3.77, p = .027).

3.3.2. Skin conductance level.

3.3.2.1. Anticipation. For SCL during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .281), Time 2 to Time 3 (p = .921) and Time 1 to Time 3 (p = .329) were not significant.

3.3.2.2. Recovery. For SCL during recovery following the speech, the Time × Group interactions from Time 1 to Time 2 (p = .603), Time 2 to Time 3 (p = .676), and Time 1 to Time 3 (p = .339) were not significant.

3.3.3. Non-specific skin conductance response.

3.3.3.1. Anticipation. Results are displayed in Figure 16. For SCR-NS during anticipation of giving a speech, the Time × Group interactions from Time 1 to Time 2 (p = .715) and Time 2 to Time 3 (p = .128) were not significant. The interaction from Time 1 to Time 3 was significant (b = -.62, CI = -1.2 to 0, p = .049, d = .35) such that high labelers in the AL group had a steeper decline in SCR-NS during anticipation than did participants in the Control condition. Tests of group differences revealed no group difference at Time 2 (p = .287), and a significant group difference at Time 3 (difference = -.89, p = .008) such that the AL group had fewer SCR-NS than did the Control group. Tests of simple slopes revealed a marginally significant decrease in SCR-NS from Time 1 to Time 3 in the Control group (p = .055), and a significant decrease in SCR-NS from Time 1 to Time 3 in the AL group (change = -.93, p = .001).

3.3.3.2. Recovery. Results are displayed in Figure 17. For SCR-NS during recovery following the speech, the Time × Group interaction from Time 1 to Time 2 was not significant (p = .385). The interaction from Time 2 to Time 3 (b = -1.87, CI = -3.3 to -.4, p = .013, d = 1.63) was significant, and the interaction from Time 1 to Time 3 was marginally significant (b = -.14, CI = -2.8 to 0, p = .055, Cohen's d = 1.21) such that high labelers in the AL group had a steeper

decline in SCR-NS during recovery than did participants in the Control group. Tests of group differences revealed no significant group difference at Time 2 (p = .536), and a significant group difference at Time 3 (difference = .47, p = .008) such that the AL group had fewer SCR-NS during recovery than did the Control group. Tests of simple slopes from Time 2 to Time 3 revealed a significant decrease in SCR-NS during recovery in the AL group (change = -1.33, p = .041), but not in the Control group (p = .143), and no significant change from Time 1 to Time 3 in either group (ps > .131).

3.3.4. Personal Report of Public Speaking Anxiety. Results are displayed in Figure 18. For PRPSA, the Time × Group interactions from Time 2 to Time 3 (p = .957) and Time 1 to Time 3 (p = .214) were not significant. The interaction from Time 1 to Time 2 was marginally significant (b = 5.37, CI = -.9 to 11.7, p = .095, d = .35) such that participants in the Control group had a steeper decline in PRPSA than did high labelers in the AL group. Tests of group differences revealed no significant group difference at Time 2 (p = .122) or Time 3 (p = .184). Tests of simple slopes revealed a significant decrease in PRPSA from Time 1 to Time 2 in the AL group (change = -5.33, p = .041) and in the Control group (change = -10.70, p < .001).

3.3.5. Subjective Units of Distress. Results are displayed in Figure 19. For SUDS, the Time × Group interactions from Time 2 to Time 3 (p = .692) and Time 1 to Time 3 (p = .209) were not significant. The interaction from Time 1 to Time 2 was marginally significant (b = .57, CI = -.1 to 1.2, p = .079, d = .38) such that participants in the Control group showed a steeper decrease in SUDS from Time 1 to Time 2 than did participants in the AL group. Tests of group differences revealed a significant group difference at Time 2 (difference = .81, p = .038) such that the AL group had higher SUDS ratings than did the Control group. Groups did not significantly differ at Time 3 (p = .104). Tests of simple slopes revealed a significant decrease in

SUDS from Time 1 to Time 2 in the AL group (change = -.70, p = .007) and in the Control group (change = -1.28, p < .001).

3.3.6. Self Statements During Public Speaking.

3.3.6.1. Positive. For SSPS positive, the Time × Group interactions from Time 1 to Time 2 (p = .908), Time 2 to Time 3 (p = .240), and Time 1 to Time 3 (p = .418) were not significant.

3.3.6.1. Negative. For SSPS negative, the Time × Group interactions from Time 1 to Time 2 (p = .853), Time 2 to Time 3 (p = .212), and Time 1 to Time 3 (p = .268) were not significant.

3.3.7. Performance Rating Form. For PRF, the Time × Group interactions from Time 1 to Time 2 (p = .841), Time 2 to Time 3 (p = .143), and Time 1 to Time 3 (p = .177) were not significant.

3.3.8. Optional Speech. For the optional speech, the Time × Group interaction from Time 1 to Time 3 was not significant (p = .649).

3.4. Aim 2. Does the Number of Anxiety Related Labels Used During Exposure Predict Greater Attenuation of Fear Responding at Re-Test?

For each model tested, the significance of the Time × Number of Anxiety Labels interaction from Time 1 to Time 2, Time 2 to Time 3, and Time 1 to Time 3 are reported. For significant and marginally significant interactions, tests of whether participants at one standard deviation below the mean (-1SD) on Number of Anxiety Labels differ from those at one standard deviation above the mean (+1SD) at Time points 1, 2 and 3.

3.4.1. Heart rate.

3.4.1.1. *Anticipation*. For HR during anticipation of giving a speech, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .525), Time 2 to Time 3 (p = .387), and Time 1 to Time 3 (p = 783) were not significant.

3.4.1.2. *Recovery*. For HR during recovery following the speech, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .695), Time 2 to Time 3 (p = .325), and Time 1 to Time 3 (p = .517) were not significant.

3.4.2. Skin conductance level.

3.4.2.1. *Anticipation*. For SCL during anticipation of giving a speech, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .307), Time 2 to Time 3 (p = .878), and Time 1 to Time 3 (p = .400) were not significant.

3.4.2.2. *Recovery*. For SCL during recovery following the speech, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .926), Time 2 to Time 3 (p = .969), and Time 1 to Time 3 (p = .896) were not significant.

3.4.3. Non-specific skin conductance response.

3.4.3.1. *Anticipation*. Results are displayed in Figure 20. For SCR-NS during anticipation of giving a speech, the Time × number of anxiety labels interaction from Time 1 to Time 2 was not significant (p = .402). The interactions from Time 2 to Time 3 (b = -1.42, CI = -2.8 to 0, p = .045), and Time 1 to Time 3 were significant (b = -1.92, CI = -3.2 to -.6, p = .004) such that participants who used more affect labels during exposure had a steeper decline in SCR-NS over time. Tests of the difference between SCR-NS during anticipation for participants at +1SD and -1SD from the mean revealed no significant difference at Time 1 or Time 2 (ps > .117), and a significant difference at Time 3 (difference = 1.70, p = .001) such that participants at

+1SD from the mean on use of anxiety labels had fewer SCR-NS during anticipation of giving a speech than did participants at -1SD from the mean.

3.4.3.2. *Recovery*. For SCR-NS during recovery following the speech, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .574), Time 2 to Time 3 (p = .321), and Time 1 to Time 3 (p = .151) were not significant.

3.4.4. Personal Report of Public Speaking Anxiety. Results are displayed in Figure 21. For PRPSA, the Time × number of anxiety labels interaction from Time 2 to Time 3 was not significant (p = .961). The interaction from Time 1 to Time 2 (b = 15.76, CI = 1.6 to 29.9, p = .029) was significant, and the interaction from Time 1 to Time 3 was marginally significant (b = 15.40, CI = -2.7 to 33.5, p = .095) such that participants who used fewer affect labels during exposure had a steeper decline in PRPSA over time (interaction f^2 = .05). Tests of the difference between PRPSA for participants at +1SD and -1SD from the mean on number of anxiety labels revealed no significant difference at Time 1 (p = .368), and significant differences at Time 2 (difference = 11.70, p = .011) and Time 3 (difference = 11.52, p = .032) such that participants at +1SD from the mean on use of anxiety labels had higher PRPSA scores than did participants at -1SD from the mean.

3.4.5. Subjective Units of Distress. Results are displayed in Figure 22. For SUDS, the Time × number of anxiety labels interaction from Time 2 to Time 3 was not significant (p = .850). The interaction from Time 1 to Time 2 was significant (b = 1.72, CI = .3 to 3.2, p = .019), and the interaction from Time 1 to Time 3 was marginally significant (b = 1.58, -.1 to 3.2, p = .058) such that participants who used fewer affect labels during exposure had a steeper decline in SUDS over time (interaction f^2 = .05). Tests of the difference between SUDS for participants at +1SD and -1SD from the mean on number of anxiety labels revealed a marginally significant

difference at Time 1 (difference = .67, p = .083), and significant differences at Time 2 (difference = 1.52, p < .001) and Time 3 (difference = 1.45, p = .003) such that participants at +1SD from the mean on use of anxiety labels had higher SUDS scores than did participants at -1SD from the mean.

3.4.6. Self-Statements During Public Speaking.

3.4.6.1. *Positive*. For SSPS Positive, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .887), Time 2 to Time 3 (p = .760) and Time 1 to Time 3 (p = .938) were not significant.

3.4.6.2. *Negative*. For SSPS Negative, the Time × number of anxiety labels interactions from Time 1 to Time 2 (p = .615), Time 2 to Time 3 (p = .966), and Time 1 to Time 3 (p = .984) were not significant.

3.4.7. Performance Rating Form. Results are displayed in Figure 23 (higher scores indicate better self-rated performance). For PRF, the Time × number of anxiety labels interactions from Time 2 to Time 3 (p = .144) and Time 1 to Time 3 were not significant (p = .501). The interaction from Time 1 to Time 2 was significant (b = -9.87, CI = -18.0 to -1.8, p = .017) such that participants who used fewer affect labels during exposure had a steeper increase in PRF over time (interaction f^2 = .15). Tests of the difference between PRF scores for participants at +1SD and -1SD from the mean on number of anxiety labels revealed a marginally significant difference at Time 1 (difference = 4.86, p = .054), and significant differences at Time 2 (difference = 9.76, p < .001) and Time 3 (difference = 6.67, p = .024) such that participants at +1SD from the mean on use of anxiety labels had lower PRF scores than did participants at -1SD from the mean.

3.4.8. Optional speech. For optional speech, the Time × number of anxiety labels interaction from Time 1 to Time 3 was not significant (p = .666).

3.5. Aim 3. Does Incidental Emotion Regulation at Baseline Moderate or Predict Response to Exposure with Affect Labeling Versus Exposure Alone?

For each dependent variable, incidental emotion regulation is first tested as a moderator of response to exposure plus affect labeling versus exposure alone. The significance of the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2, Time 2 to Time 3, and Time 1 to Time 3 are reported. For significant and marginally significant interactions, the following simple effects are tested: (1) Group mean differences at Time 1, Time 2, and Time 3 at +1SD and -1SD from the mean of incidental emotion regulation. (2) Group slope differences from Time 1 to Time 2, Time 2 to Time 3, and/or Time 1 to Time 3 (depending on significant interactions) at +1SD and -1SD from the mean of incidental emotion regulation.

When the three-way interaction is either marginally significant or not significant, incidental emotion regulation is tested as a predictor of treatment outcome, and the significance of the Time × incidental emotion regulation interaction is reported for Time 1 to Time 2, Time 2 to Time 3, and Time 1 to Time 3. For significant and marginally significant interactions, tests of whether participants at -1SD from the mean on incidental emotion regulation differ from those at +1SD from the mean at Time points 1, 2 and 3.

3.5.1. Heart rate.

3.5.1.1. *Anticipation*. Moderation results are displayed in Figure 24. For HR during anticipation of giving a speech, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 (p = .269) and Time 2 to Time 3 (p = .344) were not significant, whereas the interaction from Time 1 to Time 3 was significant (b = 7.56, CI = .3 to 14.9, p = .042)

(interaction f^2 = .04). Tests of group mean differences at -1SD and +1SD from the mean revealed no differences at Time 1 (*p*s > .745), or Time 2 (*p*s > .443). At Time 3, for participants at -1SD from the mean on incidental emotion regulation, participants in the AL group had significantly lower HR than did participants in the Control group (difference = 6.77, p = .014). No group difference was found for participants at +1SD from the mean on incidental emotion regulation at Time 3 (p = .592). Tests of Group slope differences from Time 1 to Time 3 revealed that for participants at -1SD from the mean on incidental emotion regulation, the AL group had a significantly more negative slope than did the Control group (slope difference = 7.57, p = .019). No group slope difference was found for participants at +1SD from the mean on incidental emotion regulation (p = .543).

3.5.1.2. *Recovery*. Moderation results are displayed in Figure 25. For HR during recovery following the speech, the Time × Group × incidental emotion regulation interaction from Time 1 to Time 2 was not significant (p = .446), whereas the interaction from Time 2 to Time 3 was marginally significant (b = 5.09, CI = -.3 to 12.5, p = .063), and the interaction from Time 1 to Time 3 was significant (b = 8.48, CI = 2.2 to 14.8, p = .008) (interaction f^2 = .07). Tests of group mean differences at -1SD and +1SD from the mean revealed no differences at Time 1 (*p*s > .207) or Time 2 (*p*s > .301). At Time 3, for participants at -1SD from the mean on incidental emotion regulation, participants in the AL group had marginally significantly lower HR than participants in the Control group (difference = 4.72, p = .061). No group mean differences were observed for participants at +1SD from the mean on incidental emotion regulation at Time 3 (p = .329). Tests of Group slope differences from Time 1 to Time 3 revealed that for participants at -1SD from the mean on incidental emotion regulation, the AL group had a significantly more negative slope than did the Control group (slope difference =

7.56, p = .007). No group slope difference was observed for participants at +1SD from the mean on incidental emotion regulation (p = .262).

3.5.2. Skin conductance level.

3.5.2.1. *Anticipation*. For SCL during anticipation of giving a speech, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 (p = .602), Time 2 to Time 3 (p = .218), and Time 1 to Time 3 (p = .449) were not significant.

For SCL during anticipation of giving a speech, the Time × incidental emotion regulation interactions Time 1 to Time 2 (p = .664), Time 2 to Time 3 (p = .153), and from Time 1 to Time 3 (p = .296) were not significant.

3.5.2.2. *Recovery*. For SCL during recovery following the speech, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 (p = .272), Time 2 to Time 3 (p = .218) and Time 1 to Time 3 (p = .578) were not significant.

For SCL during recovery following the speech, the Time × incidental emotion regulation interactions from Time 1 to Time 2 (p = .565), Time 2 to Time 3 (p = .153), and Time 1 to Time 3 (p = .120) were not significant.

3.5.3. Non-specific skin conductance response.

3.5.3.1. *Anticipation*. For SCR-NS during anticipation of giving a speech, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 (p = .906), Time 2 to Time 3 (p = .369), and Time 1 to Time 3 (p = .267) were not significant.

Predictor results are displayed in Figure 26. For SCR-NS during anticipation of giving a speech, the Time × incidental emotion regulation interactions from Time 1 to Time 2 (p = .180) and Time 1 to Time 3 were not significant (p = .125), whereas the Time × incidental emotion regulation interaction from Time 2 to Time 3 was significant (b = .53, CI = .1 to .9, p = .009)

such that participants with deficits in incidental emotion regulation had more negative slopes from Time 2 to Time 3 than did participants with strengths in incidental emotion regulation. Tests of the difference between SCR-NS during anticipation for participants at +1SD and -1SD from the mean revealed no significant differences at Time 1 or Time 3 (*p*s > .331), and a significant difference at Time 2 (difference = .90, $p = .032$) such that participants at +1SD from the mean on incidental emotion regulation had lower SCR-NS than did participants at -1SD from the mean.

3.5.3.2. *Recovery*. Moderator results are displayed in Figure 27. For SCR-NS during recovery following the speech, the Time × Group × incidental emotion regulation interaction from Time 1 to Time 3 was not significant ($p = .419$), whereas the interaction from Time 2 to Time 3 was marginally significant ($b = -1.74$, CI = -3.7 to .2, $p = .080$), and the interaction from Time 1 to Time 2 was significant ($b = 2.37$, CI = .5 to 4.2, $p = .012$). Tests of group mean differences at -1SD and +1SD revealed no differences at Time 1 (*p*s > .432) or Time 3 (*p*s > .200). At Time 2, for participants at +1SD from the mean on incidental emotion regulation, participants in the AL Group had significantly higher SCR-NS than participants in the Control group (difference = .48, $p = .010$). At Time 2, no group differences emerged for participants at -1SD from the mean on incidental emotion regulation ($p = .487$). Tests of Group slope differences from Time 1 to Time 2 revealed that for participants at +1SD from the mean on incidental emotion regulation, the AL group had a significantly more positive slope than did the Control group (slope difference = .58, $p = .027$). No group slope difference was observed for participants at -1SD from the mean on incidental emotion regulation ($p = .236$). Tests of Group slope differences from Time 2 to Time 3 revealed that for participant at +1SD from the mean on incidental emotion regulation, the AL group had a significantly more negative slope than did the

Control group (slope difference = .71, p = .027). No group slope difference was observed for participants at -1SD from the mean on incidental emotion regulation (p = .670).

3.5.4. Personal Report of Public Speaking Anxiety. For PRPSA, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 (p = .389), Time 2 to Time 3 (p = .787), and Time 1 to Time 3 (p = .656) were not significant.

For PRPSA, the Time × incidental emotion regulation interactions from Time 1 to Time 2 (p = .470), Time 2 to Time 3 (p = .270) and Time 1 to Time 3 (p = .165) were not significant.

3.5.5. Subjective Units of Distress. Moderator results are displayed in Figure 28. For SUDS, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 3 (p = .838) and Time 2 to Time 3 (p = .138) were not significant, whereas the interaction from Time 1 to Time 2 was marginally significant (b = -.81, CI = -1.7 to .1, p = .085) (interaction f^2 = .05). Tests of group mean differences at -1SD and +1SD from the mean on incidental emotion regulation revealed no differences at Time 1 (ps > .457), Time 2 (ps > .102) or Time 3 (ps > .786). Tests of Group slope differences from Time 1 to Time 2 revealed no differences at +1SD or -1SD from the mean on incidental emotion regulation (ps > .122).

Predictor results are displayed in Figure 29. For SUDS, the Time × incidental emotion regulation interactions from Time 1 to Time 2 (p = .376) and Time 2 to Time 3 (p = .248) were not significant. The interaction from Time 1 to Time 3 was marginally significant (b = .46, CI = 0 to .9, p = .061) such that greater deficits in incidental emotion regulation were associated with greater decreases in SUDS over Time (interaction f^2 = .01). Tests of the difference between SUDS for participants at +1SD and -1SD from the mean on incidental emotion regulation revealed no significant differences at Time 1, Time 2, or Time 3 (ps > .113).

3.5.6. Self Statements During Public Speaking.

3.5.6.1. *Positive*. Moderator results for SSPS Positive are displayed in Figure 30 (higher scores indicate more positive cognitions). For SPSS Positive, the Time × Group × incidental emotion regulation interactions from Time 2 to Time 3 (p = .634) and Time 1 to Time 3 (p = .272) were not significant, whereas the interaction from Time 1 to Time 2 was marginally significant (b = 2.15, CI = -.1 to 4.3, p = .055) (interaction f^2 = .06). Tests of group mean differences at -1SD and +1SD from the mean on incidental emotion regulation revealed no differences at Time 1 (ps > .440). At Time 2, for participants at -1SD from the mean on incidental emotion regulation, participants in the AL Group had marginally significantly lower SSPS Positive scores than participants in the Control group (difference = 1.77, p = .096). No group differences emerged for participants at +1SD from the mean on incidental emotion regulation at Time 2 (p = .585). At Time 3, for participants at -1SD from the mean on incidental emotion regulation, participants in the AL Group had marginally significantly lower SSPS Positive scores than participants in the Control group (difference = 2.20, p = .071). No group differences emerged for participants at +1SD from the mean on incidental emotion regulation at Time 3 (p = .653). Tests of Group slope differences from Time 1 to Time 2 revealed no differences at +1SD or -1SD from the mean on incidental emotion regulation (ps > .141).

Predictor results for SSPS Positive are displayed in Figure 31 (higher scores indicate more positive cognitions). For SSPS Positive, the Time × incidental emotion regulation interaction from Time 2 to Time 3 was not significant (p = .605). The interactions from Time 1 to Time 2 (b = -1.28, CI = -2.3 to -.2, p = .016) and Time 1 to Time 3 (b = -1.56, CI = -2.9 to -.2, p = .025) were significant such that greater deficits in incidental emotion regulation were associated with greater increase in SSPS Positive over Time (f^2 = .01). Tests of the difference in SSPS Positive for participants at +1SD and -1SD from the mean on incidental emotion

regulation revealed no significant differences at Time 2, or Time 3 (*p*s > .746). At Time 1, participants at +1SD from the mean on incidental emotion regulation had significantly higher SSPS Positive scores than did participants at -1SD from the mean (difference = 1.70, $p = .025$).

3.5.6.2. *Negative*. Moderator results for SSPS Negative are displayed in Figure 32. For SPSS Negative, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 ($p = .389$) and from Time 1 to Time 3 ($p = .237$) were not significant, whereas the interaction from Time 2 to Time 3 was marginally significant ($b = 2.20$, CI = -.2 to 4.6, $p = .068$) (interaction $f^2 = .03$). Tests of group mean differences at -1SD and +1SD revealed no differences at Time 1 (*p*s > .388), Time 2 (*p*s > .278), or Time 3 ($p > .438$). Tests of Group slope differences from Time 2 to Time 3 revealed a marginally significant difference for participants at +1SD from the mean on incidental emotion regulation such that the AL group had a significantly more positive slope than did the Control group (slope difference = 1.96, $p = .060$). No group slope difference was found for participants at -1SD from the mean on incidental emotion regulation from Time 2 to Time 3 ($p = .446$).

Predictor results for SSPS Negative are displayed in Figure 33. For SSPS Negative, the Time × incidental emotion regulation interaction from Time 1 to Time 2 was not significant ($p = .241$). The interactions from Time 2 to Time 3 ($b = 1.13$, CI = 0 to 2.2, $p = .047$) and Time 1 to Time 3 ($b = 1.80$, CI = .4 to 3.2, $p = .013$) were significant such that greater deficits in incidental emotion regulation were associated with greater decreases in SSPS Negative over Time (interaction $f^2 = .00$). Tests of the difference in SSPS Negative for participants at +1SD and -1SD from the mean on incidental emotion regulation revealed no significant differences at Time 2, or Time 3 (*p*s > .255). At Time 1, participants at +1SD from the mean on incidental emotion

regulation had significantly lower SSPS Negative scores than did participants at -1SD from the mean (difference = 1.83, $p = .041$).

3.5.7. Performance Rating Form. For PRF, the Time × Group × incidental emotion regulation interactions from Time 1 to Time 2 ($p = .274$), Time 2 to Time 3 ($p = .316$), and Time 1 to Time 3 ($p = .944$) were not significant.

For PRF, the Time × incidental emotion regulation interactions from Time 1 to Time 2 ($p = .470$), Time 2 to Time 3 ($p = .216$), and from Time 1 to Time 3 ($p = .165$) were not significant.

3.5.8. Optional speech. For the optional speech, the Time × Group × incidental emotion regulation interaction from Time 1 to Time 3 was not significant ($p = .988$).

For the optional speech, the Time × incidental emotion regulation interaction from Time 1 to Time 3 was not significant ($p = .689$).

CHAPTER 4

Discussion

The current study had three primary aims. The first aim was to test whether exposure combined with affect labeling resulted in greater attenuation of fear responding than did exposure alone. The second aim was to assess whether the number of anxiety labels used during exposure in the affect labeling group predicted greater fear attenuation. The final aim was to test whether incidental emotion regulation capacity at baseline could be used to predict who would benefit from exposure plus affect labeling compared to exposure alone. If no moderation was found, incidental emotion regulation was assessed as a predictor of who would respond to exposure regardless of group assignment. All three aims were tested on the same set of dependent variables. These included heart rate, skin conductance level, and non-specific skin conductance response during anticipation of and recovery from a speech, self-reported trait public speaking anxiety, anxiety level during public speaking, negative and positive self statements during public speaking, self rated public speaking performance, and avoidance of giving an additional speech.

4.1. Aim 1: Does Affect Labeling Enhance Exposure Effectiveness Compared to Exposure Alone?

Consistent with hypotheses, participants in the exposure plus affect-labeling group had a steeper decline in heart rate and non-specific skin conductance responses during recovery following the speech than did participants in the exposure alone condition. This finding is consistent with previous research showing that exposure combined with affect labeling results in greater reduction in galvanic skin response than exposure alone (Kircanski et al., 2012; Tabibnia

et al., 2008). It is notable that the effect was found only for skin conductance response and heart rate during recovery following the speech and not anticipation of the speech. Although research on public speaking tasks generally focuses on anticipation of rather than recovery from speaking, post-event processing is common in patients with social anxiety and relates both to the severity of social anxiety symptoms and predicts subsequent avoidance of similar social situations (Rachman, Grüter-Andrew, & Shafran, 2000). Therefore, the effect of affect labeling on reducing physiological activation during recovery following the speech may reflect reduction in negative post event processing and rumination.

Additional analyses were conducted comparing participants in the exposure alone condition to those in the exposure plus affect labeling condition who were "high" labelers (i.e. in the top 50th percentile of number of labels chosen). Because there were participants who chose no or very few affect labels during exposure, limiting the sample to those in the top 50th percentile included only those participants who chose at least 7 labels (out of 20 possible) during exposures. When only high labelers were included in the group comparison, consistent with hypotheses, participants in the affect labeling group had marginally significantly lower heart rate and significantly fewer non-specific skin conductance responses during anticipation of giving a speech, and significantly lower heart rate and marginally significantly fewer non-specific skin conductance responses during recovery following the speech. Although these findings replicate previous research, causality cannot be inferred given that participants were not randomly assigned to the high labeling group within the affect labeling condition. It is possible that high labelers in the affect labeling condition differed in some meaningful way from participants assigned to the exposure alone condition that explains the significant group differences. However, it is important to note that the analytic approach controlled for baseline levels of the

dependent variables, so group differences at time points 2 and 3 cannot be explained by preexisting baseline group differences on the dependent measures. Because many participants did not choose the affect labels provided, a more effective approach for future studies may be to allow participants to generate their own affective labels as in Kircanski et al (2012). However, researchers have yet to assess whether matching participants' labels to their own affective experience produces greater benefit than providing predetermined labels from which participants can choose. Regardless of whether participants generate their own labels or not, future studies should ensure that all participants in the labeling group engage, at least to some extent, in affect labeling and are not given the option to completely refrain from labeling.

Contrary to hypotheses, on self-report measures of anxiety, participants in the affect labeling condition had marginally significantly less improvement in self rated performance than participants in the exposure alone condition. In addition, high labelers in the affect labeling condition had marginally significantly less improvement in trait public speaking anxiety and anxiety while speaking than did participants in the exposure alone condition. These findings however were limited to the change from Time 1 to Time 2 and were no longer significant at Time 3, and no group differences remained by the third time point. In a previous study, the benefit of affect labeling during exposure was found only for galvanic skin response and not for self-report measures (Kircanski et al., 2012). One possible explanation provided by Kircanski and colleagues is that people tend to predict that affect labeling will not be an effective strategy for reducing distress (Lieberman et al., 2011). Another possibility is that by repeatedly labeling anxiety and feared outcomes during exposure, high labelers in the affect labeling group were trained to report increased anxiety symptoms on self report measures administered during subsequent assessments. Consistent with mindfulness based approaches to the treatment of

anxiety such as Acceptance and Commitment Therapy (ACT), it is possible that participants who engaged in affect labeling became more willing to report anxiety symptoms at follow-up assessments, but were less distressed by these symptoms, and therefore less physiologically reactive. A phrase often used in ACT regarding mindfulness practice is that the goal is not to feel better, but to get better at feeling (Hayes et al., 1999). Therefore, although not assessed in the current study, perhaps through labeling, participants became more attuned to and accepting of their emotions and anxiety symptoms, which led to increased symptom reporting, but decreased physiological activation. Future studies that include training in affect labeling should measure acceptance of anxiety in addition to anxiety symptoms.

Inconsistent with previous research (Kircanski et al., 2012), we did not find that affect labeling reduced avoidance of public speaking. This failure to replicate previous findings may be attributable to floor effects on our measure of avoidance that limited statistical power to detect group differences. Participants were given the option to give an additional speech in exchange for $5. Although approximately 39% of participants avoided the additional speech at the baseline assessment, following exposure and at one-week follow-up, only 13% of participants avoided the additional speech. Given the sample size, this dichotomous outcome did not allow enough variability to detect group differences. Future studies should use a measure of avoidance that allows for greater variability (e.g. amount of time speaking) or should use a larger sample size to increase statistical power.

4.2. Aim 2: Does the Number of Anxiety Related Words Used During Exposure Predict Greater Attenuation of Fear Responding at Re-Test?

Consistent with hypotheses, the more anxiety labels participants chose during exposure, the fewer non-specific skin conductance responses participants had during anticipation of giving

a speech by the one-week follow-up. These findings are consistent with previous research in spider fearful participants where it was found that participants who used more anxiety words during affect labeling had a greater reduction in spontaneous skin conductance response at re-test than did participants who used fewer anxiety words (Kircanski et al., 2012). Perhaps the use of more affect labels during exposure lead to greater activation in PFC-Amygdala pathways. If participants with anxiety show weaker connectivity between areas of the PFC (such as RVLPFC) and the amygdala, it is possible that the number of repetitions of activation in these pathways during exposure is positively correlated with the strength of PFC-amygdala connectivity following completion of exposure. Consistent with the principle of neural plasticity, which states that repetition of a process can increase efficiency and efficacy of that process through changes in neuron function, chemical profile, and structure (Anderson, 2010; Kandel & Schwartz, 1982), greater activation of PFC-amygdala neural pathways as a result of more frequent labeling may have produced greater neural change and ultimately more effective down regulation of physiological fear responding.

Contrary to hypotheses, following exposure, participants who used fewer affect labels during exposure showed greater improvement in trait public speaking anxiety, anxiety while speaking, and self reported public speaking performance than did participants who used more affect labels during exposure. Consistent with findings from study Aim 1, self reported symptoms did not align with physiological measures of anxious responding. Perhaps participants who chose more anxiety labels during exposure reported more symptoms in order to maintain consistency across different self-report assessments. Festinger's theory of cognitive dissonance (1957) suggests that individuals are motivated to maintain internal consistency, and discomfort arises when beliefs and actions are inconsistent across different settings. Therefore,

perhaps when participants engaged in more frequent labeling of negative emotions, in order for their responses to be consistent from one setting to the next, they reported more anxiety symptoms despite having less physiological arousal. Given a longer lag time between labeling and the assessment of self-report symptoms, perhaps the importance of consistency on self-report measures would be diminished. It is also possible that, had participants been followed for a longer period of time, those who labeled more frequently would show continued improvement on self-report measures, while those who labeled less frequently would show a return of symptoms.

4.3. Aim 3: Does Incidental Emotion Regulation at Baseline Moderate or Predict Response to Exposure with Affect Labeling Versus Exposure Alone?

Contrary to hypotheses, we found that participants who had deficits incidental emotion regulation at baseline benefited more from exposure combined with affect labeling than exposure alone on measures of physiology including heart rate during speech anticipation and recovery, and non-specific skin conductance responses during recovery. Based on previous research that supports the matching hypothesis (Engebretson, Matthews, & Scheier, 1989; Niles et al., 2014; Stanton et al., 2000), we hypothesized that participants with strengths in incidental emotion regulation at baseline would show greater benefit from an intervention that included affect labeling. Previous research however did not measure physiological activation as the outcome, but instead measured self-reported symptoms. Therefore, the differences in findings may be attributed to the different method of outcome assessment. In healthy participants, affect labeling leads to a reduction in self-reported distress while viewing negative images by increasing activation in areas of the PFC and decreasing activation in the amygdala. It is possible that participants who showed less benefit from incidental emotion regulation have greater deficits in PFC-Amygdala connectivity, and as a result benefited more from an intervention that

specifically targeted these connectivity deficits. Neural activation however was not directly assessed in the current project, and future studies that include affect labeling as a strategy for enhancing exposure effectiveness would benefit from inclusion of measures of neural activation before and after the intervention.

Contrary to hypotheses, from Time 1 to Time 2, we found that participants with greater deficits in incidental emotion regulation did better in exposure alone than exposure plus affect labeling on self-reported negative and positive cognitions and anxiety during public speaking. Results should be interpreted with caution given that effects were marginally significant and were not maintained at one-week follow-up. Consistent with findings from study Aims 1 and 2, findings on the self-report measures were not consistent with findings on the physiological measures. Again, one possibility is that by labeling anxiety and feared outcomes during exposure, participants in the affect labeling group were trained to report increased anxiety symptoms on self report measures administered during subsequent BATs. The moderation effects however suggest that only participants with deficits in incidental emotion regulation later reported more symptoms. It is possible that these participants' incidental emotion regulation ability improved, and as a result, they were more likely to report symptoms because they found this strategy more effective at reducing physiological arousal.

For marginally significant moderator findings, incidental emotion regulation capacity was also tested as an overall predictor of outcome (regardless of group). Significant prediction effects were found for negative and positive cognitions during public speaking, and a marginally significant effect was found for self-reported anxiety during public speaking. Across all three dependent measures, participants with deficits in incidental emotion regulation at baseline showed the greatest improvement in anxious responding following exposure. More specifically,

participants with greater deficits in incidental emotion regulation reported significantly more anxiety symptoms at baseline than participants with less deficit, but following completion of exposure, those with deficits no longer differed from those without deficits. Because incidental emotion regulation is a proxy for prefrontal-amygdala connectivity, these findings imply that exposure, regardless of the inclusion of affect labeling, is particularly effective for participants with greater deficits in prefrontal-amygdala circuitry.

4.4. Differential Findings for Self-Report and Physiological Measurement

The lack of synchrony between the physiological measurement and self reported anxiety is an important consideration for research on the treatment of anxiety. At baseline, the increase in heart rate, skin conductance level, and non-specific skin conductance responses during anticipation did not correlate with self-report measures of anxiety. The only significant correlation with a self-report measure was found between non-specific skin conductance fluctuation increase during anticipation and depressive symptoms. This suggests that physiology may index a different facet of anxious responding than does self-reported anxiety. It is also notable that the only significant correlate of increased avoidance of the optional speech at baseline was the amount of increase in skin conductance response from baseline to recovery. Self-report measures of anxiety were not significantly associated with behavioral avoidance. Given the importance of avoidance in the maintenance of anxiety disorders, physiology, particularly during recovery following a stressor, may be an important indicator of disorder severity in addition to self-reported symptoms.

4.5. Summary and Implications

In sum, the current research supports the theory that affect labeling can enhance exposure effectiveness. Patients with anxiety show deficits in prefrontal amygdala connectivity (Hahn et

al., 2011; Kim et al., 2011), and repeated activation of prefrontal regions that project to the amygdala through exposure and affect labeling can lead to a reduction in physiological activation in response to an anxiety provoking stimulus. The results of the current study indicate that the more a fearful individual labels their emotional experience during exposure, the greater the reduction in galvanic skin response and heart rate (measures of fear arousal) when they next encounter the feared stimulus. The benefit of affect labeling was not shown for self-reported anxiety, which may require longer-term follow up than was possible in the current study. Finally, adding affect labeling to exposure was particularly beneficial (on physiological outcomes only) for individuals who showed deficits in incidental emotion regulation, which is an indicator of poor prefrontal-amygdala connectivity. This finding provides further evidence that targeting prefrontal-amygdala circuitry in anxiety patients using tasks that activate key regions involved in emotion regulation can improve treatment effectiveness, and that such interventions will be particularly effective for patients who show the greatest deficits in this circuit.

Table 1. Measures Used at Each Stage of the Study.

	BAT-1	Exp-1	Exp-2	BAT-2	BAT-3
MINI	X				
PRPSA	X			X	X
SSPS	X			X	X
PHQ	X			X	X
SPIN	X			X	X
PRF	X			X	X
SUDS	X	X	X	X	X
HR, SCR-NS, SCL	X			X	X
Optional Speech	X			X	X
% Anxiety Labels		X	X		
Incidental Emotion Regulation		X			
ECQ		X			

Note. MINI=MINI Neuropsychiatric Interview for Social Anxiety; PRPSA=Personal Report of Public Speaking Anxiety; SSPS=Self-Statements During Public Speaking Questionnaire; PHQ=Patient Health Questionnaire; SPIN=Social Phobia Inventory; PRF=Performance Rating Form; SUDS=Subjective Units of Distress Scale; HR=Heart Rate; SCR-NS=Non Specific Skin Conductance Response; SCL=Skin Conductance Level; % Anxiety Labels=Percent of anxiety labels chosen; ECQ=Exposure Credibility Questionnaire

Table 2. Number of Missing Data Points and Reason for Missing Data for Study Variables by Time Point

	Time 1	Time 2	Time 3	Reasons Missing
Age	6	--	--	Participant Did Not Complete Experimenter Error
Gender	0	--	--	
Ethnicity	5	--	--	Participant Did Not Complete Experimenter Error
Write in a Journal	10	--	--	Participant Did Not Complete Experimenter Error
Student	3	--	--	Participant Did Not Complete Experimenter Error
English First Language	5	--	--	Participant Did Not Complete Experimenter Error
Born in United States	7	--	--	Participant Did Not Complete Experimenter Error
MINI	1	--	--	Inadvertently Deleted
PRPSA	4	2	1	Inadvertently Deleted
SSPS	5	2	1	Inadvertently Deleted Experimenter Error
PHQ	4	2	1	Inadvertently Deleted
SPIN	4	2	1	Inadvertently Deleted
PRF	5	2	1	Inadvertently Deleted Experimenter Error
SUDS	1	0	0	Computer Program Failure
HR	1	2	1	Experimenter Error
SCR-NS/SCL	9	10	7	Participant Non Response
Optional Speech	0	0	0	
% Anxiety Labels	0	0	--	
Incidental Emotion Regulation	8	--	--	Participant Did Not Complete Experimenter Error
ECQ	2	--	--	Experimenter Error

Note. MINI=MINI Neuropsychiatric Interview for Social Anxiety; PRPSA=Personal Report of Public Speaking Anxiety; SSPS=Self-Statements During Public Speaking Questionnaire; PHQ=Patient Health Questionnaire; SPIN=Social Phobia Inventory; PRF=Performance Rating Form; SUDS=Subjective Units of Distress Scale; HR=Heart Rate; SCR-NS=Non Specific Skin Conductance Response; SCL=Skin Conductance Level; % Anxiety Labels=Percent of anxiety labels chosen; ECQ=Exposure Credibility Questionnaire

Table 3. Descriptive Statistics and Tests of Baseline Group Differences for Demographics

	Overall (n=100)	AL (n=52)	Control (n=48)	Statistical Test
Age (mean (sd))	25.3 (9.1)	24.7 (8.9)	26.1 (9.3)	$t(92) = .7$
Female	80% (80/100)	77% (40/52)	83% (40/48)	$\chi^2(1) = .6$
Ethnicity				Fisher's $p = .83$[a]
Asian	55% (52/95)	57% (28/49)	52% (24/46)	
Hispanic	16% (15/95)	14% (7/49)	17% (8/46)	
African American	6% (6/95)	6% (3/49)	7% (3/46)	
Caucasian	14% (13/95)	10% (5/49)	17% (8/46)	
Other	9% (9/95)	12% (6/49)	7% (3/46)	
Write in a Journal	17% (15/90)	14% (6/44)	20% (9/46)	$\chi^2(1) = .6$
Student	92% (89/97)	94% (47/50)	89% (42/47)	$\chi^2(1) = .7$
English First Language	63% (60/95)	63% (30/48)	64% (30/47)	$\chi^2(1) = 0$
Born in United States	75% (70/93)	79% (37/47)	72% (33/46)	$\chi^2(1) = .6$
Social Anxiety Disorder	36% (36/99)	35% (18/51)	38% (18/48)	$\chi^2(1) = .8$
PRPSA Score	133.0 (14.7)	133.2 (14.7)	132.8 (16.1)	$t(94) = -.1$
PHQ Score	5.6 (4.3)	5.9 (4.4)	5.3 (4.2)	$t(94) = -.7$
SPIN Score	6.4 (3.0)	6.7 (2.8)	6.1 (3.2)	$t(94) = -1.0$

* $p < .05$

[a]Fisher's exact test was used to test for significance due to small cell sizes

Note. AL = Affect Labeling Group; Control = Control Group; PRPSA=Personal Report of Public Speaking Anxiety; PHQ=Patient Health Questionnaire; SPIN=Social Phobia Inventory

Table 4. Descriptive Statistics by Group and Time for BAT Measures

	AL			Control		
	T1 (n=52)	T2 (n=43)	T3 (n=41)	T1 (n=48)	T2 (n=42)	T3 (n=40)
	Mean (SD)					
HR						
Baseline	77.1 (12.6)	77.3 (11.7)	77.4 (12.5)	72.4 (10.0)	73.9 (11.0)	76.5 (13.6)
Anticipation	81.9 (13.3)	81.3 (12.2)	80.4 (13.3)	78.3 (12.1)	78.1 (11.9)	79.8 (13.1)
Recovery	75.5 (13.1)	75.5 (10.5)	74.7 (12.7)	71.4 (9.7)	71.0 (10.3)	72.8 (12.0)
SCL						
Baseline	2.0 (1.1)	1.9 (1.2)	1.9 (1.2)	2.1 (1.1)	1.8 (1.1)	2.1 (1.7)
Anticipation	3.0 (1.6)	2.6 (1.6)	2.6 (1.5)	3.0 (1.2)	2.6 (1.2)	2.9 (1.8)
Recovery	3.1 (1.4)	3.2 (1.4)	3.3 (1.2)	3.2 (1.2)	3.0 (1.2)	3.3 (1.8)
SCR-NS						
Baseline	.8 (1.3)	.5 (1.2)	.8 (1.3)	.3 (.8)	.2 (.8)	.8 (1.4)
Anticipation	2.5 (1.9)	1.9 (2.0)	1.8 (2.4)	2.5 (1.7)	1.9 (2.2)	2.0 (1.8)
Recovery	.8 (1.3)	.6 (1.3)	.3 (.7)	.6 (.9)	.4 (.7)	.6 (.9)
PRPSA	133.2 (14.7)	126.2 (17.6)	122.7 (17.8)	132.8 (16.1)	121.5 (16.1)	117.3 (15.1)
SUDS	4.9 (1.4)	3.7 (1.8)	3.3 (1.8)	4.7 (1.6)	3.5 (1.5)	3.4 (1.6)
SSPS Positive	14.4 (3.9)	15.7 (3.5)	15.9 (4.1)	14.8 (4.0)	16.5 (3.3)	17.5 (3.1)
SSPS Negative	16.5 (4.3)	14.3 (4.7)	13.4 (4.5)	16.5 (4.1)	14.2 (3.8)	12.5 (3.8)
PRF	25.7 (9.1)	35.6 (10.1)	36.9 (10.1)	24.0 (8.0)	32.6 (8.3)	37.6 (9.9)
Optional Speech (%)	60% (31/52)	88% (38/43)	88% (36/41)	63% (30/48)	83% (35/42)	85% (34/40)

Note. HR=Heart Rate; SCL=Skin Conductance Level; SCR-NS=Non Specific Skin Conductance Response; PRPSA=Personal Report of Public Speaking Anxiety; SUDS=Subjective Units of Distress Scale; SSPS=Self-Statements During Public Speaking Questionnaire; PRF=Performance Rating Form

Table 5. Descriptive Statistics by Group and Time for EXP measures

	EXP 1 (n=49)		EXP 2 (n=43)	
	Mean Percentage (SD)			
Affect Labels				
Anxiety Words	36% (26)		37% (26)	
Anger Words	7% (11)		9% (11)	
Sadness Words	10% (13)		10% (13)	
Other	46% (35)		43% (33)	
Feared Outcome	73% (34)		75% (32)	
	AL (n=49)	Control (n=46)	AL (n=43)	Control (n=43)
	Mean (SD)			
SUDS				
Beginning	4.7 (1.8)	4.0 (1.7)	3.6 (1.9)	4.0 (1.7)
End	5.1 (1.9)	4.4 (2.0)	3.8 (1.8)	3.5 (1.7)
ECQ	19.0 (5.5)	19.6 (6.4)	--	--

Note. SUDS = Subjective Units of Distress; ECQ = Exposure Credibility Questionnaire

Table 6. Correlations Among Key Study Variables

	PRPSA	SSPSp	SSPSn	PHQ	SPIN	PRF	SUDS	HRb	HRa-b	HRr-b	SCLb	SCLa-b	SCLr-b	SCRb	SCRa-b	SCRr-b	Opt	% anx	% fo
PRPSA	1.00																		
SSPSp	-.28**	1.00																	
SSPSn	.50***	-.36***	1.00																
PHQ	.27**	-.15	.29**	1.00															
SPIN	.59***	-.22*	.33**	.27**	1.00														
PRF	-.43***	.26*	-.57***	-.17	-.20*	1.00													
SUDS	.32**	-.10	.46***	.21*	.19	-.40***	1.00												
HRb	.10	-.10	.00	-.19	-.03	-.04	-.08	1.00											
HR a-b	.07	-.09	.05	-.06	-.03	-.05	.12	-.06	1.00										
HR r-b	.00	-.04	-.02	.17	.16	.10	-.04	-.26*	.45***	1.00									
SCLb	.05	.08	.03	-.10	.03	.00	.16	.25*	.13	.08	1.00								
SCL a-b	.09	-.05	.07	.25*	.12	-.13	.07	-.17	-.04	-.04	-.67***	1.00							
SCL r-b	-.05	-.10	.01	.18	.02	-.04	-.02	-.22*	-.16	-.02	-.80***	.83***	1.00						
SCRb	.00	.03	-.09	-.10	.05	.09	-.02	.39***	.08	.02	.48***	-.41***	-.52***	1.00					
SCR a-b	.16	-.10	.14	.22*	.03	-.16	.19	-.09	.06	.01	-.08	.42***	.20	-.35***	1.00				
SCR r-b	-.12	-.06	.09	.21*	-.06	-.02	.11	-.30**	-.18	.01	-.27**	.33**	.49***	-.58***	.36***	1.00			
Opt	.05	.20	-.18	-.19	.07	.15	-.19	-.10	-.05	.06	-.02	-.12	-.10	.00	-.15	-.34***	1.00		
% anx	.09	-.28	.31*	.30*	.15	-.25	.24	-.15	.05	.02	-.19	-.08	-.03	-.04	-.07	-.02	.04	1.00	
% fo	.06	-.12	.44**	.26	.11	-.35*	.35*	-.18	-.11	-.13	-.16	.01	.06	-.08	.04	.15	-.09	.74***	1.00
IER	-.01	.26*	-.25*	-.23	.00	-.02	-.16	.08	.06	.04	-.08	.02	-.04	.00	-.14	-.16	.06	-.08	-.22

* $p < .05$, ** $p < .01$, *** $p < .001$

Note. PRPSA = Personal Report of Public Speaking Anxiety; SSPSp = Self Statements During Public Speaking – Positive; SSPSn = Self Statements During Public Speaking – Negative; PHQ = Patient Health Questionnaire; SPIN = Social Phobia Inventory; PRF = Performance Rating Form; SUDS = Subjective Units of Distress; HR = Heart Rate; SCL = Skin Conductance Level SCR-NS = Non Specific Skin Conductance Response; b = anticipation; a-b = anticipation minus baseline; r-b = recovery minus baseline; Opt = optional speech; % anx = percent anxiety labels chosen; % fo = percent feared outcomes chosen; IER = Incidental Emotion Regulation

Table 7. Descriptive Statistics Completers vs. Dropout for Relevant Study Variables

	Completers (n=81)	Dropout (n=19)	*t* statistic
	Mean (SD)		
HR			
Baseline	74.0 (11.5)	78.7 (11.6)	t(97) = -1.6
Anticipation	78.9 (1.4)	85.7 (11.2)	t(97) = -2.1*
Recovery	72.5 (11.5)	78.1 (12.0)	t(96) = -1.9†
SCL[a]			
Baseline	.5 (.5)	.5 (.7)	t(89) = 0
Anticipation	1.0 (.4)	1.1 (.5)	t(89) = -.9
Recovery	1.1 (.3)	1.2 (.5)	t(89) = -1.0
SCR-NS[b]			
Baseline	.5 (1.2)	.6 (1.2)	*z*=.1
Anticipation	2.3 (1.7)	3.4 (1.9)	*z*=-1.8†
Recovery	.9 (1.2)	.6 (1.1)	*z*=-1.4
PRPSA	131.9 (15.1)	137.7 (16.0)	t(94) = -1.4
SUDS (BAT)	4.7 (1.5)	5.0 (1.5)	t(97) = -.8
SSPS Pos	15.0 (3.9)	12.8 (3.7)	t(93) = 2.1*
SSPS Neg	16.4 (4.1)	16.9 (4.7)	t(93) = -.5
PRF	25.7 (8.6)	20.9 (7.6)	t(93) = 2.2*
Optional Speech (%)	65% (53/81)	42% (8/19)	$\chi^2(1) = 3.5$†
PHQ	14.4 (4.0)	15.4 (5.4)	t(94) = -.8
SPIN	9.2 (3.1)	10.1 (2.6)	t(94) = -1.1
SUDS (exposure)			
Beginning	4.2 (1.7)	4.9 (1.9)	t(93) = -1.5
End	4.7 (2.0)	5.1 (2.0)	t(88) = -.7
ECQ	19.1 (5.6)	19.8 (7.2)	t(96) = -.5

† $p < .10$, * $p < .05$

[a] SCL values were log transformed

[b] Mann-Whitney test was used because variable is non-normal (count)

Note. HR=Heart Rate; SCL=Skin Conductance Level; SCR-NS=Non Specific Skin Conductance Response; PRPSA=Personal Report of Public Speaking Anxiety; SUDS=Subjective Units of Distress Scale; SSPS=Self-Statements During Public Speaking Questionnaire; PRF=Performance Rating Form; PHQ = Patient Health Questionnaire; SPIN = Social Phobia Inventory; ECQ = Exposure Credibility Questionnaire

Figure 1. Study Procedure

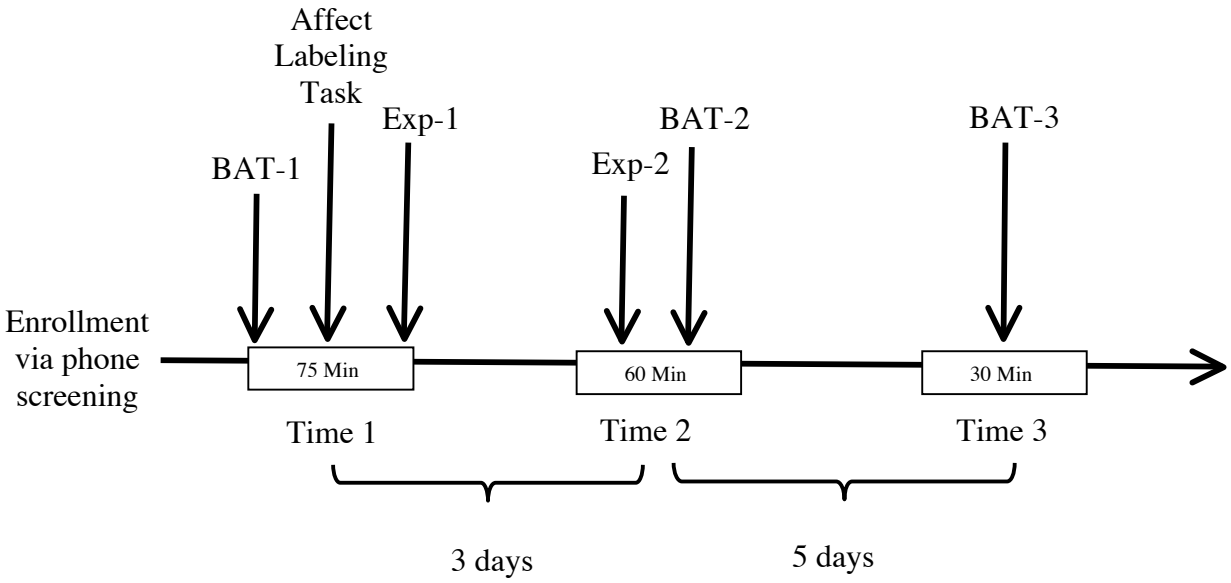
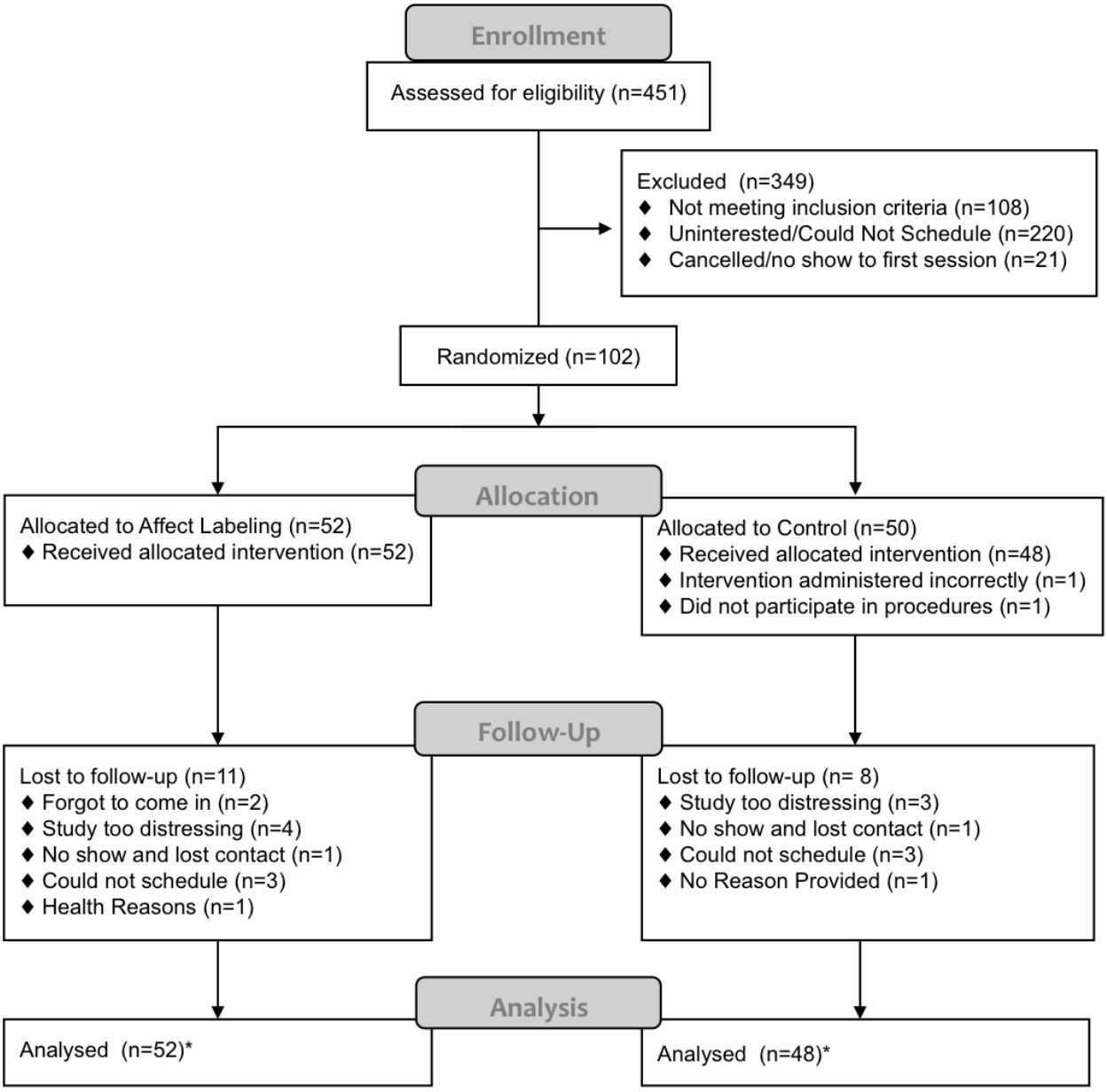


Figure 2. Participant Flow Chart



* See Table 2 for additional information regarding missing data

Figure 3. Heart Rate Over Time by Group during Anticipation

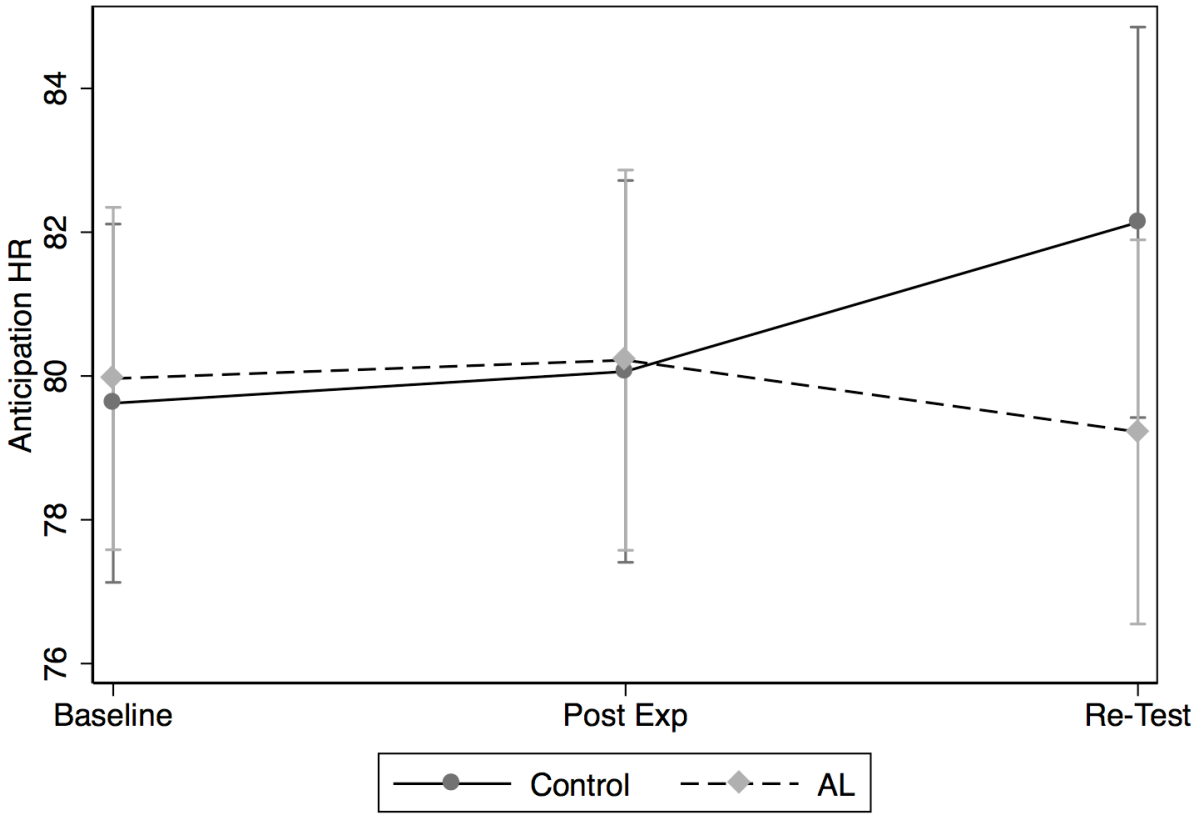


Figure 4. Heart Rate During Recovery Over Time by Group

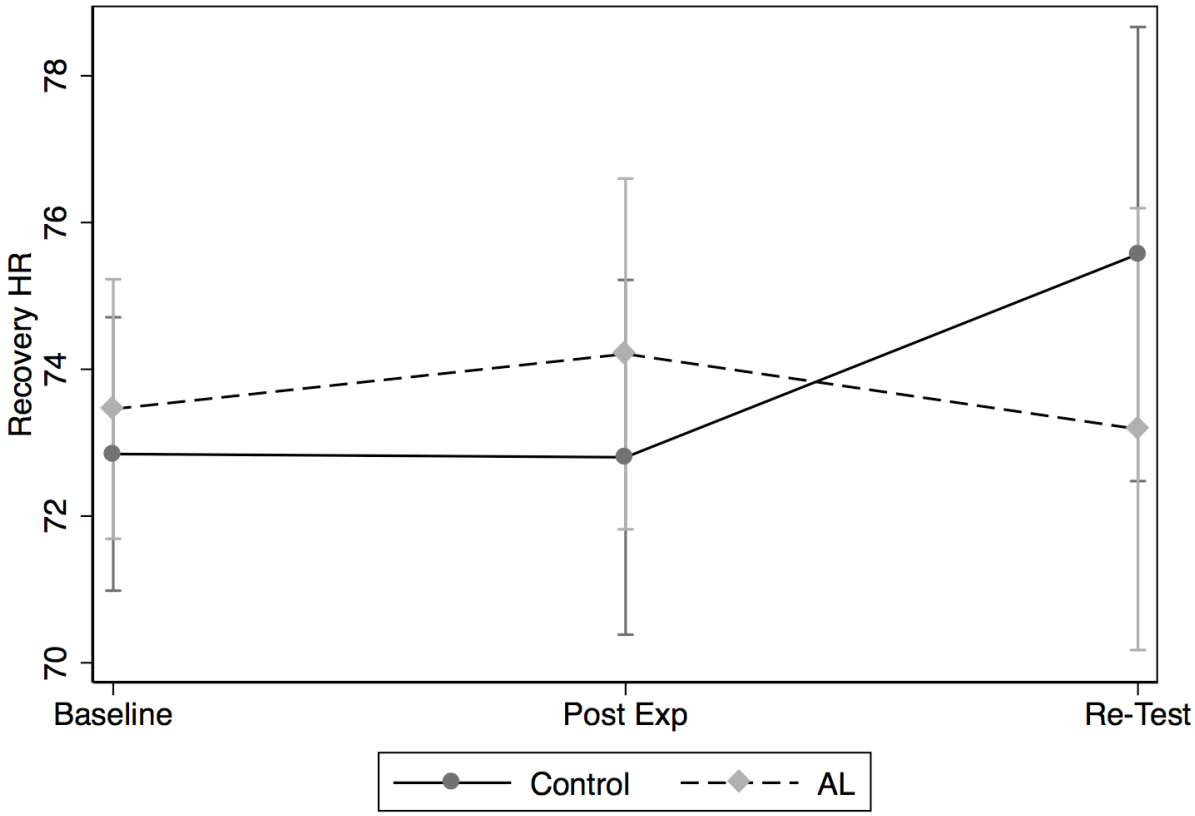


Figure 5. Skin Conductance Level During Anticipation Over Time by Group

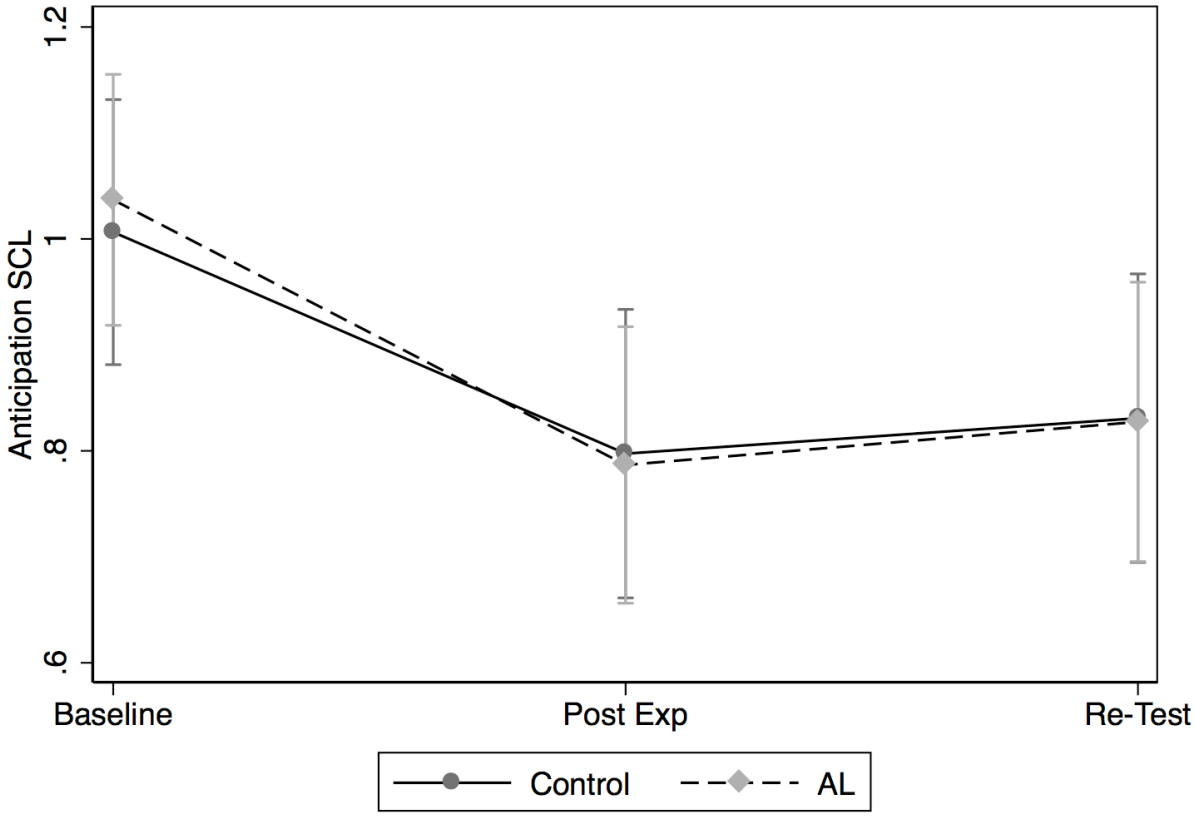


Figure 6. Skin Conductance Level During Recovery Over Time by Group

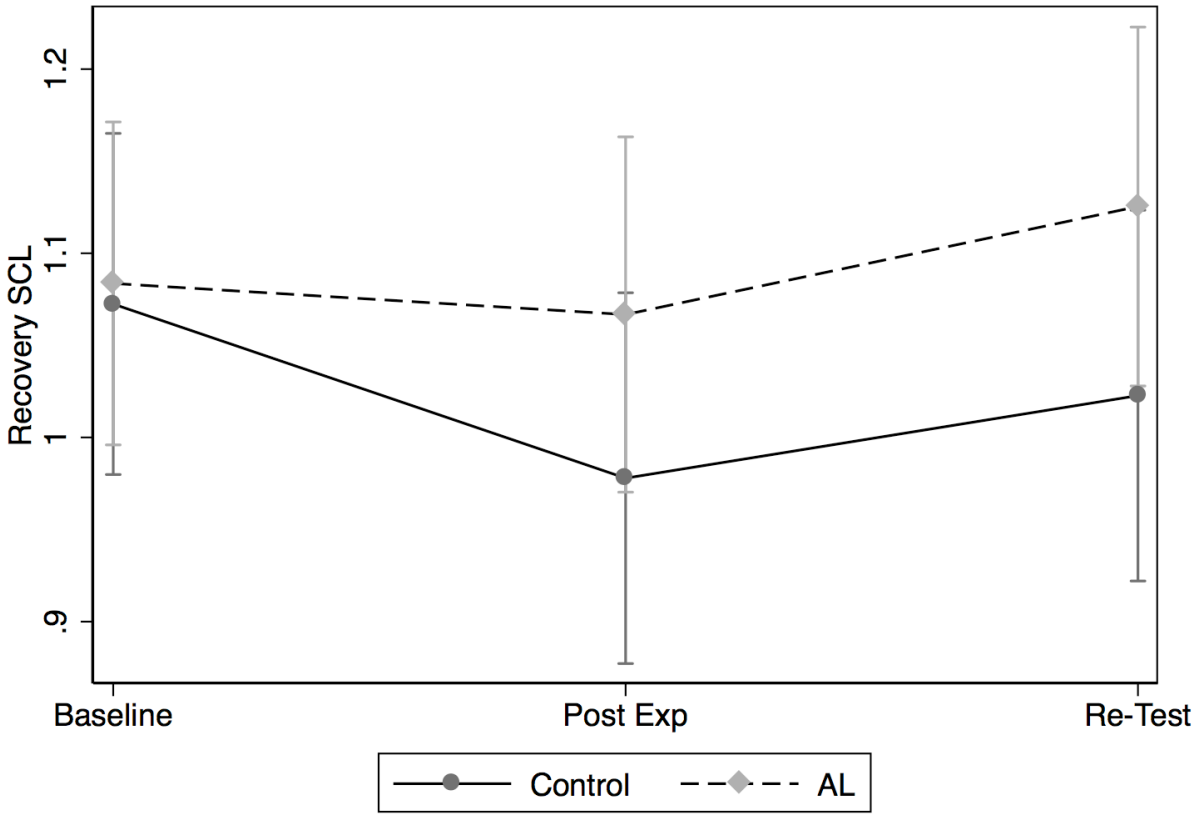


Figure 7. Non-Specific Skin Conductance Response During Anticipation Over Time by Group

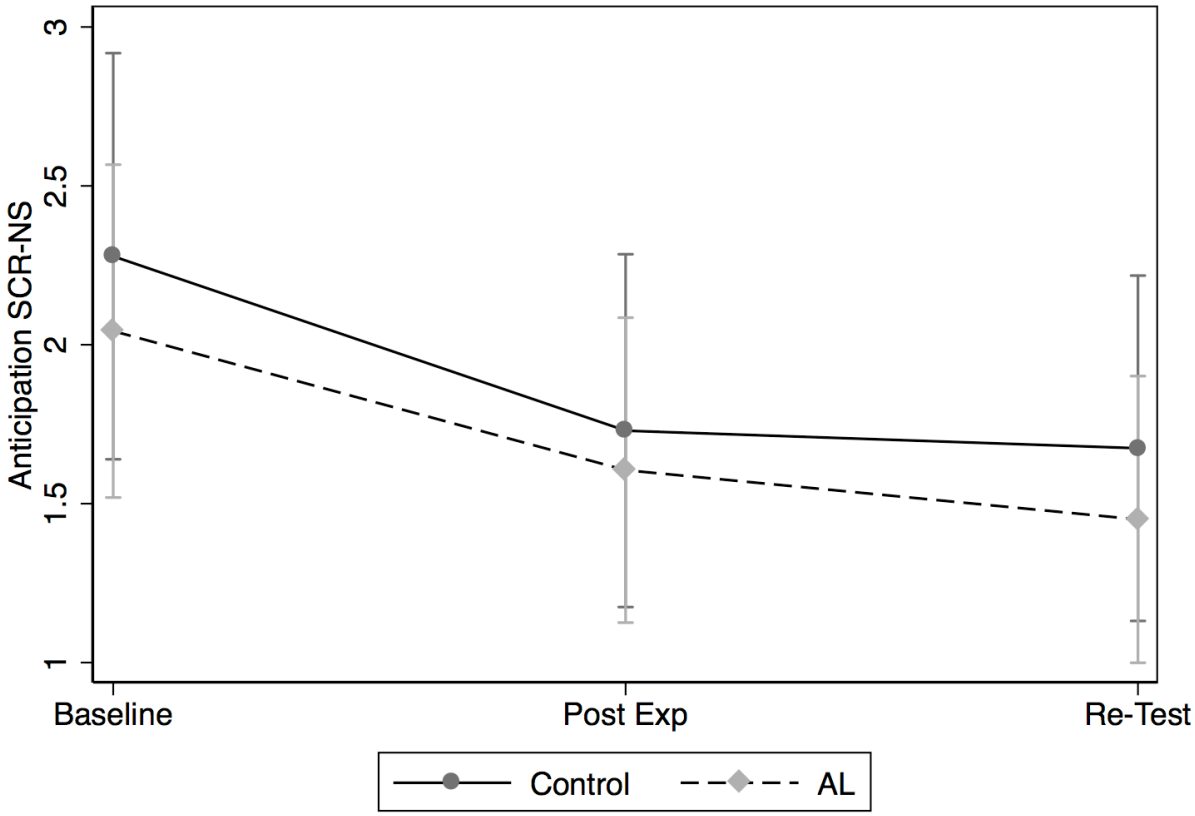


Figure 8. Non-Specific Skin Conductance Response During Recovery Over Time by Group

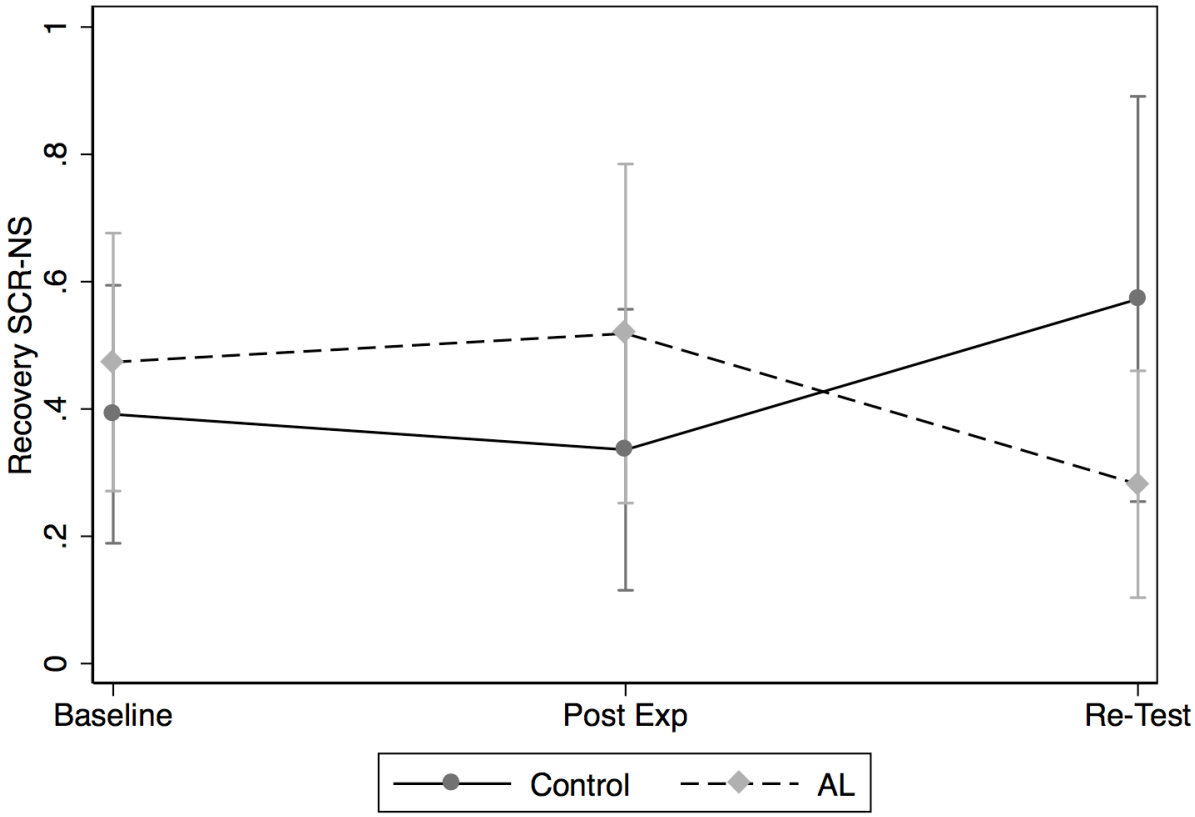


Figure 9. Personal Report of Public Speaking Anxiety Over Time by Group

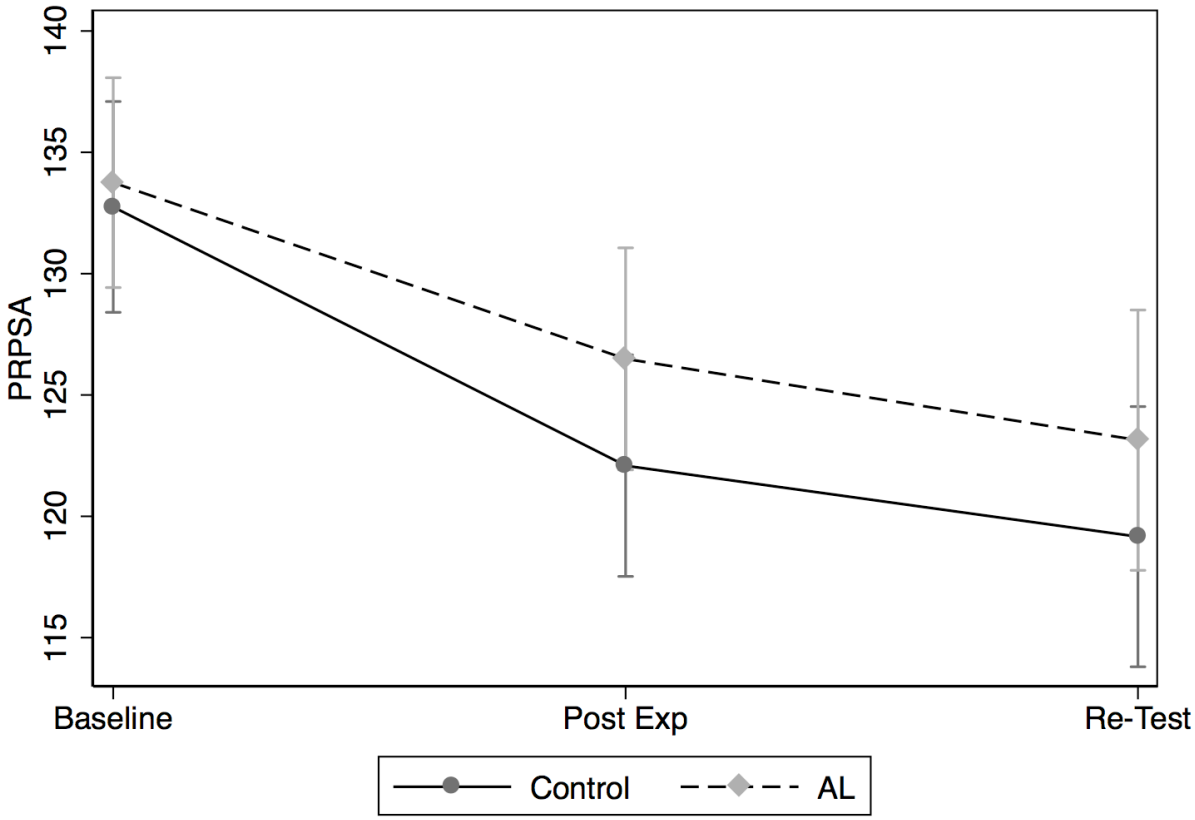


Figure 10. Subjective Units of Distress Over Time by Group

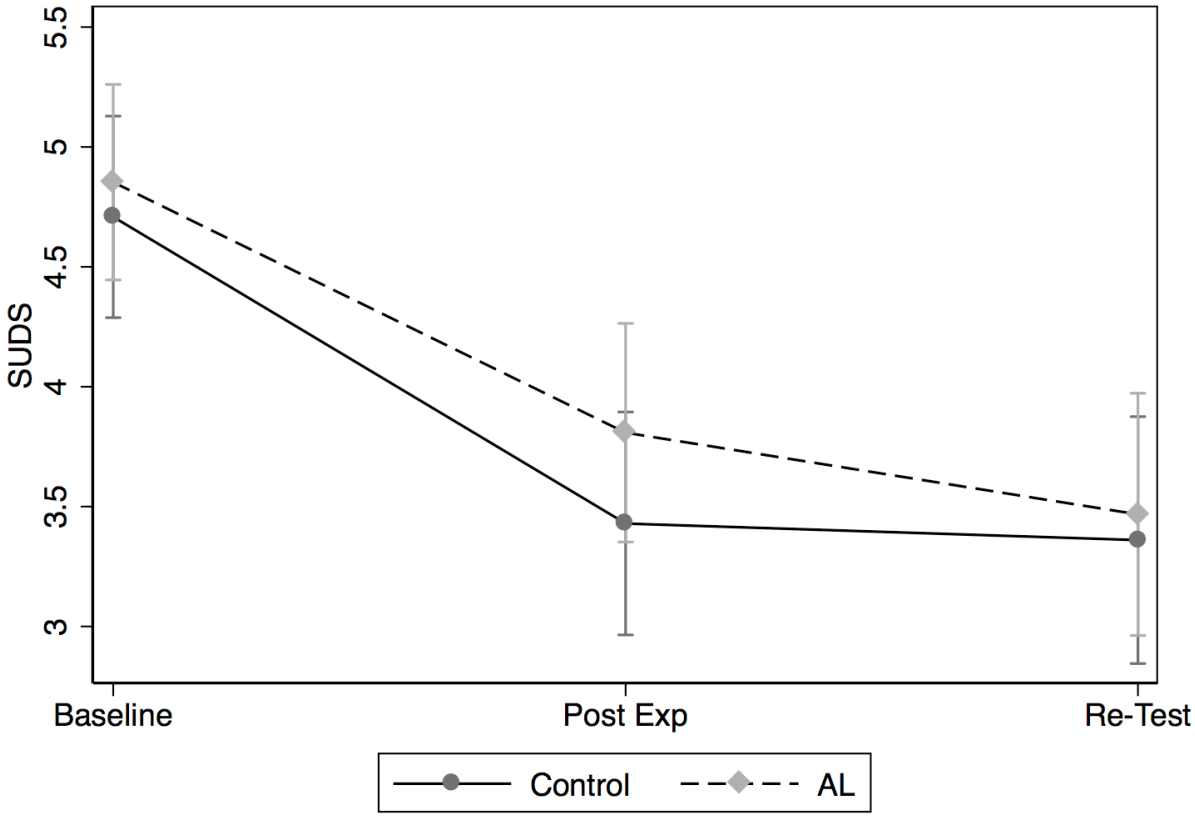
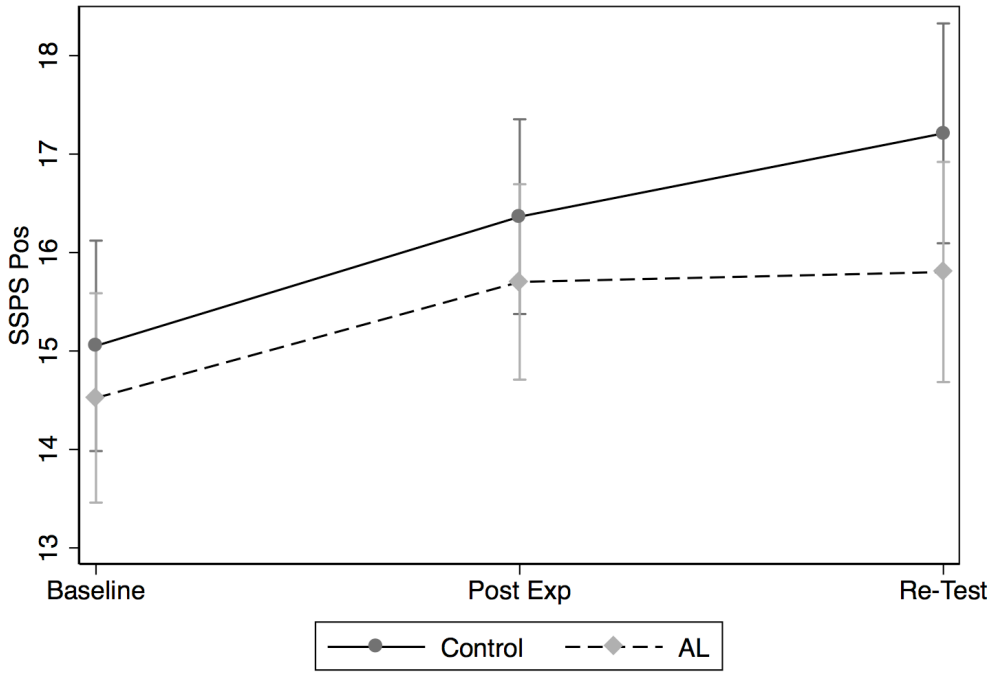


Figure 11. Self Statements During Public Speaking Over Time by Group: (A) Positive Self Statements; (B) Negative Self Statements

(A)



(B)

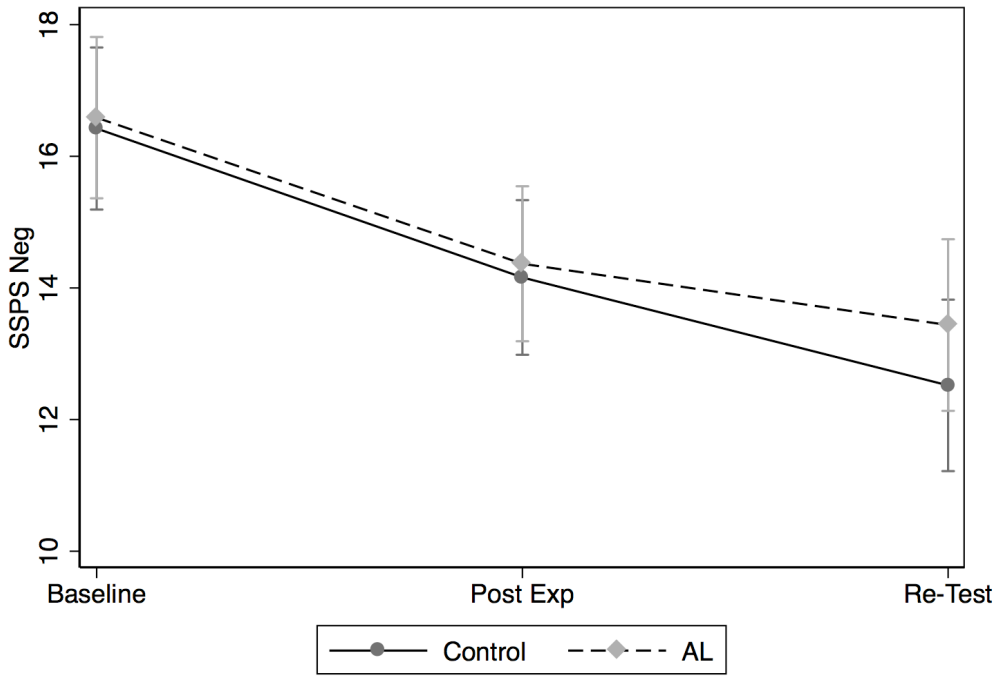


Figure 12. Performance Rating Form Over Time by Group

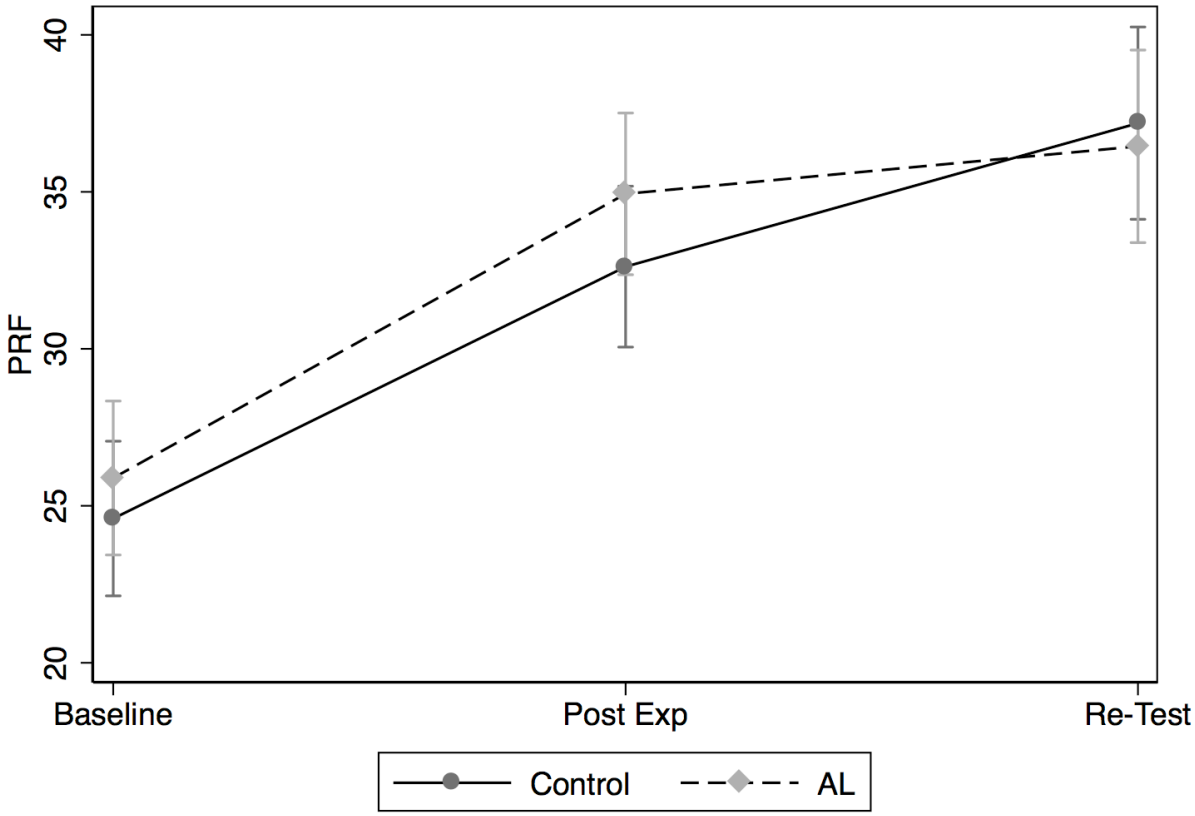


Figure 13. Optional Speech Over Time by Group

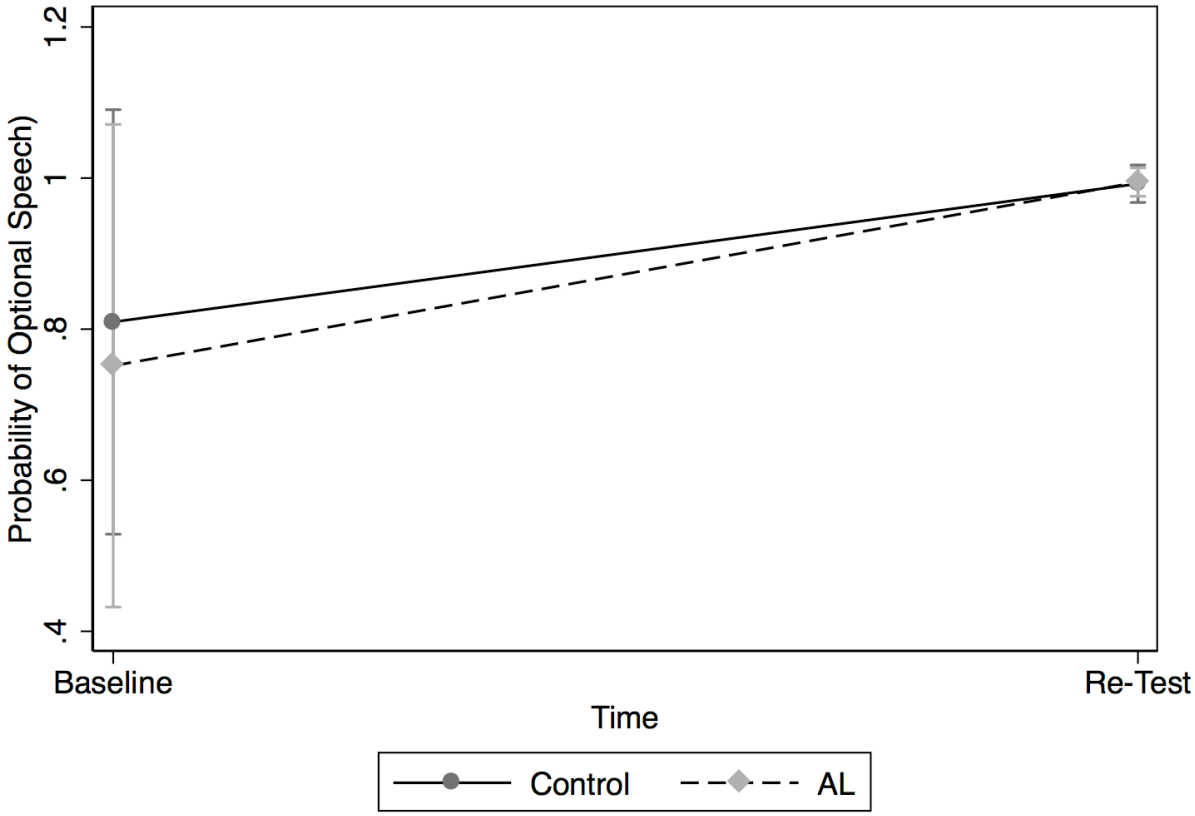


Figure 14. Heart Rate During Anticipation over Time by Group for High Labelers Only

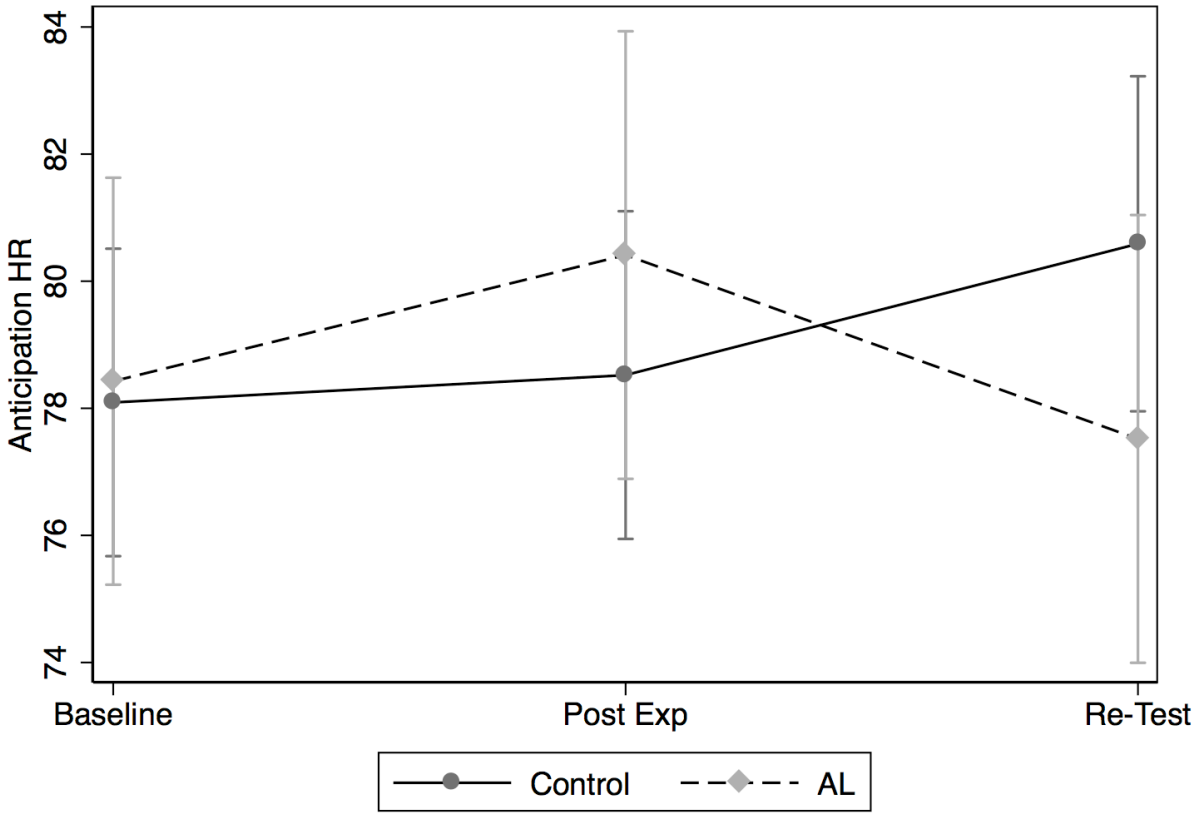


Figure 15. Heart Rate During Recovery over Time by Group for High Labelers Only

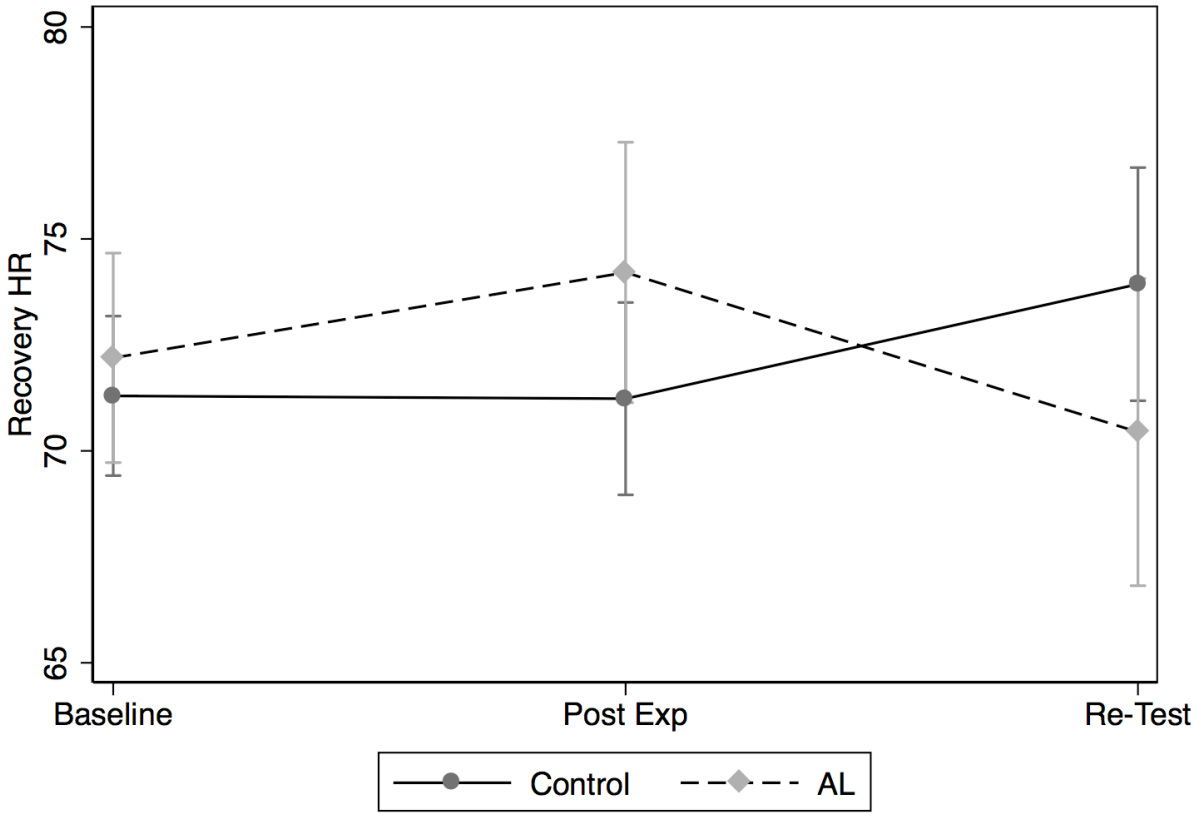


Figure 16. Non-Specific Skin Conductance Response during Anticipation over Time by Group for High Labelers Only

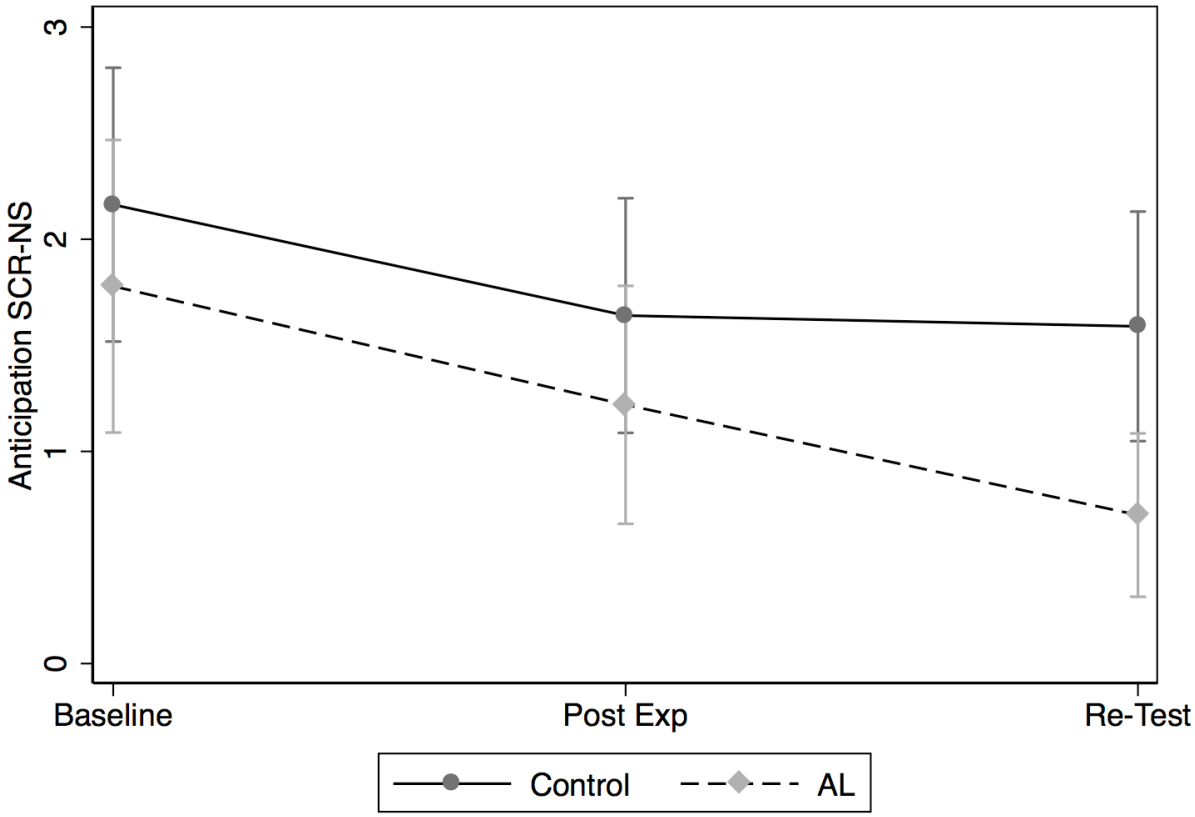


Figure 17. Non-Specific Skin Conductance Response During Recovery over Time by Group for High Labelers Only

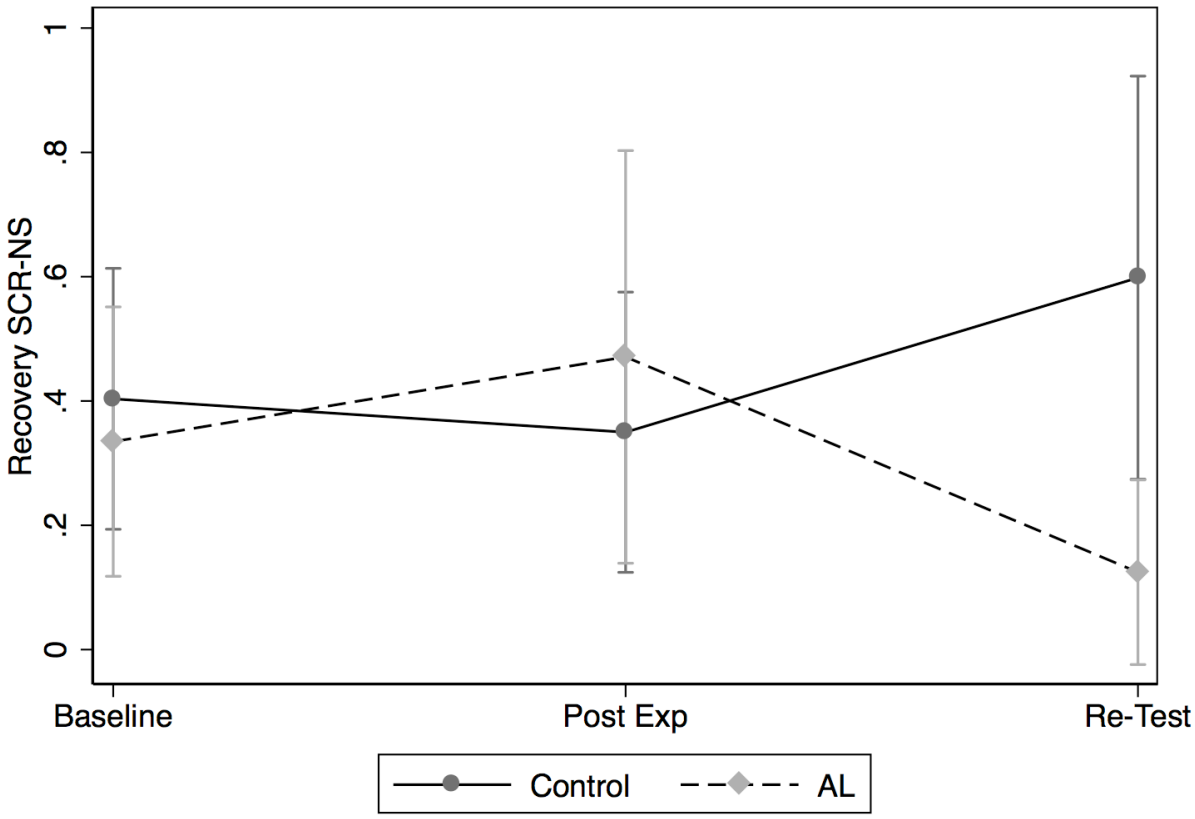


Figure 18. Personal Report of Public Speaking Anxiety Over Time by Group for High Labelers Only

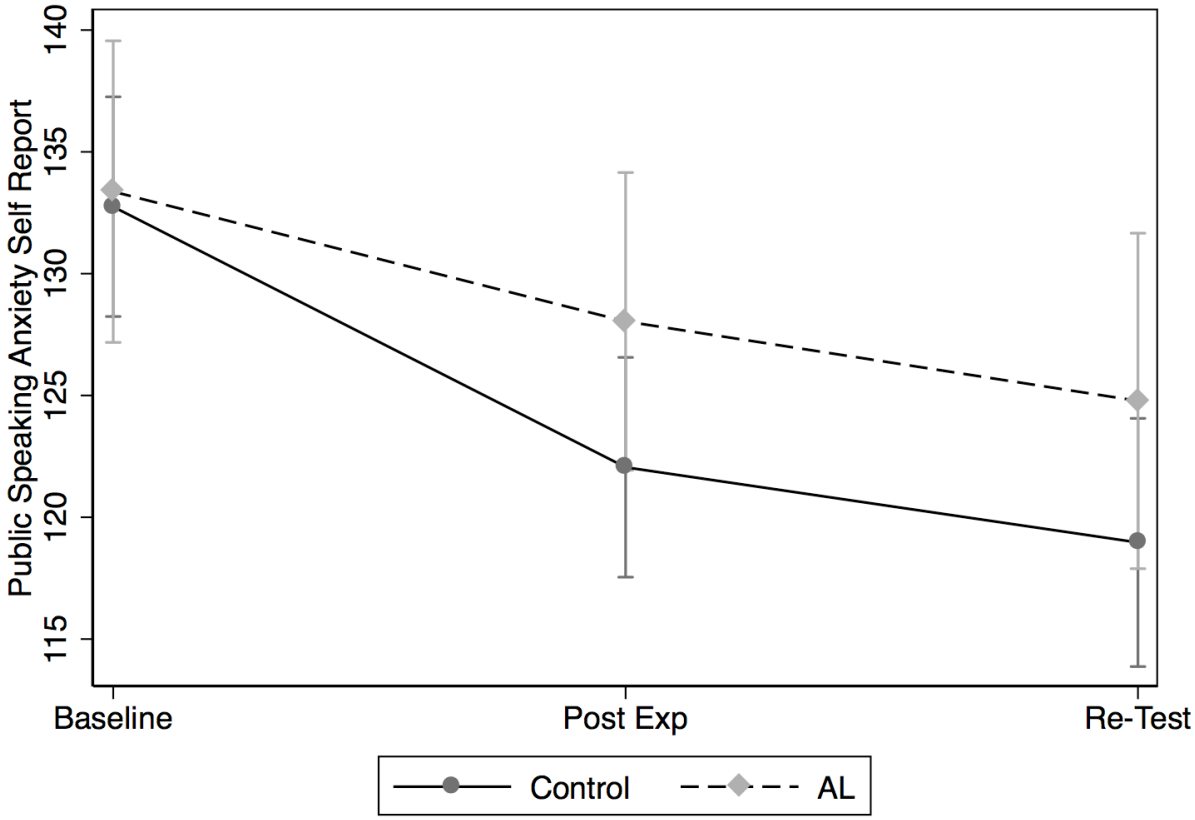


Figure 19. Subjective Units of Distress Over Time by Group for High Labelers Only

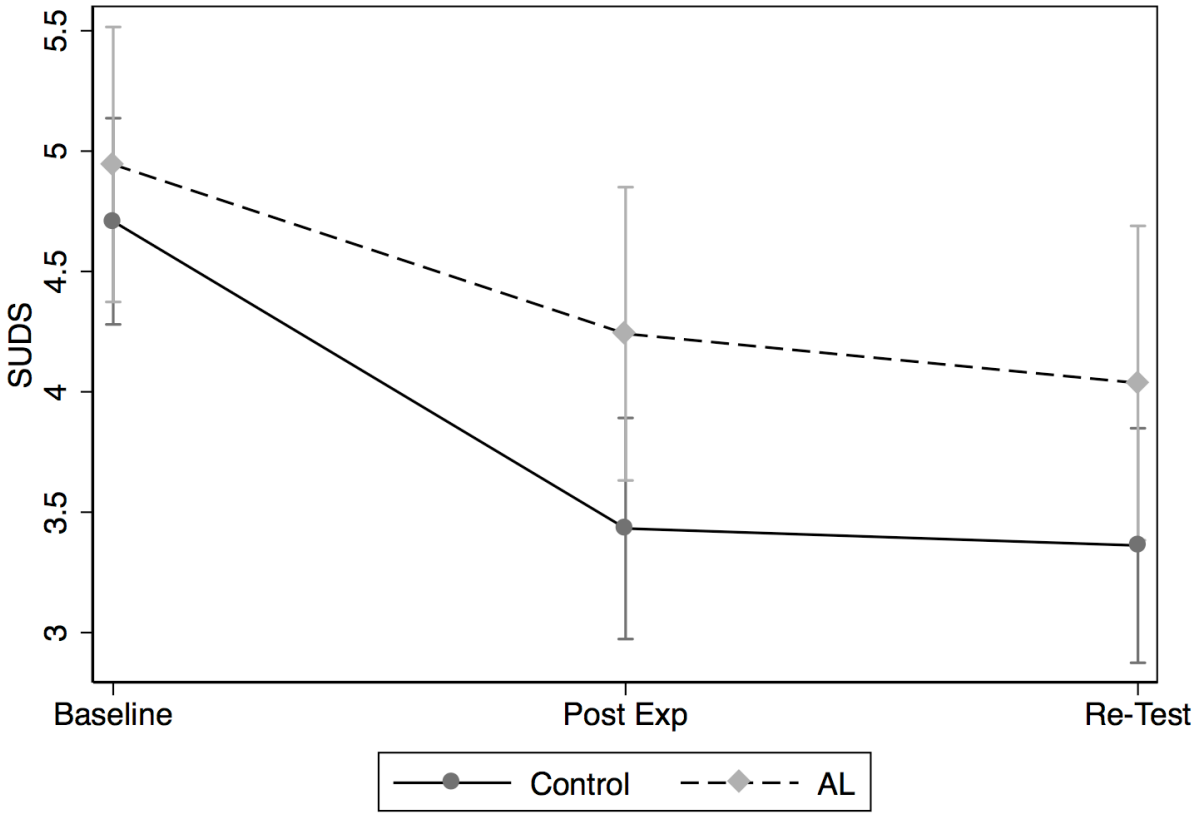


Figure 20. Association Between Number of Anxiety Words Chosen and Non-Specific Skin Conductance Response during Anticipation over Time

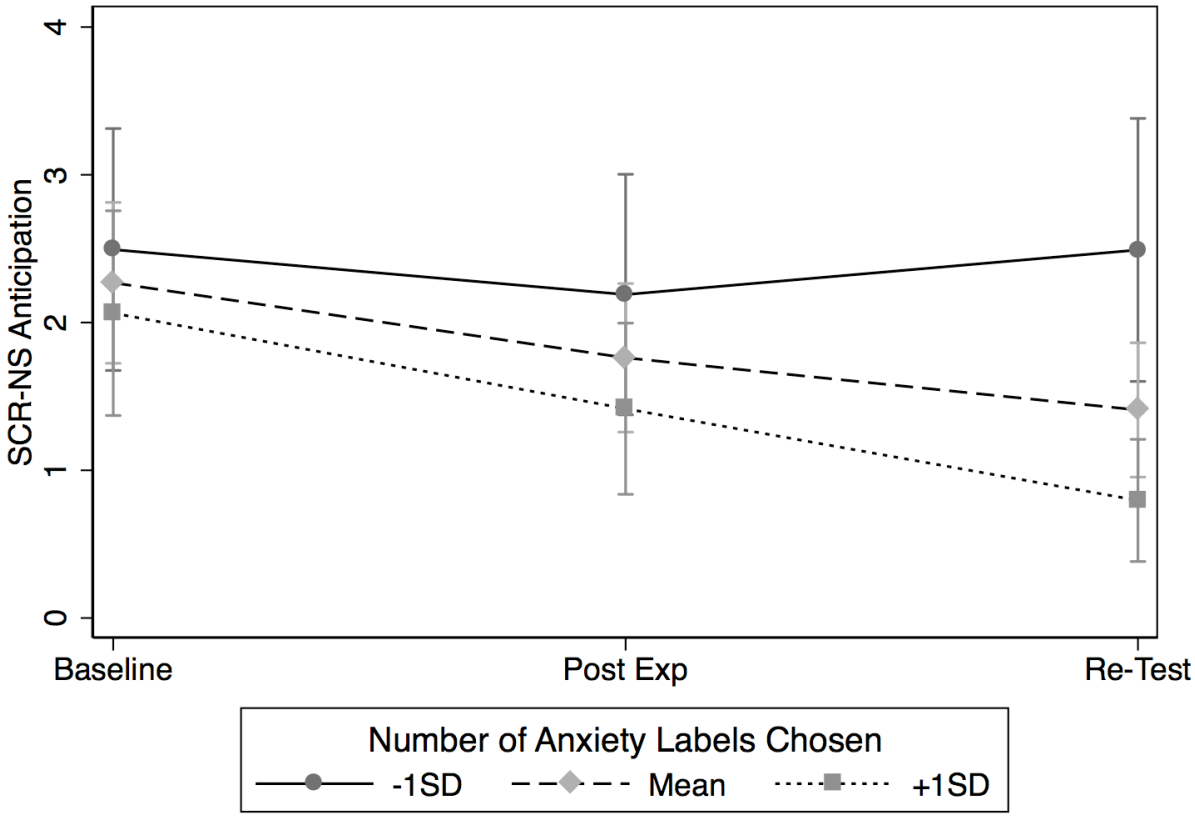


Figure 21. Association Between Number of Anxiety Words Chosen and Personal Report of Public Speaking Anxiety Over Time

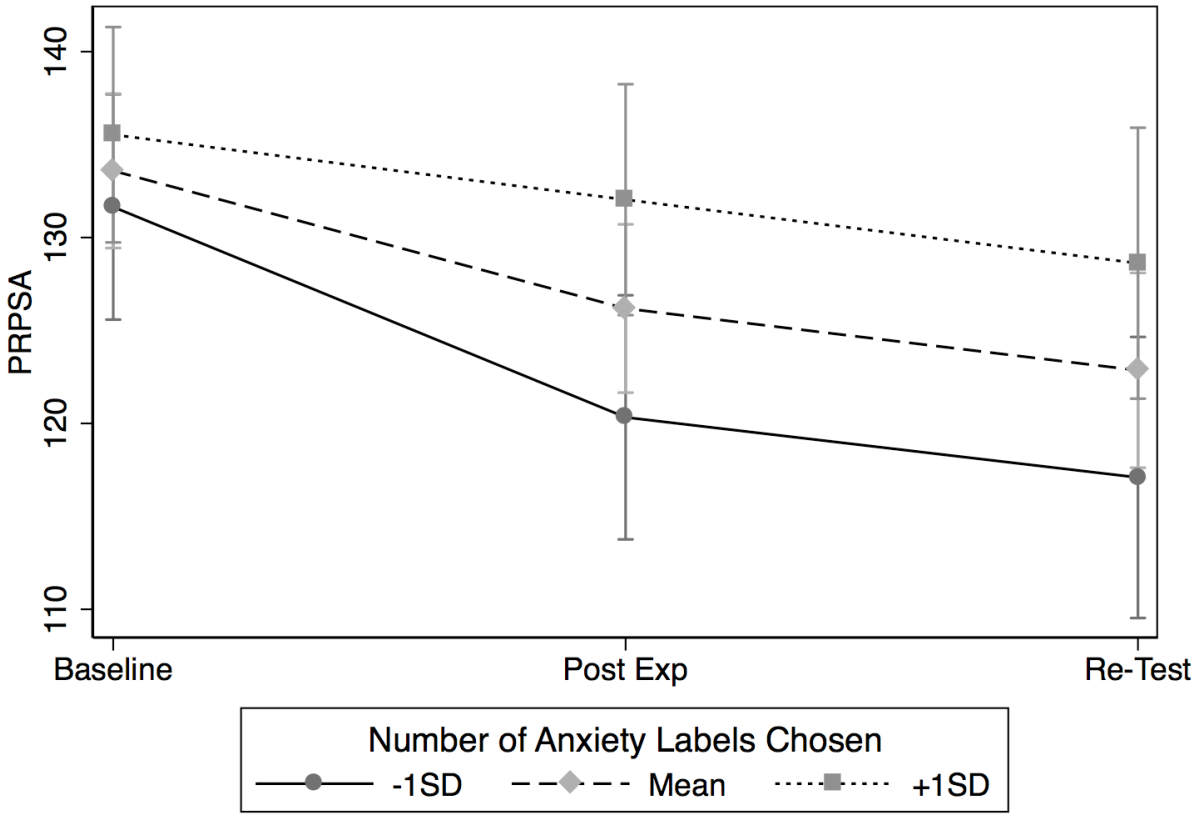


Figure 22. Association Between Number of Anxiety Words Chosen and Subjective Units of Distress Over Time

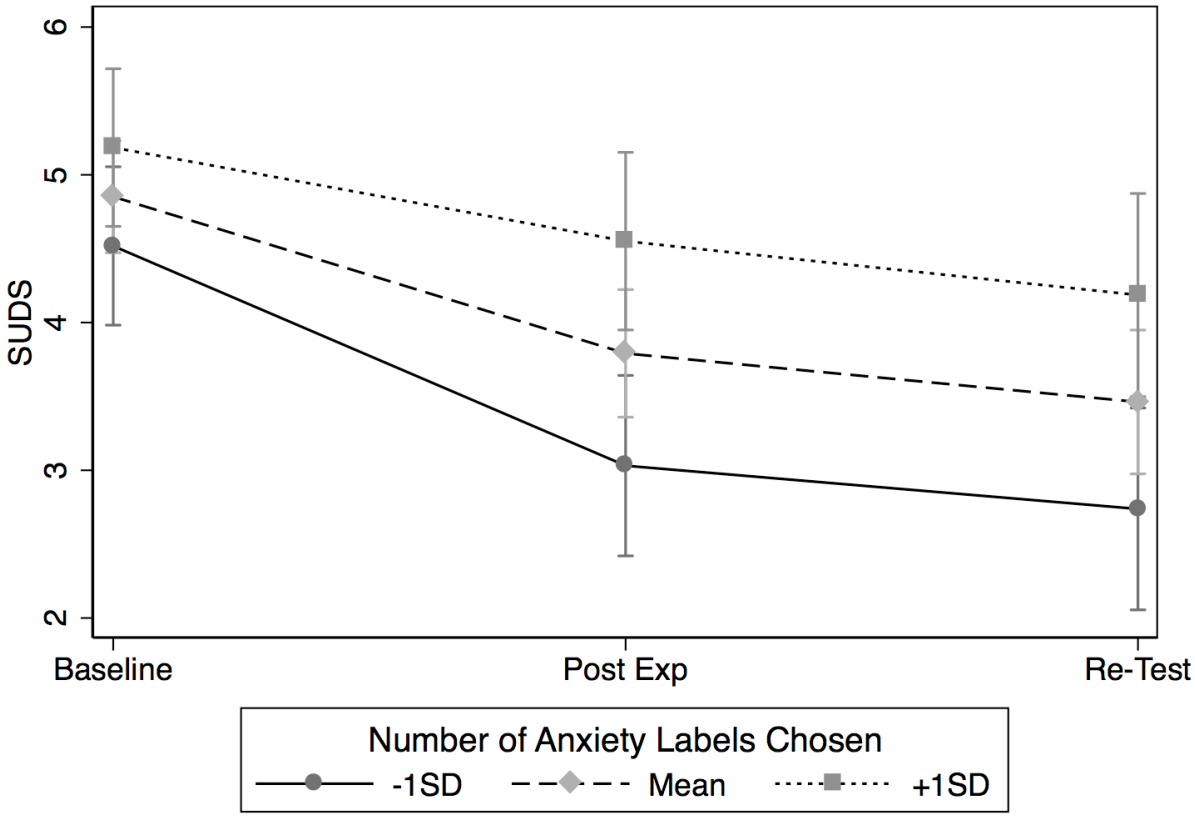


Figure 23. Association Between Number of Anxiety Words Chosen and Performance Rating Form Over Time

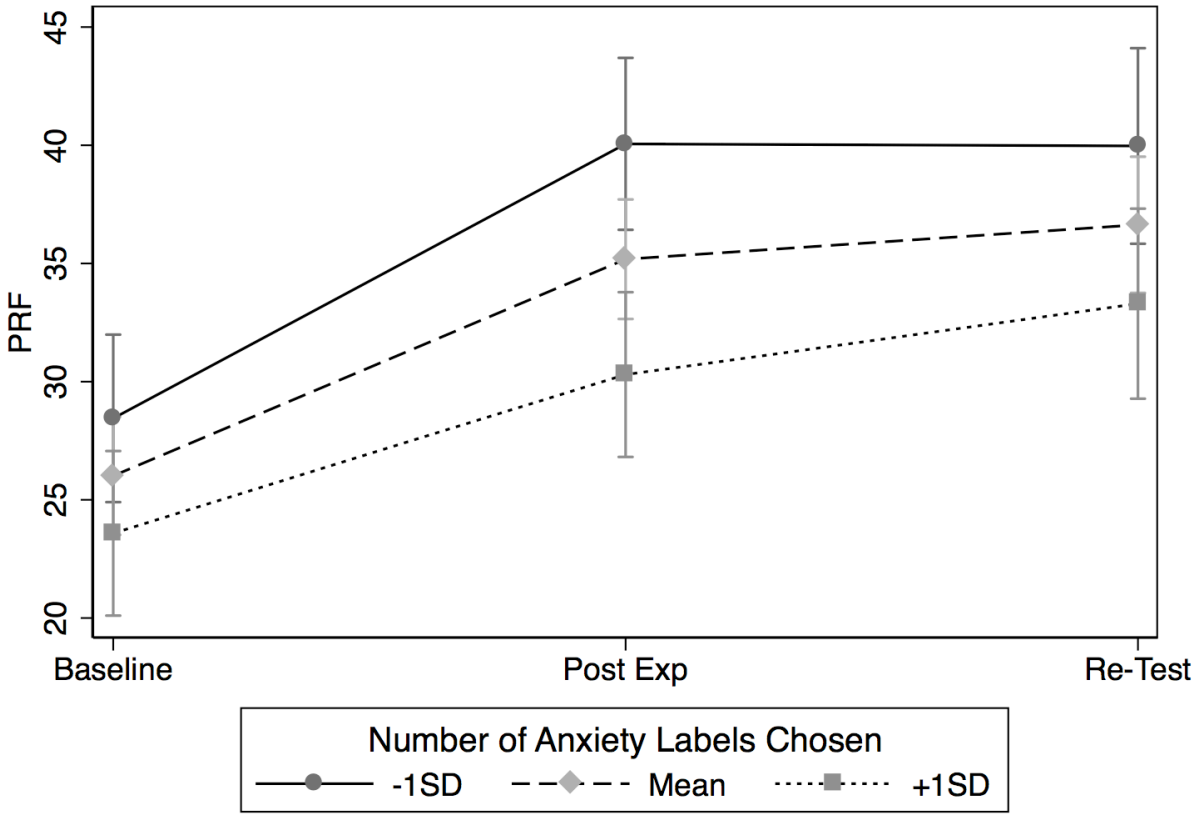


Figure 24. Moderation of Heart Rate during anticipation over Time by Incidental Emotion Regulation and Group

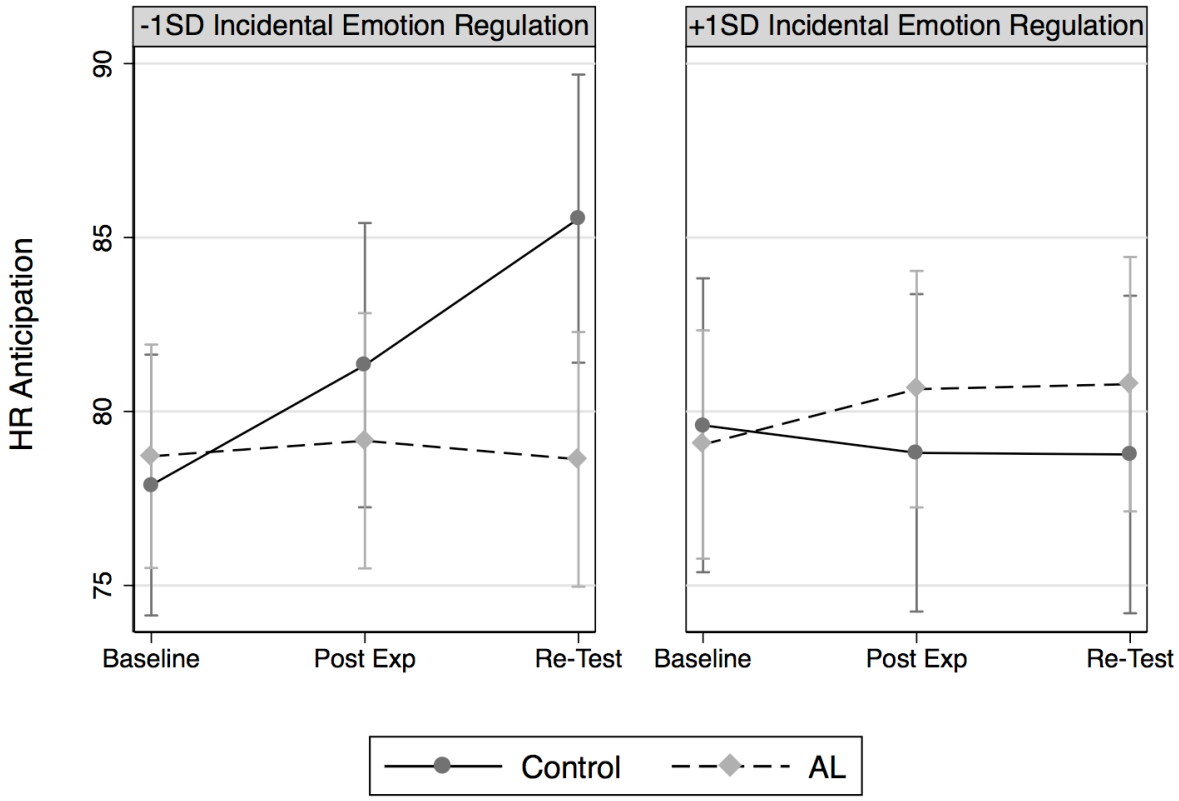


Figure 25. Moderation of Heart Rate during recovery over Time by Incidental Emotion Regulation and Group

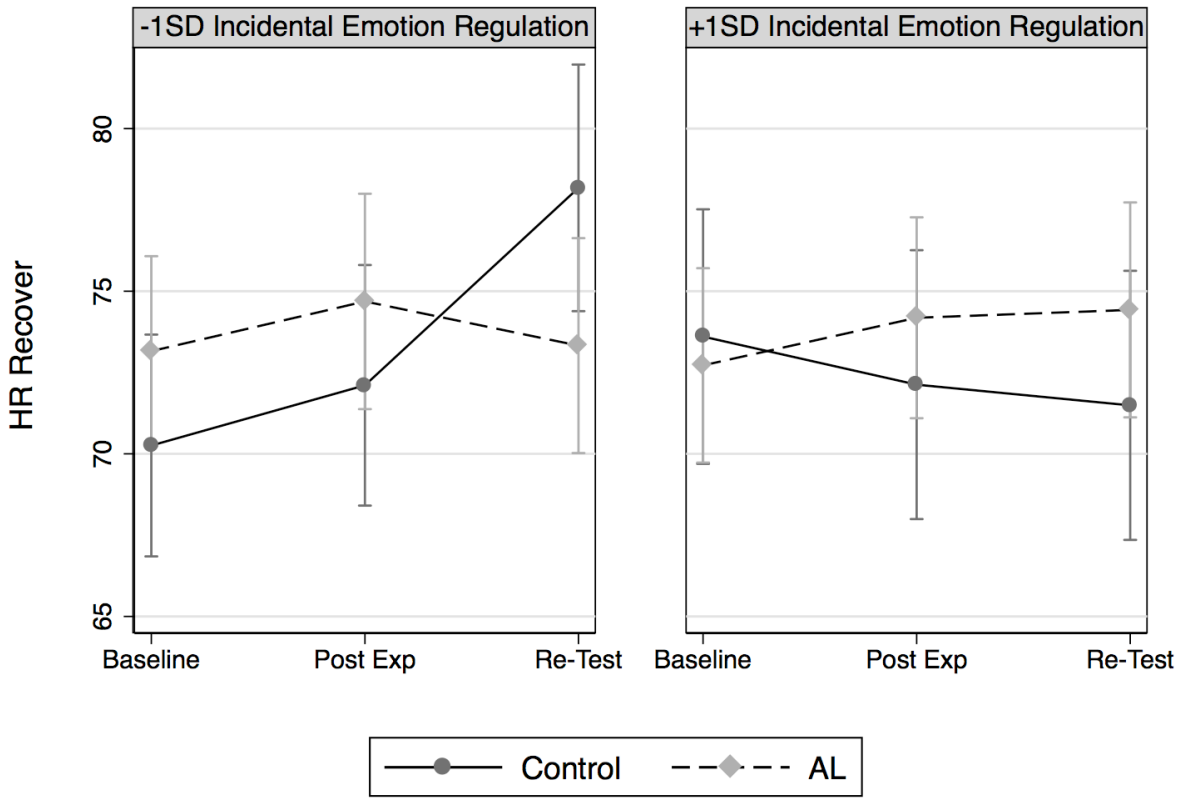


Figure 26. Prediction of Non-Specific Skin Conductance Response during anticipation over Time by Incidental Emotion Regulation

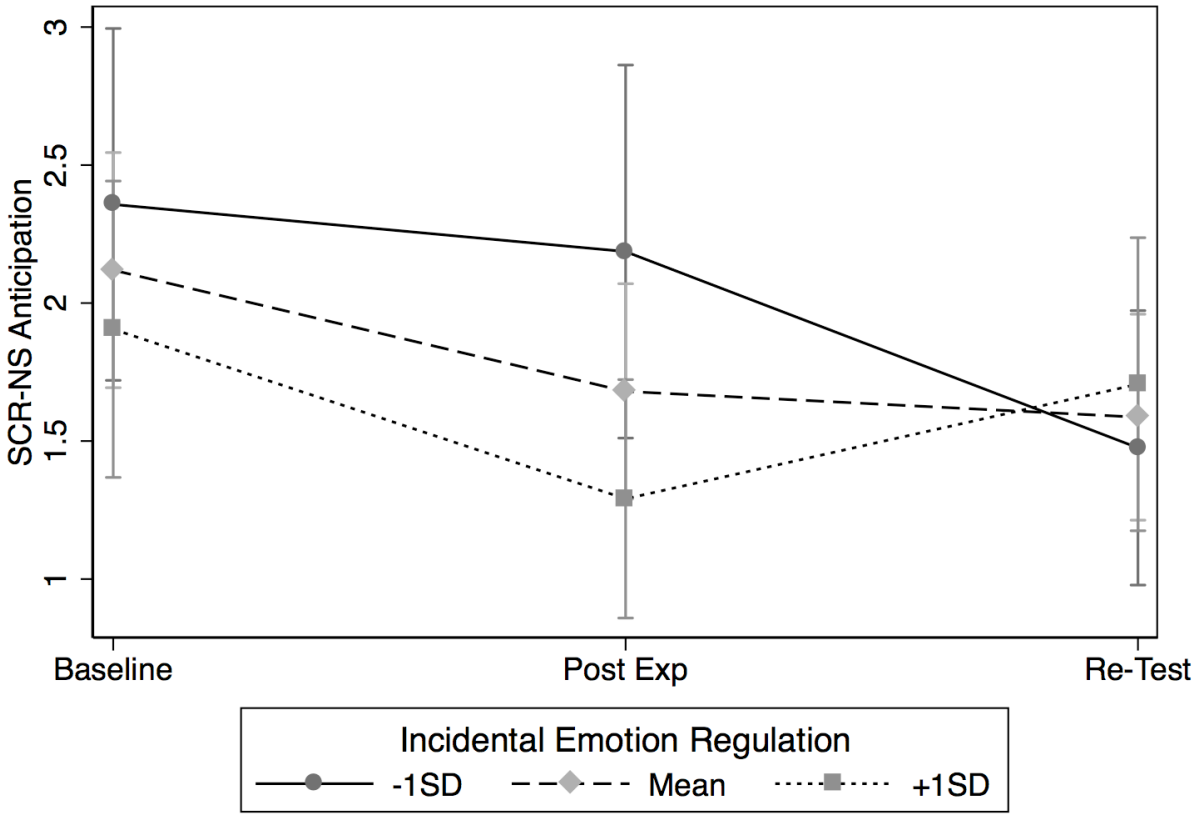


Figure 27. Moderation of Non-Specific Skin Conductance Response During Recovery Over Time by Incidental Emotion Regulation and Group

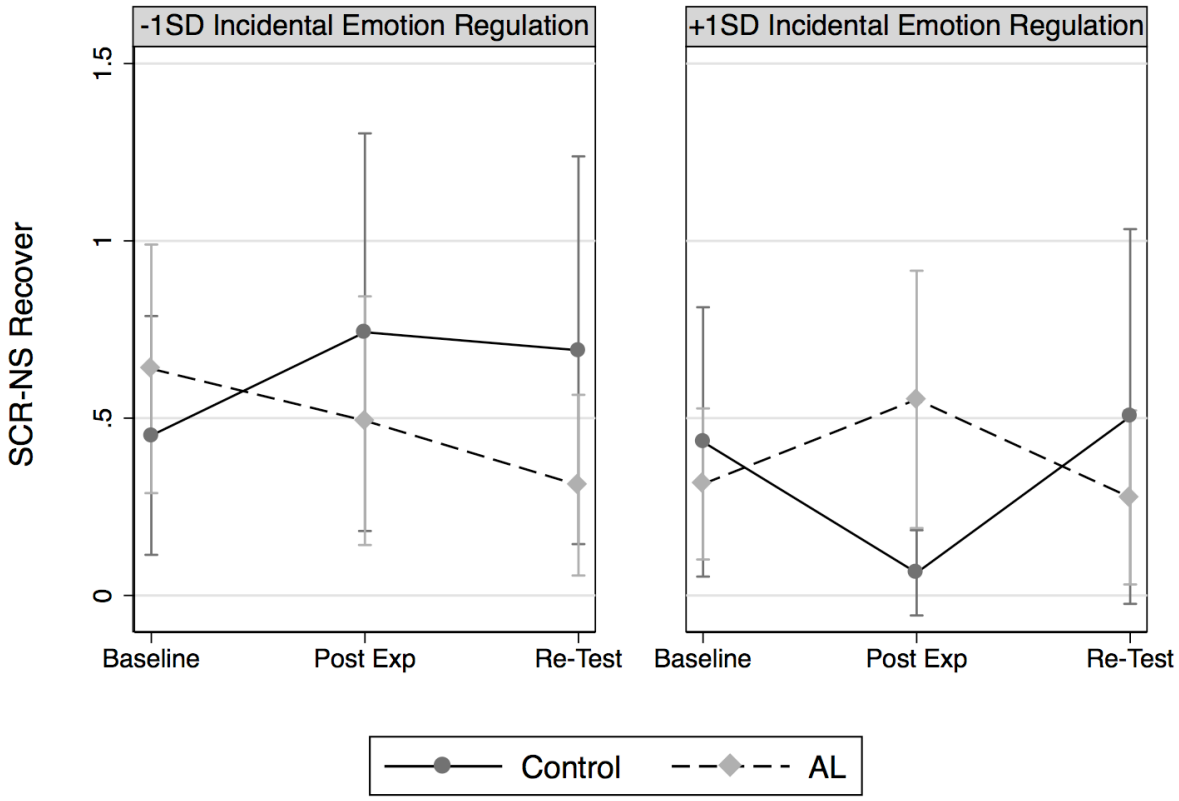


Figure 28. Moderation of Subjective Units of Distress Over Time by Incidental Emotion Regulation and Group

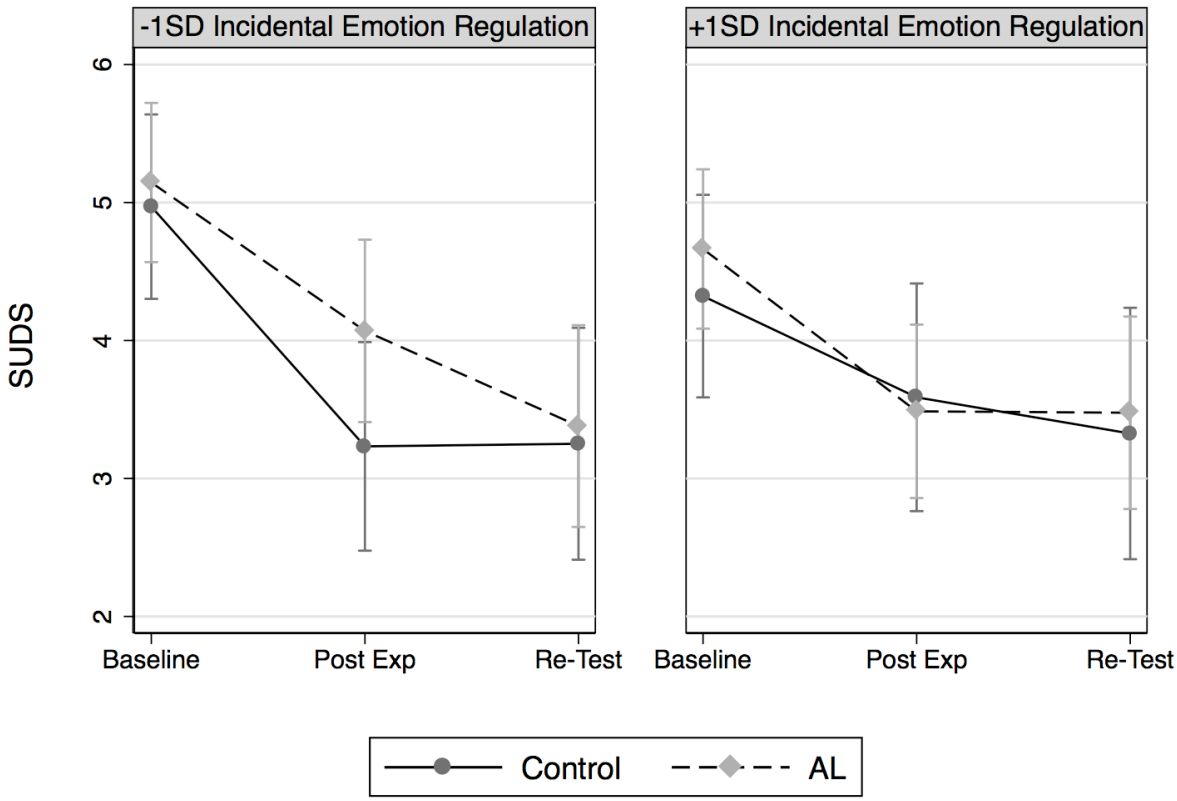


Figure 29. Prediction of Subjective Units of Distress Over Time by Incidental Emotion Regulation

Figure 30. Moderation of Self Statements During Public Speaking Positive Over Time by Incidental Emotion Regulation and Group

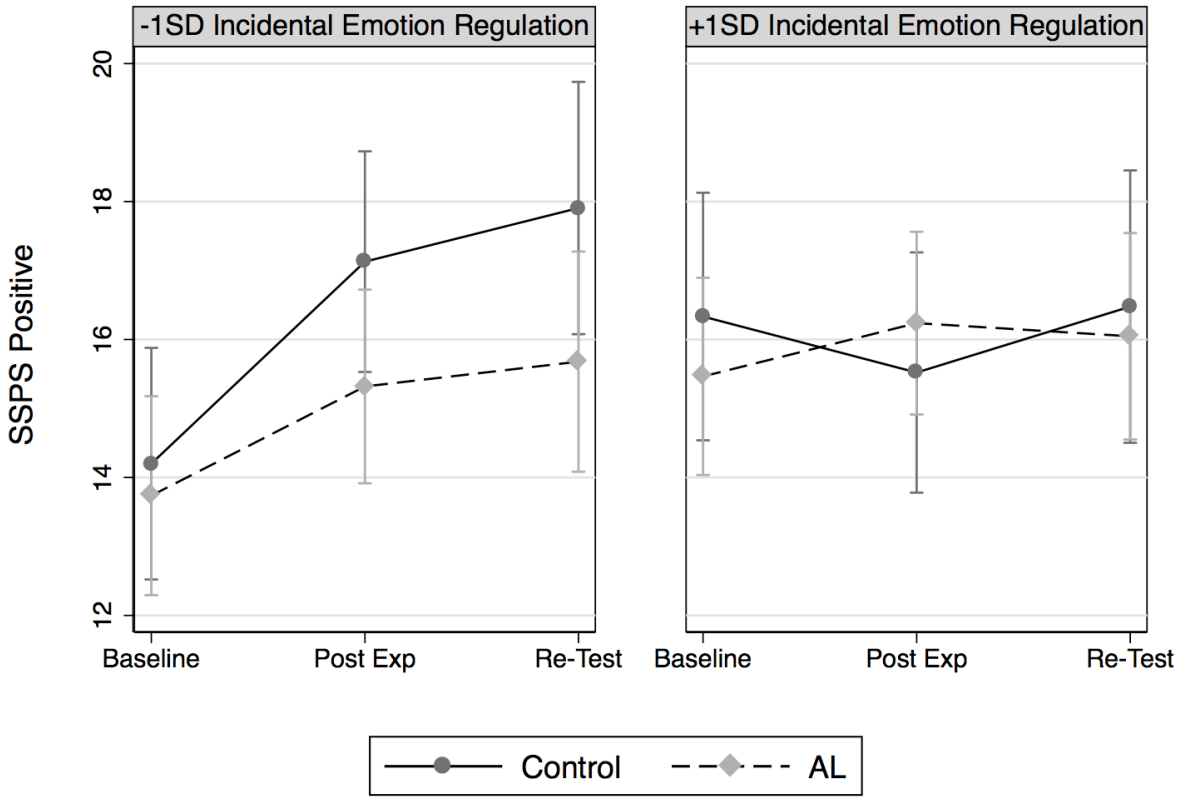


Figure 31. Prediction of Self Statements During Public Speaking Positive Over Time by Incidental Emotion Regulation

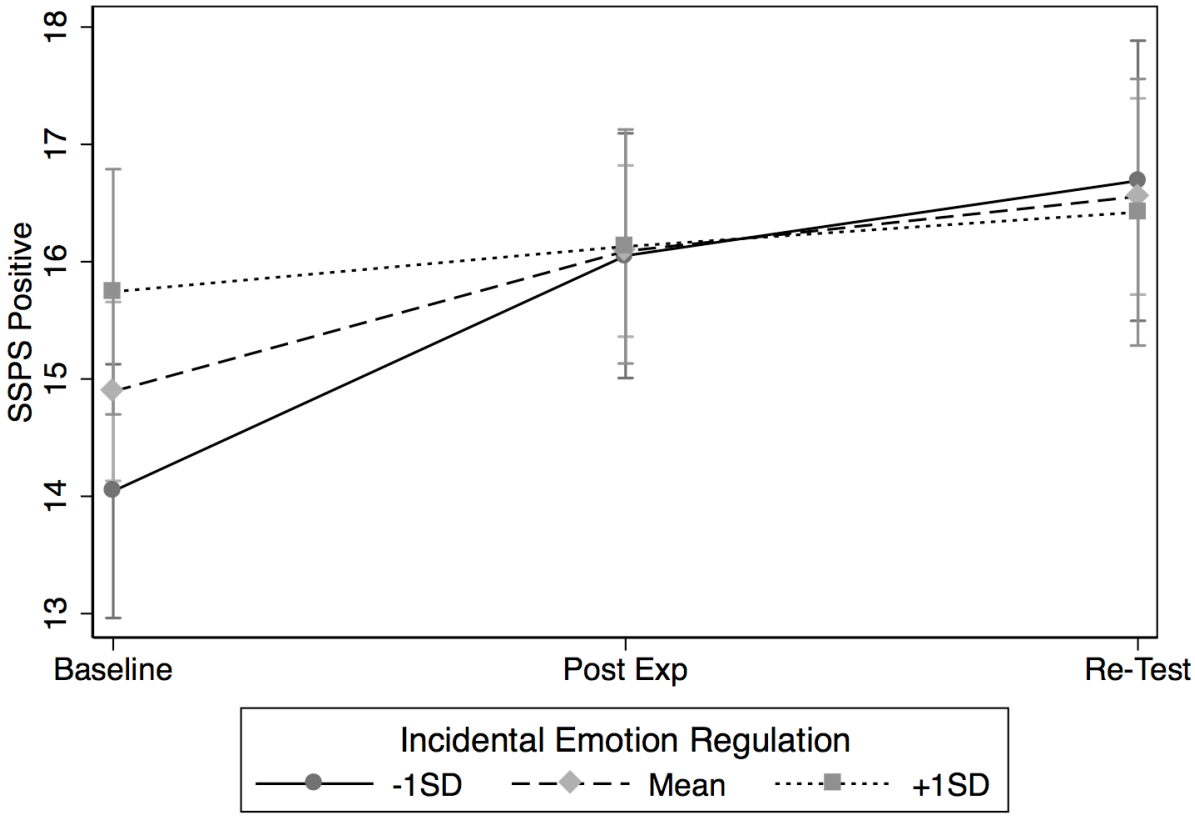


Figure 32. Moderation of Self Statements During Public Speaking Negative over Time by Incidental Emotion Regulation and Group

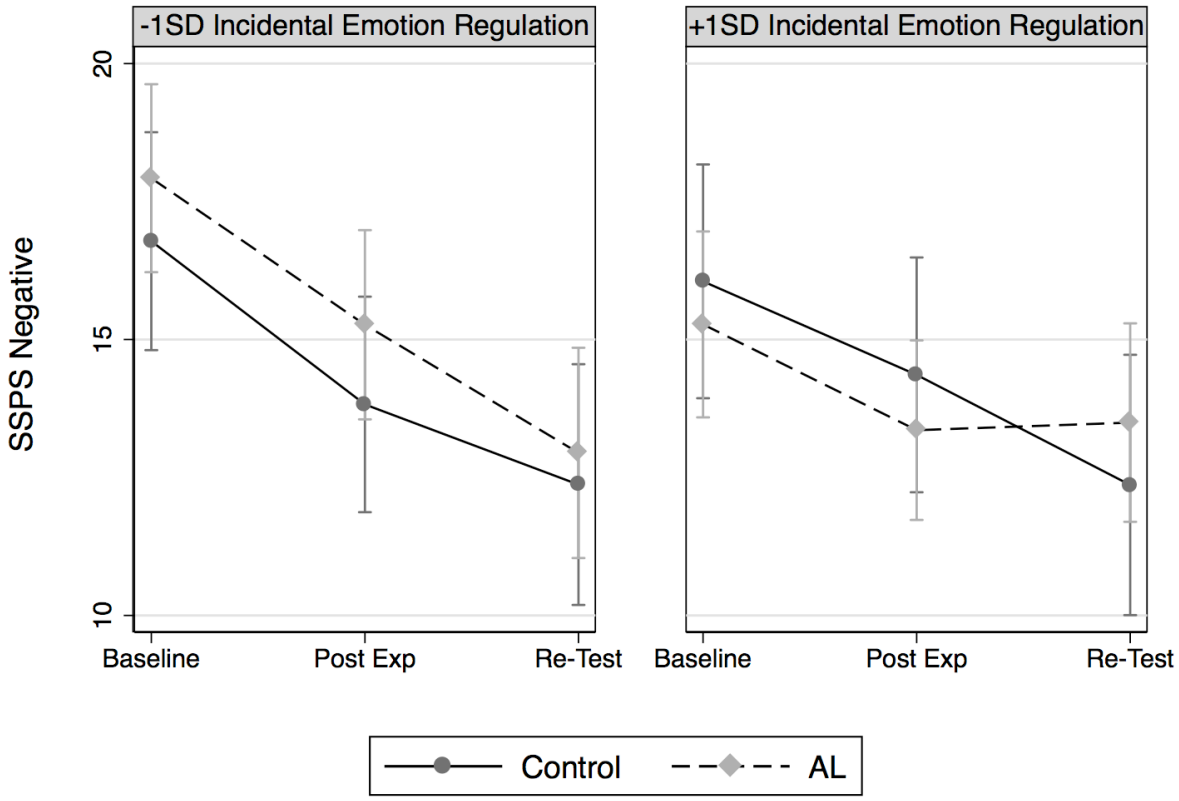


Figure 33. Prediction of Self Statements During Public Speaking Negative Over Time by Incidental Emotion Regulation

Appendix A: Questionnaires

PUBLIC SPEAKING SCREENER

Please indicate the degree to which you agree with each of the following items. Simply circle your response to each item, using a 0 to 8 point scale.

1. How anxious would you feel giving a formal speech before a live audience?

None				Moderate				Extremely
0	1	2	3	4	5	6	7	8

2. How likely would you be to avoid taking a class that required an oral presentation?

Never				Sometimes				Always
0	1	2	3	4	5	6	7	8

Personal Report of Public Speaking Anxiety

Directions: Below are 34 statements that people sometimes make about themselves. Please indicate whether or not you believe each statement applies to you by marking whether you:

Strongly Disagree = 1; Disagree = 2; Neutral = 3; Agree = 4; Strongly Agree = 5.

_____1. While preparing for giving a speech, I feel tense and nervous.

_____2. I feel tense when I see the words "speech" and "public speech" on a course outline when studying.

_____3. My thoughts become confused and jumbled when I am giving a speech.

_____4. Right after giving a speech I feel that I have had a pleasant experience.

_____5. I get anxious when I think about a speech coming up.

_____6. I have no fear of giving a speech.

_____7. Although I am nervous just before starting a speech, I soon settle down after starting and feel calm and comfortable.

_____8. I look forward to giving a speech.

_____9. When the instructor announces a speaking assignment in class, I can feel myself getting tense.

_____10. My hands tremble when I am giving a speech.

_____11. I feel relaxed while giving a speech.

_____12. I enjoy preparing for a speech.

_____13. I am in constant fear of forgetting what I prepared to say.

_____14. I get anxious if someone asks me something about my topic that I don't know.

_____15. I face the prospect of giving a speech with confidence.

_____16. I feel that I am in complete possession of myself while giving a speech.

_____17. My mind is clear when giving a speech.

_____18. I do not dread giving a speech.

_____19. I perspire just before starting a speech.

_____20. My heart beats very fast just as I start a speech.

_____21. I experience considerable anxiety while sitting in the room just before my speech starts.

_____22. Certain parts of my body feel very tense and rigid while giving a speech.

_____23. Realizing that only a little time remains in a speech makes me very tense and anxious.

_____24. While giving a speech, I know I can control my feelings of tension and stress.

_____25. I breathe faster just before starting a speech.

_____26. I feel comfortable and relaxed in the hour or so just before giving a speech.

_____27. I do poorer on speeches because I am anxious.

_____28. I feel anxious when the teacher announces the date of a speaking assignment.

_____29. When I make a mistake while giving a speech, I find it hard to concentrate on the parts that follow.

_____30. During an important speech I experience a feeling of helplessness building up inside me.

_____31. I have trouble falling asleep the night before a speech.

_____32. My heart beats very fast while I present a speech.

_____33. I feel anxious while waiting to give my speech.

_____34. While giving a speech, I get so nervous I forget facts I really know.

Patient Health Questionnaire

Over the last 2 weeks, how often have you been bothered by any of the following problems?

1	2	3	4
Not at all	Several days	More than half the days	Nearly Every day

_____a. Little interest or pleasure in doing things

_____b. Feeling down, depressed, or hopeless

_____c. Trouble falling or staying asleep, or sleeping too much

_____d. Feeling tired or having little energy

_____e. Poor appetite or overeating

_____f. Feeling bad about yourself, or that you are a failure, or have let yourself or your family down

_____g. Trouble concentrating on things, such as reading the newspaper or watching television

_____h. Moving or speaking so slowly that other people could have noticed? Or the opposite - being so fidgety or restless that you have been moving around a lot more than usual

_____i. Thoughts that you would be better off dead or of hurting yourself in some way

Mini-SPIN

1. Fear of embarrassment causes me to avoid doing things or speaking to people:

0	1	2	3	4
Not at all	**A little bit**	**Somewhat**	**Very much**	**Extremely**

2. I avoid activities in which I am the center of attention

0	1	2	3	4
Not at all	**A little bit**	**Somewhat**	**Very much**	**Extremely**

3. Being embarrassed or looking stupid are among my worse fears

0	1	2	3	4
Not at all	**A little bit**	**Somewhat**	**Very much**	**Extremely**

Self-Statements During Public Speaking Questionnaire

It is obvious that people think a variety of things when they are giving a speech. Below is a list of things that you may or may not have thought to yourself during your speech. Now read each item below and rate the degree of your agreement on a scale between 0 (if you would not agree at all with the statement) and 5 (if you would agree extremely with the statement).

0 ----------1----------2----------3----------4----------5
Do not agree at all Agree extremely

1. _____What do I have to lose it's worth a try
2. _____I'm a loser
3. _____This is an awkward situation but I can handle it
4. _____A failure in this situation would be more proof of my incapacity
5. _____Even if things don't go well, it's no catastrophe
6. _____I can handle everything
7. _____What I say will probably sound stupid
8. _____I'll probably "bomb out" anyway
9. _____Instead of worrying I could concentrate on what I want to say
10. _____I feel awkward and dumb; they're bound to notice

Exposure Questionnaire

How logical does this type of exposure seem to you for helping people reduce fear and anxiety?

0 ----- 1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7 ----- 8
not at all logical — very logical

How confident are you that this exposure will be successful in eliminating your fear of spiders?

0 ----- 1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7 ----- 8
not at all confident — very confident

How confident would you be in recommending this exposure program to a friend who has a strong fear of spiders?

0 ----- 1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7 ----- 8
not at all confident — very confident

How successful do you feel this exposure would be in decreasing other problems involving fear and anxiety, like headaches, insomnia, etc.?

0 ----- 1 ----- 2 ----- 3 ----- 4 ----- 5 ----- 6 ----- 7 ----- 8
not at all helpful — very helpful

PERFORMANCE RATING FORM - SELF

We would like you to rate yourself on the features listed below. For each feature, please circle the appropriate number to indicate how you felt you actually performed. Please answer as honestly as you can - your evaluation will remain completely confidential.

Your name:_____ Date:_____

	not at all	slightly	moder-ately	much	very much
Content was understandable	0	1	2	3	4
Kept eye contact with audience	0	1	2	3	4
Stuttered	0	1	2	3	4
Had long pauses (more than 5 seconds)	0	1	2	3	4
Fidgeted	0	1	2	3	4
"Um"ed and "Ah"ed	0	1	2	3	4
Had a clear voice	0	1	2	3	4
Seemed to tremble or shake	0	1	2	3	4
Sweated	0	1	2	3	4
Blushed	0	1	2	3	4
Face twitched	0	1	2	3	4
Voice quivered	0	1	2	3	4
Appeared confident	0	1	2	3	4
Appeared nervous	0	1	2	3	4
Kept audience interested	0	1	2	3	4
Generally spoke well	0	1	2	3	4
Made a good impression	0	1	2	3	4

Appendix B. Options for affect labeling during exposure

Emotions:

Anxiety	**Other Negative**	
Afraid	Frustrated	Blue
Nervous	Angry	Sad
Anxious	Annoyed	Downhearted
Jittery	Furious	Lonely
Scared	Irritable	Discouraged
Panicked	Resentful	Depressed
Frightened	Mad	Hopeless
Worried	Exasperated	Worthless
Terrified	Hostile	Gloomy
Afraid	Frustrated	Blue

Feared outcomes:

Audience Action	**Audience Judgment**	**Audience Notice Anxiety**
Laugh at me	Be disinterested	Notice I'm nervous
Make fun of me	Think I'm stupid	Notice I'm anxious
Be hostile	Dislike my speech	Notice I'm sweating
Be upset	Think I'm not cool	Notice my mistakes
Point and laugh	Find me boring	Notice I'm shaking
Lose interest in me	Think I'm unintelligent	Notice my errors
Make faces at me	Think I'm an idiot	Notice I'm uncomfortable
Yell at me	Think I'm weird	Notice I'm awkward
Be disappointed	Think I'm weak	Notice I'm blushing
Be angry	Judge me negatively	Notice I'm trembling

Appendix C. Speech topics for the three behavioral approach tests and two exposure sessions

BAT Speech Topics

Health Care	President Obama	Global Warming
Gay Marriage	Affirmative Action	Abortion

Exp1 Speech Topics	**Exp2 Speech Topics**
Favorite Movie	Favorite Food
Favorite Sport	Los Angeles Weather
Career Goals	Mac vs. PC
Hobbies	Los Angeles Traffic
Favorite Animal	Obesity Epidemic
Academic Strengths	Favorite Fast Food
Favorite Music	Public Transportation
Dogs vs. Cats	Favorite Restaurant
Social Networking	Reality TV
Smart Phones	Favorite Book

Appendix D. Exposure instructions

During the next task, you will be giving 10 short speeches in front of a small audience. You will be directed through the task by a computer program. I will now describe what you will see on the computer screen during this task.

During this task, you will be asked to provide your anxiety level on a scale from 0 to 8. When you see this screen, please enter your anxiety level into the computer.

You may also be asked to identify the emotion that you are feeling. When you see this screen, please choose the emotion from those listed that best matches the emotion you are feeling at that moment. Please try to choose one of the emotions listed, however, if you really do not feel that any of the words matches your experience, you can choose "other."

You may also be asked to choose how you think the audience might respond to you while you are speaking. If you see this screen, please choose the outcome from the list that best matches your expectations of how the audience will respond. Please try to choose one of the responses listed, however, if you really do not feel that any of the options matches your experience, you can choose "other."

You may also be asked to match shapes. If you see this screen, please choose the shape at the bottom of the screen that matches the larger shape at the top of the screen. If the shape at the top of the screen does not match the shape at the bottom, you can choose "other."

You may also see the same shape-matching screen in blue. Again, simply choose the matching shape from the bottom of the screen or choose other.

You may or may not see each of the screens that we just reviewed. Some participants see some questions, and others see different questions. I do not know which ones you will see. Do you have any questions?

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