The Impact of Brief Clinical Interventions on Cardiovascular Reactions to Acute Stress

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Stress is a major public health concern due to its’ harmful effects on physical and mental health. An important goal for clinical health practitioners is to help patients reduce the psychological and physiological burden of stress. The present study sought to examine the impact brief clinical interventions have on cardiovascular reactions to acute stress. Additionally, we explored how depressive and anxiety symptomatology influence cardiovascular reactivity and recovery, and their moderating effects on treatment response to interventions. To address these aims, subjects were randomized into one of three conditions (Acceptance and Commitment Therapy, Autogenic Training, Attention-only Control group) prior to undergoing an acute psychosocial stress paradigm, the Trier Social Stress Test (TSST). Psychosocial measures were given at baseline, and heart rate and blood pressure measurements were obtained at various time points throughout the protocol. Our results indicated a time by group interaction for heart rate (HR) and diastolic blood pressure (DBP), demonstrating that brief interventions differentially affected heart rate and diastolic blood pressure over time. Within the Autogenic Training group results showed that subjects with higher levels of depressive symptomatology had lower systolic blood pressure reactivity compared to those with lower levels of depressive symptoms. Additionally, within the Autogenic Training group, greater recovery in both
systolic and diastolic blood pressure was found in subjects with higher levels of social anxiety. Our results offer important implications for clinical assessment and intervention, particularly within the field of behavioral medicine.
The Impact of Brief Clinical Interventions on Cardiovascular Reactions to Acute Stress

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The Impact of Brief Clinical Interventions on Cardiovascular Reactions to Acute Stress

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Stress

Stress is defined as a real or implied threat to one’s psychological or physical integrity. It evokes behavioral and physiological responses to psychosocial, environmental, and physical challenges, and thus can be viewed and measured from a variety of perspectives (Clark, Bond, & Hecker, 2007; Holmes & Rahe, 1967; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Lazarus & Folkman, 1984; Logan & Barksdale, 2008). Stress is a major public health concern. Over the past several decades, there has been a growing awareness of stress’s detrimental health effects, thereby necessitating a need for research into the pathways linking stress and health (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004a).

The most common form of stress is acute stress. In moderation acute stress is not harmful, however, if experienced frequently it can cause psychological and physical distress. Stress has long been linked to maladaptive functioning and various psychopathologies, having been found to play a large causal and maintaining role in psychological disorders (Kessler, 1997; Nugent, Tyrka, Carpenter, & Price, 2011). For example, individuals prone to experience acute stress are more likely to be over-aroused, irritable, and anxious. Additionally, acute stress has been found to trigger panic attacks and psychotic episodes in those predisposed to thought disorders (Chrousos, 2009).

Several epidemiological studies have also emerged evidencing stress as a major psychosocial risk factor for various medical illnesses, such as cardiovascular disease (McEwen, 2000; Yusef et al., 2004). Individuals who experience frequent acute stress are more prone to experience headaches, allergic reactions (e.g., asthma, eczema), hypertension, pain, and gastrointestinal symptoms (Black & Garbutt, 2002; Chrousos,
Currently, however, the exact mechanisms whereby stress exert its’ effects on human physiological and behavioral systems are not well understood (Epel et al., 2006).

**Allostasis and Allostatic Load**

The concepts of allostasis and allostatic load are useful frameworks for investigating the detrimental health effects of stress. Sterling and Eyer (1988) introduced allostasis as a concept that represents the active, adaptive process of regulatory physiological systems to physical, psychosocial, and environmental challenges.

Stemming from allostasis theory, allostatic load, according to McEwen and Stellar (1993), is the wear and tear on the body caused by repeated activation of the stress response. The concept of allostatic load is used to explain how frequent acute and chronic stress causes repeated activation of the stress response, resulting in damage and pathology (Clark et al., 2007; McEwen, 1998; Logan & Barksdale, 2008).

As McEwen (2000) explains there are four main conditions that lead to allostatic load: repeated frequency of the stress response, failure to habituate to repeated stressors, failure to turn off the stress response in a timely manner, and inadequate response leading to hyperactivity of other mediators. Allostatic load is measured through a composite index of indicators of cumulative strain. It is measured in physiological systems as chemical imbalances in the autonomic nervous system, central nervous system, and neuroendocrine immune system, and is thought to lead to disease through a number of routes. For example, chemical messengers, such as cortisol and epinephrine are released as part of the stress response. These chemicals can be affected by a variety of lifestyle factors (e.g., sleep, diet) and are generally beneficial. However, when the body is overexposed to them, cumulative changes occur. Repeated or dysregulated (i.e., over or
under-active) exposure to chemical mediators leads to tissue and organ specific effects, such as elevated blood pressure and increased heart rate. These specific effects lead to subsequent outcomes, which are the actual disorders that arise from allostatic load (McEwen, 2003). Examples of chronic illnesses thought to be associated with allostatic load are atherosclerosis, obesity, impaired physical capacity, anxiety, depression, and cognitive decline (Clark et al., 2007; Logan & Barksdale, 2008; Schulkin, 2004; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; McEwen, 2004).

Responses to acute stress may be mediated through a variety of behavioral pathways, including lifestyle factors such as smoking, substance use, obesity, and socioeconomic status (McEwen, 2000). As we know, the brain perceives stress (whether real or imagined) and produces behavioral responses to cope with it. These responses are determined by interpretations by the brain, which are greatly affected by psychosocial and individual differences, such as anxiety, depression, and social support (Logan & Barksdale, 2008; McEwen, 2002). For example, one person may perceive a stressful event as threatening, while another may not. Additionally, one may respond to an event to avoid a threat, where another may respond in such a way as to increase danger (McEwen, 2000). In sum, how a person interprets and copes with an event determines behaviors and habits that are highly influenced by psychosocial and individual differences. These behaviors make life more or less dangerous in the short-run and increase allostatic load over time.

In addition to behavioral pathways, direct physiological pathways also contribute to associations between psychosocial factors and the stress response. Chronic, acute physical symptoms common in anxiety disorders and depressive illnesses, suggest that
physiological stress-related processes, particularly those involving the autonomic nervous system, greatly influence these conditions (American Psychiatric Association, 2000; Chrousos, 2009; Chrousos & Gold, 1992; Sullivan, Coplan, Kent, & Gorman, 1999). Currently, there is a discrepancy among the literature in how psychosocial factors affect the stress response. For example, some studies have found positive psychological states and traits, anxiety, neuroticism, and negative affect to be associated with reduced hypothalamic-pituitary-adrenal (HPA) axis and cardiovascular reactivity to stress and poor recovery, while other studies have found hyperactive responses due to these factors (Logan & Barksdale, 2008; McEwen, 1998; McEwen, 2007; Tsigos & Chrousos, 2002). This inconsistency indicates that psychosocial factors may differentially influence individual stress responses (Chida & Hamer, 2008).

**Stress, depression, anxiety, and cardiovascular disease.** Within the allostatic load literature, the best-studied system in the human body is the cardiovascular system (McEwen, 1998). Researchers have repeatedly linked stress to cardiovascular disease, one of the most frequent causes of death in the United States (Armario et al. 2003, Rosamond et al., 2007). In addition, other psychosocial factors, including depression and anxiety, have been shown to increase one’s risk for cardiovascular disease. There have been several large reviews about the links between depression, anxiety, and heart disease. For example, Kubzansky, Kawachi, Weiss, and Sparrow (1998) found that chronic anxiety was a major risk factor for coronary heart disease and Glassman and Shapiro (1997) demonstrated that depression was associated with increased mortality from coronary heart disease. However, the mechanisms by which these relationships exist are not as well known (Logan & Barksdale, 2008).
Anxiety and depression are believed to affect the heart through three main pathways: increasing ill-health behaviors, promoting atherogenesis via hypertension arising from chronic sympathetic nervous system activation and/or dysregulation, and triggering fatal coronary events. In regards to acute stress, researchers have hypothesized that repeated episodes of acute psychological stress can result in elevated blood pressure, heart rate, and inflammatory processes (i.e., markers of allostatic load), increasing atherosclerotic plaques and stiffness of large arteries causing greater risk for cardiovascular disease (McEwen, 2002). In fact, epidemiologic studies have found an association between heightened cardiovascular reactivity in response to stress, extensive progression of atherosclerosis, and incidence of ischemic heart disease and mortality (Black & Garbutt, 2002). In sum, it is thought that anxiety and depression make individuals more susceptible to stress, resulting in repeated exposure to the stress response and an increase in allostatic load.

Currently, there is uncertainty in the literature over whether increased cardiovascular reactivity, poor cardiovascular recovery, or both lead to disease states. Additionally, research has shown mixed results on the effects depressive and anxiety symptomatology have on reactivity and recovery. For example, studies investigating cardiovascular responses to acute psychosocial stressors have suggested that depression and anxiety are related to enhanced cardiovascular reactivity, as evidenced by increased blood pressure and heart rate (de Rooij, Schene, Phillips, Roseboom, 2010; Kibler & Ma, 2004; Mausbach et al., 2005). Others, however, have found negative associations implicating a hyporeactivity to acute psychosocial stress (Carroll, Phillips, Hunt, & Der, 2007; Jezova, Makatsori, Duncko, Moncek, & Jakubek, 2004; Salomon, Clift,
Karisdottir, & Rottenberg, 2009). For example, Heopniemi et al. (2007) found that delayed heart rate recovery and lower cardiac autonomic reactivity were associated with higher carotid atherosclerosis. Mixed results in the literature may be in part due to an integrated stress response pattern, thereby supporting the concept of allostatic load that psychosocial factors could lead to different acute stress responses (Chida & Hamer, 2008).

In sum, current explanations of stress have failed to adequately clarify its association with health and chronic illness. The concept of allostatic load has applicability to subclinical and clinical psychological symptoms and suggests ways in which allostasis theory can be applied to the development of clinical interventions to increase resilience and the likelihood of better health outcomes (Logan & Barksdale, 2008). Further, responses to acute psychological challenge in the laboratory may not be considered of great clinical importance in and of themselves. However, they adequately index the way individuals respond to ordinary psychological demands, thus offering a way to investigate how disturbed stress responses have pathophysiological significance in everyday life (Chida & Hamer, 2008; Salomon et al., 2009).

**Acute Stress Testing**

As previously mentioned, acute stress has long been thought to play a role in the etiology of human psychopathologies, such as anxiety and depressive disorders, as well as medical illnesses, like cardiovascular disease (Black & Garbutt, 2002; Chrousos, 2009; Kessler, 1997; McEwen, 2000; Nugent et al., 2011; Yusef et al., 2004). Due to the clinical significance of acute stress reactions, investigating the various ways stress impacts pathological and psychopathological mechanisms is of great importance.
In order to investigate these mechanisms, standardized procedures to induce acute psychosocial stress have been widely developed (Childs, Vicini, & De Wit, 2006). One useful tool for this type of research has been laboratory-based mental stress testing. This particular testing involves the measurement of acute physiological responses, such as cardiac, neuroendocrine, and autonomic nervous system functions, to standardized stimuli (e.g., public speaking, mental arithmetic tasks, emotionally demanding social interaction) (Chida & Hamer, 2008; Hellhammer & Schubert, 2012; Kirschbaum, Pirke, Hellhammer, 1993). Although psychosocial stress testing involves artificial stimuli, it allows for the induction of consistent physiological responses with good test–retest reliability and offers a way to eliminate confounds, thereby elucidating causal factors (Brotman, Golden, & Wittstein, 2007; Chida & Hamer, 2008).

In contrast to protocols that involve physical exertion or pharmaceutical agents, acute psychosocial stress stimuli exert physiological, behavioral, and subjective stress responses (Kudielka, Wust, Kirschbaum, & Hellhammer, 2007). In a meta-analysis of 208 studies, Dickerson and Kemeny (2004) found that psychosocial stress tasks perceived as uncontrollable and that involve social-evaluative threat elicit the largest physiological response and longest recovery times. Tasks that are defined as uncontrollable are those in which outcomes are perceived to be unaffected by individual behavioral responses. Social-evaluative threat involves the perception that others could negatively judge one’s task performance, thereby impacting social esteem, self-evaluation, and social goals (Kudielka et al., 2007). The magnitude of changes in physiological parameters has been shown to depend on intensity of threat, context, and vulnerability, as well as protective
factors (Dickerson & Kemeny 2004).

**Trier Social Stress Test.** A variety of standardized procedures have been developed to induce acute psychosocial stress in a laboratory setting. One of the most widely used protocols to study physiological responses to psychosocial stress is the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993). The TSST involves a public speech and mental arithmetic task that is performed in front of an evaluative panel and therefore, involves elements of uncontrollability and social evaluation. It has been used in research with healthy study participants and clinical populations. For example, the TSST has been successfully applied in studies with patients suffering from depressive illnesses, anxiety disorders (e.g., social phobia, posttraumatic stress disorder), burnout, exhaustion, functional somatic syndromes (e.g., chronic fatigue syndrome, fibromyalgia, irritable bowel syndrome), pain, and breast cancer survivors (Kudielka et al., 2007). The TSST is one of the few available protocols that reliably elicits cardiovascular parameters, having been shown to produce moderate increases in both heart rate and blood pressure (Dickerson & Kemeny, 2004; Nater et al., 2005). It has also been proven to produce moderate increases in cortisol and subjective reports of anxiety (Childs et al., 2006; Kudielka et al., 2004a; Williams, Hagerty, & Brooks, 2004).

The standardized TSST protocol lasts for a total of thirteen minutes. It involves a brief preparation period (3 minutes) followed by a test period during which subjects are asked to deliver a free speech (5 minutes). Lastly, subjects are given a time to perform a mental arithmetic task (5 minutes). They are asked to take over the role of a job applicant invited for an interview with the company’s selection committee. The selection committee is comprised of 2-3 males or females unacquainted with the subject and has
been trained to respond in an unresponsive, neutral manner. Selection committee members are introduced to subjects as being trained to monitor nonverbal behavior. Subjects are informed that their speech will be taped and video recorded for later analysis. During the speech period, the selection committee members are given directions to respond to subjects in a standardized way. For example, if a subject ends the speech in less than five minutes, members are given prepared questions (e.g., what are your personal strengths? what are your major shortcomings?) The mental arithmetic task involves asking the subject to serially subtract an uneven number from a larger number as fast and as accurately as possible. If the subject makes an error, they are asked to restart (Kirschbaum et al., 1993).

Over the past two decades, the TSST has shown to be a useful tool in basic, applied, and clinical psychobiological research. Eliciting a range of psychobiological outcome variables, the TSST protocol has been utilized in a multitude of studies with children, adults, and the elderly to investigate the stress-disease connection (Kudielka et al., 2004a). It has even recently been successfully applied in both group and virtual reality formats (Child et al., 2006; Jonsson et al., 2010). Inducing endocrine and cardiovascular responses in 70–80% of subjects tested, the TSST procedure has been shown to significantly increase parameters such as free cortisol, total plasma cortisol, adrenocorticotropic hormone, catecholamines, growth hormone, prolactin, testosterone, blood pressure, heart rate, immune parameters, and measures of hemoconcentration and blood coagulation (Kirschbaum et al., 1993; Kudielka et al., 2007; Nater et al., 2005).

As previously mentioned, the TSST has been utilized in research investigating cardiovascular reactions to acute psychosocial stress in nonclinical, healthy populations.
For example, in a more recent study, Taylor et al. (2010) found a main effect of time showing a strong increase in heart rate, systolic blood pressure, and diastolic blood pressure from baseline to peak stress. Additionally, these researchers found that although cardiovascular parameters decreased after the post-stress task, full recovery was incomplete, as evidenced by significant baseline to recovery differences. In another study conducted by Nater et al. (2005), researchers found a significant difference in heart rate between the TSST and rest conditions and a positive relationship between salivary alpha amylase and sympathetic tone during stress. In conclusion, the TSST has been reliably shown to induce acute physiological reactivity and incomplete recovery from psychosocial stress.

The TSST has also been successful utilized to investigate the impact psychosocial factors have on cardiovascular reactions to acute psychosocial stress in clinical populations and subjects with subthreshold psychological symptomatology. For example, Chida and Hamer (2008) found that depressive mood or hopelessness exhibited significant associations with increased cardiovascular reactivity. Regarding cardiovascular recovery after stress, these researchers found that general life stress and anxiety, neuroticism, and negative affect were associated with poorer cardiovascular recovery. In contrast, a meta-analysis conducted by Kibler and Ma (2004), reported small, nonsignificant effect sizes linking depression to blood pressure reactivity and modest effect sizes for heart rate reactivity. Additionally, York et al. (2007), Carrol et al. (2007), and Solomon et al., (2009) found a negative association between depressive symptoms and cardiovascular reactivity to mental stress. In sum, and as previously mentioned, current research has shown mixed results on the effects psychological
symptoms, particularly depression and anxiety, have on physiological stress reactions.

The TSST has been utilized for well over two decades as a tool to study the mechanisms by which stress impacts physiological systems. Given its success, the next important step is to investigate whether the TSST protocol can be utilized to investigate the efficacy of psychosocial interventions (Kudielka et al., 2007).

**Psychosocial Interventions**

A key goal for clinical health practitioners is to help patients reduce the psychological and physiological burden of stress. Over the past few decades, researchers have been investigating whether social, behavioral, and psychotherapeutic interventions can help practitioners achieve this goal (McEwen, 2007). Studies have focused on psychosocial interventions aimed at managing stress, increasing resilience to stressful experiences, and treating physiologic responses to stress to improve health outcomes, such as cardiovascular function. These nonpharmacological interventions target lifestyle factors (e.g., sleep, diet, physical activity), as well as interpersonal relationships, self-efficacy, and self-esteem (Logan & Barksdale, 2008). Others teach relaxation techniques to help one cognitively, behaviorally, and physiologically cope with, counteract, or alter the way in which one experiences and recovers from stressful experiences. Psychosocial interventions can be offered in both long-term and brief formats, and are thought to offer a useful, nonpharmacological means to prevent the negative effects stress has in both healthy and clinical populations. Two such psychosocial interventions are Autogenic Training (Schultz, 1932) and Acceptance and Commitment Therapy (Hayes, Strosahl, & Wilson, 1999).

**Acceptance and Commitment Therapy.** Acceptance and Commitment Therapy
(ACT) is a therapeutic approach developed by Hayes et al. (1999) that focuses on increasing flexibility and willingness to experience stressful and anxiety provoking events and feelings. It is a comprehensive psychotherapy treatment that can be delivered in both individual and group formats over both short and long-term. ACT is based on relational frame theory, a contextual theory of language and cognition (RFT; Barnes-Holmes, Hayes, & Roche, 2001). Having grown out of the cognitive-behavioral tradition, ACT is considered a third-wave behavioral therapy. However, unlike CBT, ACT makes no attempts to change unwanted thoughts or feelings; these are not necessarily viewed as problematic. As ACT teaches, acceptance and willingness, rather than control, is what leads a person to desired goals and improvements in quality of life. In order to attain goals and implement behavior change, ACT makes use of experiential methods and a number of therapeutic strategies (Hayes, Masuda, Bissett, Luoma, & Guerrero, 2004).

Many, including ACT followers, conceptualize forms of psychopathology as unhealthy efforts to control emotions, memories, thoughts, and other internal experiences (Hayes, Wilson, Gifford, Follette & Strosahl, 1996). ACT teaches that attempts to control unwanted subjective experiences are counterproductive, resulting in an increase in psychological distress. ACT teaches positive psychological skills to counteract attempts to control, such as acceptance and cognitive defusion. Acceptance is taught as an alternative to experiential avoidance and involves an active embrace of private events without attempts to change their frequency or form. Cognitive defusion attempts to alter one’s sensitivity to undesirable thoughts (e.g., “I am being judged”) by reducing the literal quality of the experienced thought (e.g., I am having the thought “I am being
judged”). Cognitive defusion techniques attempt to decrease the believability/reality of thoughts (Hayes, Luoma, Bond, Masuda, & Lillis 2006).

Although a relatively new psychotherapy treatment, ACT has shown empirical evidence for its effectiveness in treating a variety of psychopathologies (Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Hayes et al., 2004). In a recent review of the literature, Hayes et al. (2006) found that the average post-treatment effect size for randomized controlled clinical trials of ACT ranged from .55 to .99 and average follow-up effect sizes ranged from .55 to .80. Studies have shown promising results for its efficacy in treating depression (Forman et al., 2007; Zettle & Hayes, 1986; Zettle & Rains, 1989), anxiety (Woods, Wetterneck, & Flessner, 2006; Zettle, 2003), thought disorders (Bach & Hayes, 2002), substance abuse (Hayes et al., 2004), and workplace stress (Bond & Bunce, 2003).

For example, in a published study relevant to ours, Block (2002) examined the efficacy of ACT for social anxiety symptoms. In that study, 39 college students with public speaking anxiety were semi-randomly assigned to six weeks of either ACT, cognitive-behavioral group therapy, or a no-treatment, waitlist control. Results showed that scores on social anxiety decreased for both treatment groups compared to the control condition. In addition, willingness to engage in public speaking situations increased for both treatment conditions but only the ACT group showed significant decreases in behavioral avoidance (Block, 2002). Additionally, in another study conducted by Forman et al. (2007), 101 university counseling center patients reporting moderate to severe levels of anxiety or depression were randomly assigned to traditional cognitive therapy or ACT (medium length of treatment = 15-16 sessions). Results indicated that
both cognitive therapy and ACT evidenced large, equivalent improvements in depression, anxiety, functioning difficulties, quality of life, life satisfaction, and clinician-rated functioning. In regards to mechanisms of action, researchers found that changes in “observing” and “describing” one’s experiences appeared to mediate outcomes for cognitive therapy, whereas “experiential avoidance,” “acting with awareness,” and “acceptance” mediated outcomes for ACT (Forman et al., 2007).

ACT has also been shown to be a useful therapeutic approach in behavioral medicine populations. ACT has been implemented with chronic pain (McCracken & Eccleston, 2006), cigarette smoking cessation (Gifford et al., 2004), diabetes (Gregg, Callaghan, Hayes, Glenn-Lawson, 2007), and epilepsy (Lundgren, Dahl, & Hayes, 2004) patients. One hypothesized mechanisms by which ACT benefits medical patient populations is through its emphasis on emotional acceptance rather than suppression. Research has shown that certain emotion-regulation processes, such as rumination and worry, increase distress and prolong physiological stress responses. In contrast, acceptance-oriented coping strategies promote efficient cardiovascular habituation and recovery from stress (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Low, Stanton, & Bower, 2008).

For example, in a recent study conducted by Low et al. (2008), 81 participants were randomly assigned to write about ongoing stressful experience while either (1) evaluating the appropriateness of their emotional response, (2) attending to their emotions in accepting way or, (3) simply describing the objective details of their experience. Results indicated that writing about emotions in an evaluative way led to less efficient heart rate habituation and recovery than processing emotions in an accepting manner.
(Low et al., 2008). In another study by Campbell-Sills et al. (2006), researchers compared subjective and physiological effects of emotional suppression and acceptance in 60 subjects diagnosed with anxiety and mood disorders. Subjects were randomly assigned to either a group that listened to a rationale for suppressing emotions or a group that listened to a rationale for accepting emotions. Subjective distress, heart rate, skin conductance level, and respiratory sinus arrhythmia were measured before, during, and after watching an emotion-provoking film during which the subjects were told to apply the learned instructions. Results indicated that both groups reported similar levels of subjective distress during the film but that the acceptance group displayed less negative affect during the post-film recovery period. Additionally, the suppression group showed an increased heart rate while the acceptance group showed a decreased heart rate in response to the film (Cambell-Sills et al., 2006). In sum, there is solid and growing evidence that ACT is a beneficial intervention for both nonclinical and clinical populations.

**Autogenic Training.** Autogenic Training (AT) is a relaxation technique performed through passive concentration aimed at promoting relaxation and stress relief. Johannes Schultz, a German psychiatrist and neurologist, first presented AT in the 1930s. Schultz theorized that mentally connecting with parts of one’s body produces a state similar to that experienced under hypnosis and found that his technique of AT improved health and reduced stress in many of his patients (Schultz, 1932). AT was further developed by Wolfgang Luthe and in its’ current form involves a series of statements aimed at reducing autonomic arousal (Linden, 1990; Luthe, 1969; Luthe & Schultz, 1959). AT teaches passive concentration on breathing, heartbeat, warmth, and heaviness
of body parts, and is normally taught and learned over a period of eight to ten weeks (Kanji, 1997; Morgan & Jorm, 2008). AT is based on three main principles: reduction of exteroceptive and proprioceptive afferent stimulation, mental repetition of psychophysiologicaly adapted verbal statements, and mental activity conceived as 'passive concentration' (Schulz, 1932; Kanji, 1997). Some believe that AT may be superior to other behavioral relaxation techniques because it teaches individuals how to self-regulate physiological responses and, once learned, can be practiced without a therapist (Cowings, Toscano, Timbers, Casey, & Hufnagel, 2005; Schlamann, Naglatzki, de Greiff, Forsting, & Gizewski, 2010).

AT offers individuals a way in which to exert calming effects on their mind and body. A meta-analysis by Stetter & Kupper (2002) on 60 randomized controlled trials found main effects in the range of medium-to-large effect sizes for pre-post comparisons of disease specific AT-effects. They also found that unspecific AT-effects, such as mood, cognitive performance, physiological variables, and quality of life, were larger than main effects. Analyzing study populations separately, they found positive effects in the medium effect size range for migraines, hypertension, coronary heart disease, pain disorder, anxiety disorders, mild-to-moderate depression, and functional sleep disorders. However, they also observed that AT had not been compared with cognitive or exposure therapy or psychopharmacological treatments for depression and anxiety, and that comparison with medical treatments were rare. In sum, Stetter & Kupper (2002) stated that AT results in medium-to-large clinical main effects that are stable at follow-up, but further comparison to other efficacious treatments are needed.

In a randomized controlled trial conducted by Kanji, White, & Ernst (2004a)
researchers observed the effectiveness of AT in reducing anxiety in a group of nursing students. The study investigated the differential effects interventions (e.g., AT, attention control group, time control group) have on anxiety, burnout, and cardiovascular parameters. They found a greater reduction of state and trait anxiety in the AT group than in other groups immediately after treatment but no differences between groups for burnout. They also found that the AT group had the greatest reduction immediately after treatment in systolic blood pressure, diastolic blood pressure, and heart rate. In regards to depressive symptoms, Krampen (1999) compared AT with individual psychotherapy and delayed treatment in 55 adults with depressive disorders. Subjects were randomized to one of three groups: individual psychotherapy alone, AT and individual psychotherapy, and waitlist control. In long-term follow-up (3 years), results showed that AT in combination with individual psychotherapy resulted in more positive effects than psychotherapy alone, such as lower rates of relapse and treatment reentry, and reductions in depressive and somatic symptoms. In another study with 134 adults with minor, subclinical depressive symptoms, Farne and Gnugnoli (2000) found that AT improved depressive mood after three months significantly more than in the control group. These results demonstrate that AT may be a beneficial intervention for patients suffering from minor to moderate psychological pathology.

Studies have also found AT to have positive effects in medical patients with high blood pressure, early stage cancer, irritable bowel syndrome, migraines, and acute anxiety, (Aivazyan, Zaitsev, & Yurenev, 1988; Herbert & Gutman, 1983; Hidderley & Holt, 2004; Kanji, White, & Ernst, 2004b; Kanji, White, & Ernst, 2006; Labbe, 1995; Shinozaki et al., 2010; Ter Kuile, Spinhoven, Linssen, Zitman, & Van Dyck, 1994;
In regards to cardiovascular function, Atkinson et al. (2000) found that AT showed a greater reduction immediately after treatment in blood pressure and heart rate measures compared to a control group. In 59 patients undergoing coronary angioplasty, Kanji et al. (2004b) found significant reductions in anxiety in AT group at two and five months post surgery. Additionally, in a pilot study of early stage cancer patients, Hidderley and Holt (2004) found that AT resulted in a decrease in anxiety and depression, and an increase in immune responses (as measured by T and B cell markers). In sum, AT has shown to be a beneficial relaxation intervention in both nonclinical and clinical populations.

As the literature shows, both Acceptance and Commitment Therapy and Autogenic Training have been applied successfully in a variety of study populations. However, to date, ACT and AT have yet to be applied in brief formats to investigate the differential, and possible buffering, effects these interventions have on physiological stress reactions to acute psychosocial stress.

**Present Study**

As previously mentioned, allostatic load provides a conceptual and methodological basis for elucidating behavioral and physiological mechanisms of stress. It offers a mechanistic framework for investigating how genes, early life experiences, current environments, interpersonal relationships, and lifestyle factors join to affect the structure and function of the human body. Most importantly, it offers a basis for understanding the etiology of systemic illnesses. Allostatic load allows one to consider the multiple pathways of cause and effect with consideration of interactions between
organ systems, for example, the link between cardiovascular disease and anxiety and depressive symptoms (Juster et al., 2011; McEwen, 2000).

The primary aim of the current study was to investigate physiological stress reactions to an acute psychosocial stressor and the possible buffering effects brief interventions have on these reactions. To address this aim, participants were randomized to one of three intervention conditions (Acceptance and Commitment Therapy (ACT), Autogenic Training (AT), or Attention-only Control condition) prior to undergoing an acute psychosocial stress protocol (Trier Social Stress Test; TSST).

In addition, for clinical assessment and intervention purposes, we believed it was highly warranted to investigate how certain psychological symptoms (e.g., anxiety and depression) influence acute physiological stress reactions and if they moderate the treatment response. To address these exploratory aims, participants were asked to complete a variety of psychosocial measures, including measures of anxiety and depression, prior to the TSST.

We hypothesized that brief interventions (ACT and AT) would have differential effects on physiological stress reactions. We believed that the experimental conditions (ACT and AT) would buffer the stress response and result in lower reactivity and greater recovery following the TSST. More specifically, we thought Autogenic Training would be superior to the Acceptance and Commitment Therapy condition, and that both would be superior to the Control Condition. We also hypothesized that anxiety (social anxiety, trait anxiety, anxiety sensitivity) and depressive symptoms would impact the physiological stress response in that higher symptomatology would result in greater reactivity and lower recovery following the TSST. Lastly, we hypothesized that anxiety
and depression would moderate the treatment response.

Method

Participants

Data for the present study was collected as a part of a larger study exploring the effects brief interventions have on subjective and objective stress reactions. Participants were drawn from the undergraduate population at the University of Connecticut, Storrs from Fall 2009 to Spring 2011. Participants were recruited through the Psychology 1100 and 1103 participant pool for which they registered to gain experience regarding the experimental method while earning credits as unpaid participants in psychological research. For inclusion, participants were between 18-25 years of age and were able to provide informed consent. There were no additional inclusion or exclusion criteria. A total of 123 students consented and data was collected on all of them. The average age was 19.08 (1.02) years; 66.67% were women, and 67.5% were White.

Procedure

The Institutional Review Board at the University of Connecticut approved the study, which was a randomized controlled trial utilizing a repeated measures cohort design. Participation involved one, three-hour visit to the lab. The study was broken down into three main assessment points (see Appendix A):

Time 1 lasted approximately 75 minutes. Upon arrival to the lab, informed consent was obtained. Following this process, participants completed a Demographic and Medical History Questionnaire, along with baseline physiological (heart rate and blood pressure) and psychological assessments (Anxiety Sensitivity Index, Beck Depression Inventory-II, Liebowitz Social Anxiety Scale, Spielberger State-Trait Anxiety
Inventory- Trait Form). Next participants were randomized to one of three conditions. The first condition involved an Acceptance and Commitment Therapy intervention, the second condition involved an Autogenic Training intervention, and the third condition was an Attention-only Control group (described in further detail below). All interventions lasted twenty minutes.

Time 2 lasted approximately 45 minutes. It began immediately after the intervention with a second physiological assessment (heart rate and blood pressure). Next, participants completed the Trier Social Stress Test (TSST; described in further detail below).

Time 3 lasted approximately 60 minutes. It began immediately following the conclusion of the TSST with a third physiological assessment (heart rate and blood pressure). Participants were then debriefed on the aims of the study. They were informed that the recording equipment was not turned on and that their performance was not recorded using any audiovisual equipment. They were also introduced to the two confederates who made up the selection committee. Subjects were told that these individuals were undergraduate research assistants who were trained to play roles. Following the debriefing, all participants engaged in a period of rest before a final physiological assessment (heart rate and blood pressure) was conducted. At the conclusion of the study, participants were given time to ask questions and thanked for their participation.

**Physiological Measures**

Blood pressure and heart rate were collected non-invasively utilizing a commercially available monitor (Omron Digital Blood Pressure Cuff; model number
HEM-907XL; White & Anwar, 2001). The device was used in accordance with the
directions provided with the equipment. Participants were asked to sit comfortably and
upright in a chair while the monitor’s cuff was placed around their upper arm to
simultaneously measure blood pressure and heart rate. One reading per time point was
made.

Psychological Measures

**Anxiety Sensitivity Index.** The Anxiety Sensitivity Index (ASI; Reiss, Peterson,
Gursky, & McNally, 1986) is a 16-item self-report questionnaire that assesses fear of
anxiety sensations. Subjects rate scale items on a 5-point scale (0 = very little; 4 = very
much) to indicate to what extent they agree with statements such as: ‘It scares me when I
feel faint,’ ‘It is important for me to stay in control of my emotions,’ and ‘Other people
notice when I feel shaky.’ The scale yields one total summary score ranging from 0 to
64. Various ASI reliability studies revealed a high degree of internal consistency with
Cronbach’s alpha statistics ranging from .80 to .90. The ASI has been shown to measure
a stable personality trait, as one study reported a three-year test-retest correlation of .71
(Maller & Reiss 1992; Reiss 1991). The ASI has also shown excellent diagnostic
criterion validity. Scores have been found to be strongly associated with fearfulness,
panic attacks, and subjective anxiety during biological challenge (Reiss et al., 1986;
Reiss, Peterson, & Gursky, 1988; Peterson & Sacks 1987; Maller & Reiss 1987). In the
present study, the ASI was shown to have good internal consistency (average $\alpha = .86$).

**Beck Depression Inventory-II.** The Beck Depression Inventory-II (BDI-II;
Beck, Steer, & Brown, 1996) is a 21-item self-report measure that assesses depressive
symptoms. Subjects rate scale items on a 4-point scale (0-3) to indicate which statement
in each group best describes the way they have been feeling during the past two weeks. Items measure both cognitive (e.g., self-dislike, guilt) affective (e.g., sadness, crying), and neurovegetative (e.g., appetite, sleep) symptoms. The scale yields one total summary score ranging from 0-63; higher scores indicate increasing intensity of depressive symptoms. The BDI-II is one of the most widely used depression rating scales for both clinical and research purposes. It has demonstrated excellent internal consistency with an alpha coefficient of .92, excellent concurrent validity, construct validity, and test-retest reliability across studies (Beck, Steer, Ball, & Ranieri, 1996). In the present study, the BDI-II had an average $\alpha$ of .33.

**Demographics and Medical History Questionnaire.** The Demographic and Medical History Questionnaire was created specifically for this study. It gathers basic demographic data, such as date of birth, and race and ethnicity, as well as medical information necessary for analyses of physiological data. Questions include diagnoses of mental and/or physical illness, medication(s), weight and height, substance use, and exercise.

**Liebowitz Social Anxiety Scale.** The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) is a 24-item self-report questionnaire that measures respondents’ fear and avoidance of social situations, such as social interactions (e.g., going to a party, meeting strangers) and public performances (e.g., giving a prepared oral talk, speaking up at a meeting). Each item is rated on two 4-point scales, the first measures fear/anxiety and the second measures avoidance. Scores range from 0 (no fear/never avoid) to 3 (severe fear/usually avoid). The scale yields one total summary score ranging from 0-144; higher scores indicate increasing intensity of social anxiety. Total scores have been
shown to adequately differentiate between individuals with and without social anxiety disorder (i.e., social phobia). The LSAS has demonstrated adequate reliability and validity. The scale has shown excellent internal consistency with an alpha coefficient of .95. It also has good test-retest reliability, as well as good discriminant and convergent validity (Heimberg et al., 1999; Fresco et al., 2001; Baker et al., 2002). The LSAS was shown to have good internal consistency in the present study (average $\alpha = .85$).

**Spielberger State-Trait Anxiety Inventory.** The Spielberger State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970) is a self-report questionnaire that consists of two 20-items subscales. The state subscale (“state anxiety”) assesses momentary anxiety and includes statements such as ‘I feel secure,’ ‘I am jittery,’ and ‘I feel confused.’ The trait subscale (“trait anxiety”) assesses the stable tendency to experience anxiety and includes statements such as ‘I lack self-confidence,’ ‘I feel nervous and restless,’ and ‘I feel rested.’ For the state subscale, subjects are asked to answer based on how they feel at that present moment. For the trait subscale, subjects are asked to answer based on how they generally feel. Scores for individual items range from 1 (not at all/almost never) to 4 (very much so/almost always), with total scale scores ranging from 20-80. Higher scores indicate higher degrees of anxiety. The STAI is one of the most frequently used self-report measures of state and trait anxiety with adequate reliability and validity. The trait subscale has shown good internal consistency (median alpha coefficient .90) and adequate test-retest reliability (ranging from .73 to .86). The state subscale has shown good internal consistency as well (median alpha coefficient .93) and expected low test-retest reliability given it assesses momentary anxiety (scores ranging from .16 to .62; Spielberger, 1983). In this study, we only utilized the trait
subscale (STAI- Trait), which was shown to have excellent internal consistency in the present study (average $\alpha = .92$).

**Experimental and Control Conditions**

**ACT intervention.** This experimental condition involved exercises drawn from Acceptance and Commitment Therapy (ACT; Hayes, Strosahl, & Wilson, 1999). This condition involved the researcher explaining and defining the main principles of ACT (e.g., experiential avoidance and cognitive fusion). It also utilized time to teach strategies for coping with anxiety (e.g., acceptance and defusion techniques) and involved experiential exercises (e.g., envisioning emotions as waves and observing and labeling thoughts).

**AT intervention.** This experimental condition involved Autogenic Training (AT; Schultz, 1932), which is a self-relaxation procedure. This condition involved the researcher offering a rationale for Autogenic Training. The researcher than lead participants through a series of statements, which facilitated a passive concentration on bodily perceptions (e.g., heaviness in the limbs, feelings of warmth) aimed at relieving tension and stress.

**Attention-only Control.** This control condition involved an attention-only task. The researcher read aloud from a fictional book called, “Journey Under the Sea,” which is a book from “Choose Your Own Adventure” series (Published by Chooseco LLC). These books are interactive and ask readers to make decisions throughout the story. The participants were involved in giving their input in choosing the various story lines, therefore ensuring engagement in the task.

**Challenge Task**
**Trier Social Stress Test protocol.** As previously described, the Trier Social Stress Test (TSST; Nater et al., 2005) is a widely utilized standardized protocol for the induction of psychological stress in a laboratory setting. In this study, the TSST began with the researcher leading the participant to a different room, where they were introduced to two confederates sitting at a table (trained undergraduate research assistants posing as a selection committee). In the room, there was a podium where the participant was instructed to stand, along with a video camera and a tape recorder. The researcher explained the task they were about to perform which involved assuming the role of a job applicant who is invited for an interview. Participants were told that after having a few minutes to prepare, they would be asked to give a 5-minute speech about why they are the best person for the job position. Participants were told that the selection committee was specially trained to monitor nonverbal behavior, that a video recording of their speech would be conducted, and that a voice frequency analysis of nonverbal behavior would be performed at a later date. After the instructions were read, participants were led back to room A, where they were given three minutes to prepare a speech. After their time was up, they were led back into room B where they were asked to deliver their speech. After five minutes, the committee interrupted the participant and asked him/her to serially subtract the number 13 from 1,022 as fast and as accurately as possible. On every failure the participant had to restart at 1,022 with one member of the committee interfering with, “Stop – mistake – start over at 1,022, please.” The participant was given five minutes to complete this task before being led back to room A for further assessment and debriefing.

**Data Analytic Plan**
All data analyses were performed using SPSS version 19.0 (SPSS, Inc., 2010). We first conducted exploratory data analyses to assess demographic composition of the sample and to determine whether variables of interest met assumptions of normality. Bivariate correlation matrixes were computed using data from the Demographic and Medical History Questionnaire to determine whether potential covariates should be controlled for in analyses. ANOVA’s were conducted to assess baseline differences between measures. Where appropriate, Tukey’s HSD post hoc comparisons were used for a posteriori comparisons among means. Three participants were excluded: one participant was excluded based on age, and two others did not complete the study in its entirety due to the severity of their baseline psychological symptoms and the moderately stressful nature of the TSST.

To test whether brief interventions have differential effects on physiological stress reactions over time, 3 (experimental condition) x 4 (time) repeated measures ANOVAs and ANCOVAs were conducted for heart rate, systolic blood pressure, and diastolic blood pressure. To test whether depression and/or anxiety moderate the treatment response, ANCOVAs were conducted utilizing total scores on the ASI, BDI-II, LSAS, and STAI-Trait as moderator variables, entered as covariates into analyses. To explain significant three-way interactions, ANCOVAs were conducted to describe depression and anxiety effects for each treatment group separately. ANCOVAs were also conducted to demonstrate intervention effects for low and high anxiety and depression separately. Median splits were utilized to dichotomize psychosocial variables in these analyses. Reactivity scores were calculated as the change between time 2 and time 3 cardiovascular measures. Recovery scores were calculated as change scores between time 3 and time 4.
Parameter estimates were used to graph 3-way interactions with continuous variables. This allowed us to best graphically illustrate psychological indices as moderator variables in a nonclinical sample; high and low BDI-II, STAI-Trait, ASI, and LSAS variables were defined as follows: high = 1 SD above the mean, low = 1 SD below the mean.

All analyses were first run with four time points including baseline cardiovascular measures (time 1, time 2, time 3, time 4). Separate analyses were then run on only three time points (time 2, time 3, time 4) with baseline cardiovascular values as covariates to control for baseline effects on reactivity and recovery. Next, a series of analyses were conducted to determine the influence of potentially confounding factors on baseline cardiovascular measures. Any demographic variable associated with baseline HR, SBP, or DBP at $p \leq .1$ was considered a potential confound, and thus, entered as a covariate in relevant analyses. In sum, bivariate correlations revealed that exercise was a potential confound for baseline HR; drug use, alcohol consumption, gender, and BMI were potential confounds for baseline SBP; and drug use, smoking, and BMI were potential confounds for DBP. Experimenter and time of day were also used as covariates to control for between group differences observed in baseline cardiovascular measures. Analyses were finally run with baseline cardiovascular values and the above-mentioned relevant physiological covariates. Reported results were corrected by Greenhouse-Geisser procedure where appropriate (violation of sphericity assumption), indicated by uneven degrees of freedom (Greenhouse and Geisser, 1959; Vasey and Thayer, 1987). Throughout, partial $\eta^2$ is reported as a measure of effect size.

Lastly, in order to investigate whether depressive and/or anxiety symptoms are associated with greater reactivity and lower recovery following the TSST, partial
correlations were conducted. Intervention group was entered as covariate in all analyses to control for possible intervention effects. In our analyses, power ranged from 0.4 to 1.0.

Results

Participant Characteristics

Demographic characteristics by group and gender of those included in the analyses are presented in Tables 1-2. Participants included 120 college undergraduate students. The average age of participants was 19.08 years (SD = 1.02). Two-thirds were female (n = 80). All participants reported race/ethnicity as follows: 67.5% White, Non-Hispanic; 12.5% Black/African American; 11.7% Asian; 6.6% Hispanic/Latino; Biracial/Multiracial 1.6%. Seventeen individuals (14.2%) reported a mental health diagnosis (e.g., depression, anxiety) and nine individuals (7.5%) reported a chronic medical condition (e.g., asthma, diabetes). Thirty-three individuals (27.5%) reported daily medication use (e.g., birth control, psychiatric medications). Over half the sample (57.5%) reported alcohol consumption, 5% reported cigarette smoking, and 13.3% engaged in illicit drug use (e.g., marijuana). Average body mass index was 23.44 kg/m2 (SD = 4.02) and 55% reported engagement in regular physical activity.

Descriptive Statistics for Study Measures

Descriptive statistics for all physiological measures at each time point by group are detailed in Table 3 and for each time point by gender in Table 4. Statistics for psychological measures are also included. Baseline statistics are reported below.

Baseline physiological measures. Mean baseline heart rate (bpm) for all participants (n = 120) was 76.47 bpm (SD = 14.72). ANOVAs were conducted to determine significant between group differences in baseline heart rate. A significant
difference ($p \leq 0.05$) was found, such that the AT intervention group (mean = 81.78 bpm) had a higher mean baseline heart rate compared to the ACT (mean = 73.60 bpm) and Control (mean = 74.03 bpm) groups.

Mean baseline systolic blood pressure (mmHg) for all participants (N = 120) was 126.11 mmHg (SD = 14.60). ANOVAs were conducted to determine significant between group differences in baseline systolic blood pressure. No differences between intervention groups were found.

Mean baseline diastolic blood pressure (mmHg) for all participants (n = 120) was 72.97 mmHg (SD = 8.90). ANOVAs were conducted to determine significant differences between intervention groups in baseline systolic blood pressure. A significant group difference ($p \leq 0.05$) was found between the AT (mean DBP was 76.43 mmHg, SD = 8.57) and Control (mean DBP was 69.45 mmHg, SD = 8.76) groups.

**Psychological measures.** All psychological measures were administered once at baseline. ANOVAs were conducted and no group differences were found among any of the psychological measures. Mean score for the BDI-II was 8.34 (SD = 6.87), for the LSAS was 35.50 (SD = 21.60), for the STAI-Trait was 38.88 (SD = 10.67), and for the ASI was 19.65 (SD = 9.98).

**Hypothesis 1: Impact of Brief Clinical Interventions**

For heart rate (HR), repeated measures ANOVAs revealed a main effect of time ($F(2.30, 269.55) = 59.58, p \leq .001, \eta^2 = .34$) as well as a time by group interaction ($F(4.61, 269.55) = 3.82, p = .003, \eta^2 = .06$). Post hoc tests indicate a group differences in HR at baseline among AT and ACT ($p = .032$), and AT and Control groups ($p = .045$), but at no other time point. Thus, after controlling for baseline HR, repeated measures
ANCOVAs revealed no effects. However, once relevant covariates (exercise, time of day, experimenter) were controlled for along with baseline HR, a main effect of time reemerged \((F(1.91, 216.08) = 3.25, p = .04, \eta^2 = .03)\), demonstrating a significant change in HR over time.

For systolic blood pressure (SBP), repeated measures ANOVAs revealed a main effect of time \((F(2.88, 337.22) = 53.08, p \leq .001, \eta^2 = .31)\). Repeated measures ANCOVAs, with baseline SBP as a covariate, revealed no main or interaction effects. Similarly, controlling for relevant covariates (drug use, alcohol, gender, BMI, time of day, experimenter) along with baseline SBP did not reveal any significant effects.

For diastolic blood pressure (DBP), repeated measures ANOVAs revealed a main effect of time \((F(2.9, 338.72) = 43.86, p \leq .001, \eta^2 = .27)\) as well as a time by group interaction \((F(5.79, 338.72) = 2.39, p = .03, \eta^2 = .04)\). Post hoc tests indicate a group differences in DBP at baseline among the AT and Control groups \((p = .001)\). Controlling for baseline DBP revealed no main or interaction effects. Additionally, controlling for relevant covariates (drug use, smoker, BMI, time of day, experimenter) along with baseline DBP did not reveal any significant effects.

**Hypothesis 1a: Depressive and/or Anxiety Symptoms as Treatment Moderators**

**Depression (Beck Depression Inventory-II).** For HR, repeated measures ANCOVAs, with total BDI-II score entered as a covariate to test for moderation, revealed a main effect of time \((F(2.27, 258.18) = 20.10, p \leq 0.001, \eta^2 = .15)\) as well as a time by group interaction \((F(4.53, 258.18) = 2.47, p = .04, \eta^2 = .04)\). Post hoc tests indicate a group differences in HR at baseline among AT and ACT \((p = .032)\), and AT and Control groups \((p = .045)\), but no group differences at any other time point. Thus, controlling for
baseline HR revealed no main or interaction effects. However, once relevant covariates (exercise, time of day, experimenter) were controlled for along with baseline HR, a main effect of time reemerged ($F(1.89, 208.20) = 3.60, p = .031, \eta^2 = .03$), demonstrating a significant change in HR over time.

For SBP, repeated measures ANCOVAs, with total BDI-II score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.82, 321.99) = 25.77, p \leq .001, \eta^2 = .18$) as well as a 3-way time by group by depression interaction ($F(5.65, 321.99) = 2.24, p = .043, \eta^2 = .04$), which remained after controlling for baseline SBP ($F(3.8, 214.4) = 2.46, p = .05, \eta^2 = .04$); see Figures 1-5. The 3-way interaction demonstrates that across time points, SBP is affected differently among intervention groups depending on level of depression. In the AT group, subjects with lower levels of depressive symptomatology displayed greater SBP reactivity to the TSST compared to those with higher levels of symptomatology ($F(1, 37) = 4.72, p = .036, \eta^2 = .11$); see Figure 1. There was neither a significant difference in SBP reactivity or recovery between levels of depression in either the ACT or Control conditions nor a significant difference in SBP between groups at a given level of depression at any time point ($p$’s $> .1$); See Figures 2-5. Once relevant covariates (drug use, alcohol, gender, BMI, time of day, experimenter) were controlled for along with baseline SBP, the 3-way interaction was no longer significant ($p = .06$).

For DBP, repeated measures ANCOVAs, with total BDI-II score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.9, 330.07) = 19.56, p \leq .001, \eta^2 = .15$) as well as a time by group interaction ($F(5.79, 330.07) = 73.09, p = .047, \eta^2 = .02$). Post hoc tests indicate a group difference in DBP at baseline among the
AT and Control groups ($p = .001$). Once baseline DBP was controlled for, no main or interaction effects were observed. Similarly, controlling for relevant covariates (drug use, smoker, BMI, time of day, experimenter), along with baseline DBP, did not result in any significant effects.

**Anxiety Sensitivity (Anxiety Sensitivity Index).** For HR, repeated measures ANCOVAs, with total ASI score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.28, 259.3) = 5.16, p = .004, \eta^2 = .04$). Controlling for baseline HR revealed no main or interaction effects. However, once relevant covariates (exercise, time of day, experimenter) were controlled for along with baseline HR, a main effect of time reemerged ($F(1.9, 208.64) = 3.13, p = .048, \eta^2 = .03$), indicating a significant change in HR over time.

For SBP, repeated measures ANCOVAs, with total ASI score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.86, 326.06) = 16.96, p \leq 0.001, \eta^2 = .13$). Once baseline SBP was added as a covariate, however, no main or interaction effects were observed. Similarly, controlling for relevant covariates (drug use, alcohol, gender, BMI, time of day, experimenter), along with baseline SBP, did not result in any significant effects.

For DBP, repeated measures ANCOVAs, with total ASI score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.89, 329.12) = 7.54, p \leq 0.001, \eta^2 = .06$). Once baseline DBP was controlled for, however, no main or interaction effects were observed. Similarly, controlling for relevant covariates (drug use, smoker, BMI, time of day, experimenter), along with baseline DBP, did not result in any significant effects.
**Social Anxiety (Liebowitz Social Anxiety Scale).** For HR, repeated measures ANCOVAs, with total LSAS score entered as a covariate to test for moderation, revealed a main effect of time \( (F(2.3, 255.01) = 9.18, p \leq 0.001, \eta^2 = .08) \). Controlling for baseline HR revealed no main or interaction effects. However, once relevant covariates (exercise, time of day, experimenter) were controlled for along with baseline HR, a main effect of time reemerged \( (F(1.92, 205.01) = 5.02, p = .008, \eta^2 = .05) \), indicating a significant change in HR over time.

For SBP, repeated measures ANCOVAs, with total LSAS score entered as a covariate to test for moderation, revealed a main effect of time \( (F(2.9, 322.33) = 10.39, p \leq 0.001, \eta^2 = .09) \) as well as a 3-way time by group by social anxiety interaction \( (F(5.81, 322.33) = 2.68, p = .016, \eta^2 = .05) \). This 3-way interaction remained after controlling for baseline SBP \( (F(3.91, 214.86) = 3.46 p = .01, \eta^2 = .06) \) and after controlling for relevant covariates (drug use, alcohol, gender, BMI, time of day, experimenter) along with baseline SBP \( (F(3.9, 200.77) = 3.8 p = .006, \eta^2 = .07) \); See Figures 6-10. The 3-way interaction demonstrates that across time points, SBP is affected differently among intervention groups depending on level of social anxiety. In the AT group, individuals with lower levels of social anxiety symptomatology recovered less after the TSST than those with higher levels of symptomatology \( (F(1, 37) = 6.17, p = .018, \eta^2 = .14) \); see Figure 6. At time 4, there was a significant difference in SBP between levels of social anxiety symptomatology in the AT group \( (F(1, 37) = 5.13, p = .029, \eta^2 = .12) \); see Figure 6. There was neither a significant difference in SBP reactivity or recovery between levels of social anxiety in either the ACT or Control conditions nor a significant difference in
SBP between groups at a given level of social anxiety at any time point \( (p’s > .1) \); see Figures 7-10.

For DBP, repeated measures ANCOVAs, with total LSAS score entered as a covariate to test for moderation, revealed a main effect of time \( (F(2.88, 320.10) = 10.0, p \leq 0.001, \eta^2 = .08) \) as well as a 3-way time by group by social anxiety interaction \( (F(5.77, 320.1) = 2.34, p = .034, \eta^2 = .04) \). This 3-way interaction remained after controlling for baseline DBP \( (F(3.92, 215.75) = 2.7, p = .032, \eta^2 = .05) \); see Figures 11-15. The 3-way interaction demonstrates that across time points, DBP is affected differently among intervention groups depending on level of social anxiety. In the AT group, individuals with lower levels of social anxiety symptomatology recovered less after the TSST than those with higher levels of symptomatology \( (F(1, 37) = 4.38, p = .043, \eta^2 = .11) \); see Figure 11. There was neither a significant difference in DBP reactivity or recovery between levels of social anxiety in either the ACT or Control conditions nor a significant difference in DBP between groups at a given level of social anxiety at any time point \( (p’s > .1) \); see Figures 12-15. Once relevant covariates (drug use, smoker, BMI, time of day, experimenter) were controlled for along with baseline DBP, the 3-way interaction was no longer significant \( (p = .056) \).

**Trait Anxiety (Spielberger State Trait Anxiety Inventory- Trait).** For HR, repeated measures ANCOVAs, with total STAI-Trait score entered as a covariate to test for moderation, revealed no main or interaction effects. Similarly, controlling for baseline HR, no main or interaction effects were observed. However, once relevant covariates (exercise, time of day, experimenter) were controlled for along with baseline
HR, a main effect of time was emerged ($F(1.91, 209.54) = 3.34, p = .039, \eta^2 = .03$), indicating a significant change in HR over time.

For SBP, repeated measures ANCOVAs, with total STAI-Trait score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.85, 324.61) = 5.71, p = .001, \eta^2 = .05$). Once baseline SBP was added as a covariate, however, no main or interaction effects were observed. Similarly, controlling for relevant covariates (drug use, alcohol, gender, BMI, time of day, experimenter), along with baseline SBP did not result in any significant effects.

For DBP, repeated measures ANCOVAs, with total STAI-Trait score entered as a covariate to test for moderation, revealed a main effect of time ($F(2.88, 328.36) = 4.17, p = .007, \eta^2 = .04$). Once baseline DBP was controlled for, however, no main or interaction effects were observed. Similarly, controlling for relevant covariates (drug use, smoker, BMI, time of day, experimenter), along with baseline DBP and STAI-Trait did not result in any significant effects.

**Hypothesis 1b: Association of Depressive and Anxiety Symptoms with Cardiovascular Reactivity and Recovery**

Partial correlations, with group as covariate, revealed no associations between depression (as measured by total score on the BDI-II) and HR, SBP, or DBP reactivity or recovery. Additionally, no associations were found between social anxiety (as measured by total score on the LSAS) or trait anxiety (as measured by total score on the STAI-Trait) and HR, SBP, or DBP reactivity or recovery. However, HR reactivity was found to be positively associated with anxiety sensitivity (as measured by total score on the ASI) ($pr = .21, p = .027$). No associations were observed between HR recovery and ASI.
Discussion

Purpose of the Present Study

Stress is a psychosocial risk factor that has long been linked to medical illness and various psychopathologies (Kessler, 1997; McEwen, 2003; Nugent et al., 2011). While it has been well established that behavioral factors (e.g., diet, exercise, substance use) play a critical role in the onset and maintenance of stress-related illness, the role of internal biological mechanisms of stress are less understood (Luthar, 2003; McEwen, 2000). As discussed throughout the introduction, the concepts of allostasis and allostatic load offer a useful framework for investigating the psychobiological mechanisms affected by stress, particularly those involving the cardiovascular system (McEwen & Stellar 1993; Sterling & Eyer, 1988).

In our study we set out to examine cardiovascular stress reactions to an acute psychosocial stressor and were particularly interested in investigating whether brief psychosocial interventions would attenuate these reactions. As previously mentioned, although responses to stressors in a laboratory setting may not be of great clinical importance, they indicate how a person responds to stressful demands in their everyday life and offer a useful way to examine disturbed stress responses that may have pathological consequences (Chida & Hamer, 2008; Salomon et al., 2009). Additionally, laboratory settings provide a controlled environment whereby direct physiological outcomes to clinical interventions can be validly acquired. Another aim of our study was to explore how certain psychological symptoms (e.g., anxiety and depressive symptomatology) influence acute physiological stress reactions and whether these symptoms moderate the treatment response to brief interventions. Given the current
discrepancy among the literature in regards to how psychosocial factors affect the stress response, we believe this aim offers important implications for clinical assessment and intervention.

To our knowledge, this is the first study to examine the effects brief clinical interventions have on cardiovascular reactions (e.g., heart rate, systolic blood pressure, diastolic blood pressure) to an acute psychosocial stress paradigm, and whether common psychosocial factors (e.g., anxiety and depression) moderate the treatment response.

**Hypothesis 1**

In our study, participants were randomized to one of three intervention conditions. The first experimental condition was developed based on Acceptance and Commitment Therapy (ACT), a therapeutic approach that focuses on increasing one’s flexibility and willingness to accept stressful and anxiety provoking events and feelings (Hayes et al., 1999). ACT has proven empirical evidence for its effectiveness in treating a variety of psychopathologies, including depression and anxiety, and has been shown to affect positive cardiovascular outcomes (Cambell-Sills et al., 2008; Forman et al., 2007; Hayes et al., 2004; Low et al., 2008; Woods et al., 2006; Zettle, 2003; Zettle & Hayes, 1986; Zettle & Rains, 1989).

The second experimental condition was Autogenic Training (AT), a self-relaxation procedure that involves a series of statements aimed at reducing autonomic arousal (Linden, 1990). AT has shown clinical main effects in the range of medium-to-large effect sizes in both nonclinical and clinical populations. For example, a number of studies have shown AT to be a beneficial for the reduction of anxiety and depressive symptoms, as well as a variety of cardiovascular outcomes, such as heart rate and blood
Based on literature demonstrating the positive benefits relaxation and acceptance techniques have on health outcomes, we hypothesized that brief interventions would have differential effects on heart rate, systolic blood pressure, and diastolic blood pressure following a psychosocial stress paradigm. Specifically, we thought the ACT and AT experimental conditions would attenuate the stress response, resulting in positive cardiovascular outcomes (e.g., lower reactivity and greater recovery following the TSST). Further, because we were measuring acute physiological outcomes, we hypothesized that the AT condition, being a somatic relaxation technique, would be superior to the ACT condition, and that both experimental conditions would be superior to the Attention-only Control condition. In support of our hypothesis, results showed a time by group interaction for heart rate and diastolic blood pressure, indicating that brief interventions differentially affected heart rate and diastolic blood pressure over time. However, after controlling for relevant covariates, particularly baseline differences in heart rate and diastolic blood pressure, these effects were no longer evident. Additionally, contrary to our hypothesis, we did not observe any effects on systolic blood pressure.

One way of interpreting our results is to suggest that the original time by group interactions for both heart rate and diastolic blood pressure were driven by baseline differences between groups. However, another interpretation is that intervention effects, which could no longer be measured after controlling for baseline time points, drove our results. For example, in regards to heart rate outcomes, there was a greater reduction in
heart rate from baseline to pre-TSST in the AT group compared to the ACT and Control groups. This suggests that there was a greater intervention effect for the AT group compared to the other groups. However, because we had to control for baseline differences in subsequent analyses, and therefore remove time 1, the intervention effect could no longer be displayed. Therefore, it may be that the observed time by group interaction was in fact due to the AT intervention effect rather than baseline differences in heart rate.

In regards to diastolic blood pressure, the same interpretations must be considered. Again, it may be that the group by time interaction was driven by baseline differences in diastolic blood pressure. However, another possibility is that intervention effects, which could no longer be measured after controlling for baseline time points, drove our results. On further examination, the AT group again showed a significant intervention effect compared to the ACT and Control groups, which was evidenced by a lowering of diastolic blood pressure from baseline to pre-TSST. However, because we had to control for baseline differences in subsequent analyses, and therefore remove time 1, this effect could no longer be demonstrated. Therefore, as with heart rate, it may be that the intervention effects of the AT group rather than baseline differences in diastolic blood pressure drove the observed time by group interaction.

**Hypothesis 1a**

Our second hypothesis was that anxiety and depressive symptomatology would moderate the treatment response. This hypothesis was more exploratory in nature since there is no literature, to our knowledge, investigating whether psychosocial factors moderate cardiovascular outcomes to brief clinical interventions.
We hypothesized that anxiety and depression levels would moderate the treatment response to brief clinical interventions. In regards to depressive symptomatology and systolic blood pressure, our hypotheses were supported. Results showed a significant three-way interaction indicating that across time points, SBP was affected differently among intervention groups depending on level of depression. Within the Autogenic Training group, subjects with higher levels of depressive symptomatology had lower SBP reactivity to the TSST than those with less symptomatology. There were no significant differences between levels of depression in SBP reactivity, recovery, or across any time points within either the ACT or Control conditions. This finding provides evidence that AT may be most beneficial for individuals with higher levels of depression. Once relevant covariates were controlled for, the three-way interaction was no longer significant ($p = .06$).

In regards to social anxiety symptomatology and systolic and diastolic blood pressure, our hypotheses were also supported. Results showed a significant three-way interaction indicating that across time points, after controlling for all relevant covariates, SBP was affected differently among intervention groups depending on level of social anxiety. Within the AT group, subjects with higher levels of social anxiety symptomatology had greater SBP recovery following the TSST than those with less symptomatology. Additionally, within the AT group, subjects with higher levels of social anxiety symptomatology had significantly lower SBP than those with less symptomatology at time 4. There were no significant differences between levels of social anxiety in SBP reactivity, recovery, or across any time points within either the Acceptance and Commitment Therapy or Control conditions. Overall, this finding
provides evidence that AT may be most beneficial for individuals with higher levels of social anxiety.

Additionally, results showed a significant three-way interaction indicating that across time points, DBP is affected differently among intervention groups depending on level of social anxiety. This finding held after controlling for baseline differences in diastolic blood pressure. Within the AT group, subjects with higher levels of social anxiety symptomatology had greater DBP recovery following the TSST than those with less symptomatology. There were no significant differences between levels of social anxiety in DBP reactivity, recovery, or across any time points within either the ACT or Control conditions. This finding also provides evidence that AT may be beneficial for individuals with higher levels of social anxiety. However, once other relevant covariates were controlled for along with baseline DBP, the three-way interaction was no longer significant (p = .056).

In sum, our results show that depressive and social anxiety symptomatology moderated the effectiveness of our brief clinical interventions, as measured by cardiovascular outcomes. Analyzing these findings together it is evident that subjects with greater levels of social anxiety and those with greater levels of depressive symptoms benefited more from the Autogenic Training intervention than those with lower levels. It is an interesting finding that individuals with higher levels of psychopathology showed greater cardiovascular benefits from AT. Because the intervention was brief, and therefore less potent than if it had been practiced on several occasions, some may have expected individuals with lower levels of psychopathology to benefit the most from it.

One interpretation for our findings is that nonclinical subjects with greater social
anxiety (and depression) were more motivated to participate in the intervention, and thus, learned and benefited more from it, thereby affecting cardiovascular outcomes. There is a vast literature on the importance of motivation for the acquisition and transfer of techniques and skills taught in psychosocial interventions (see Ryan et al., 2011 for a review). In fact, motivation has been found to be so important for learning and transfer that an empirically supported directive therapeutic style, Motivational Interviewing, has been developed to focus on helping patients increase it (Miller, 1983; Miller & Rollnick, 1991; Miller & Rollnick, 2002; Hettema, Steele, & Miller, 2005). Based on motivational theories (e.g., Self-Determination Theory; Deci & Ryan, 1985), it is reasonable that individuals with greater intrinsic motivation, perhaps those with more symptoms of psychopathology, would be more likely to fully engage in a brief version of Autogenic Training, which is directly aimed at reducing the symptoms they are experiencing.

**Hypothesis 1b**

As discussed in the introduction, current research has shown mixed results for the effects depression and anxiety have on physiological stress reactions. For example, studies investigating physiological responses to acute psychosocial stressors have suggested that depression and anxiety are related to enhanced cardiovascular reactivity, as evidenced by increased blood pressure and heart rate (de Rooij et al., 2010; Kibler & Ma, 2004; Mausbach et al., 2005). However, other researchers have found negative associations implicating a hyporeactivity to acute psychosocial stress (Carroll et al., 2007; Jezova et al., 2004; Salomon et al., 2009). In addition, it is important to note that there is a lack of research investigating the relationship between depressive and anxiety
symptomatology and cardiovascular recovery, rather most studies concentrate on reactivity.

Our study design offered an opportunity for us to add to the literature regarding the possible associations between certain key psychosocial factors and cardiovascular reactivity and recovery. Based on prior research, we hypothesized that after controlling for treatment group, anxiety and depressive symptomatology would be associated with greater cardiovascular reactivity and lower recovery following the TSST. Our hypothesis was partially supported in that heart rate reactivity was found to be positively associated with anxiety sensitivity (as measured by total score on the ASI). However, no additional measures were associated with any other cardiovascular parameters.

In our study, mean scores for all four measures (BDI-II, ASI, LSAS, STAI-Trait) were all in the nonclinical range. Therefore, one interpretation of these results is that certain associations were not observed because our study included a nonclinical population that displayed subthreshold symptomatology. Our results imply that lower levels of psychopathology (i.e., a few symptoms of anxiety or depression), although meaningful, may not affect physiological parameters as drastically as more severe pathology.

In further discussion of our results, we did not specifically study individual differences in regards to psychopathology. For example, a biological predisposition to certain disorders may engender more physiological effects. Additionally, certain symptoms of anxiety and depression may elicit cardiovascular responses (e.g., somatic/physical symptoms) more than others (e.g., cognitive/affective symptoms). It is also reasonable to ascertain that age of onset and length of time experiencing certain
pathology may have more or less of an effect on physiological parameters.

Unfortunately, because we did not directly study these types of individual differences, we were not able to test these possibilities. Therefore, one important implication of our study is that direct consideration of individual differences when investigating physiological outcomes is imperative.

**Limitations**

Although there are many strengths of this study, such as its novelty, there are some important limitations to mention. First, although our subjects were randomly assigned to groups, there were still baseline differences in two of three cardiovascular parameters. This indicates that our baseline data might represent anticipation stress rather than a true baseline condition. Therefore, future research utilizing a similar study design should take baseline anticipation stress into consideration by adequately measuring and treating it as a confounding factor. Second, we did not measure cardiovascular parameters during the TSST; rather we assessed reactivity immediately following the cessation of the protocol. Although this method has been utilized in various studies, assessing cardiovascular parameters during the TSST may provide a more valid measure of reactivity. Third, because of our sample population and sample size of men, we were not able to investigate possible age and gender differences in treatment response. Several studies (e.g., Kelly, Tyrka, Anderson, Price, Carpenter, 2008; Kudielka et al, 2004a) have found age and gender differences in responses to social stress challenges. Therefore, exploring age and gender differences in treatment response involving objective outcome measures is warranted. Lastly, we did not directly measure
motivation and treatment engagement, which can both be considered as possible confounds decreasing intervention effectiveness.

**Future Directions**

There is a growing awareness of the detrimental health effects stress has on individuals. This awareness has led to research endeavors investigating the exact pathways linking stress and health. Just as important as elucidating these mechanisms, there is also a need for the development and study of specific interventions to counteract the effects of stress. Our study has specifically added to this field by showing that brief clinical interventions involving somatic relaxation, particularly Autogenic Training, may be beneficial to individuals undergoing acute stress. Additionally, our study demonstrated that individuals with higher levels of depression and/or anxiety might benefit the most from these targeted interventions.

Particularly within the field of behavioral medicine, our findings offer important implications for assessment and treatment. Acute physiological reactivity from stressful medical and dental procedures is common and may lead to negative outcomes (Cohen, Fiske, & Newton, 2000; Johnsen et al., 2003; Munafo & Stevenson, 2001; Mavros et al., 2011; Pignay-Demaria, Lesperance, Demaria, Frasure-Smith, & Perrault, 2003). Moreover, it is well evidenced that mild and severe forms of anxiety and depressive disorders are high in medical and dental patient populations and are considered risk factors for disease (Demertizis & Craske 2006; Moussavi et al., 2007; Sherbourne, Jackson, Meredith, Camp, & Wells, 1996; Stoudemire, 1996; Wittchen et al., 2002). Targeting certain individuals more prone to poor outcomes (e.g., those suffering from mild or severe psychopathology) and administering brief clinical interventions prior to
stressful procedures may attenuate and/or prevent negative health outcomes. Future research investigating the effectiveness brief clinical interventions have on these populations is not only warranted, but highly feasible.
References


...


Appendix A. Diagram of Participant Involvement in Larger Study

<table>
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<th>Time 1 (~75 mins)</th>
<th>Time 2 (~45 mins)</th>
<th>Time 3 (~60 mins)</th>
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<td>Pre-Trier Assessment (~30 mins)</td>
<td>Post-Trier Assessment (~20 mins)</td>
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<td>- Physiological Measures</td>
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<tr>
<td>- Physiological Measures</td>
<td>- Psychological Measures</td>
<td>- Process Measures</td>
</tr>
<tr>
<td>- Psychological Measures</td>
<td>Trier Social Stress Test (~15 mins)</td>
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<tr>
<td>Intervention Sessions (~20 mins)</td>
<td>- Instructions</td>
<td>Debriefing (~15 mins)</td>
</tr>
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<td>- Group 1: ACT</td>
<td>- Anticipation</td>
<td>Rest Period (~15 mins)</td>
</tr>
<tr>
<td>- Group 2: AT</td>
<td>- Free Speech</td>
<td>- Physiological Measures</td>
</tr>
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<td>- Group 3: Control</td>
<td>- Subtraction</td>
<td>Wrap Up (~10 mins)</td>
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<td>- Questions</td>
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Table 1

*Participant Characteristics by Group*

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*a*Correlates with baseline HR ($p \leq 0.1$);

*b*Correlates with baseline SBP ($p \leq 0.1$);

*c*Correlates with baseline DBP ($p \leq 0.1$)
Table 2

*Participant Characteristics by Gender*

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<td>Illicit Drug Use (%)</td>
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*Correlates with baseline HR \((p \leq 0.1)\); \(^b\)Correlates with baseline SBP \((p \leq 0.1)\); \(^c\)Correlates with baseline DBP \((p \leq 0.1)\)
Table 3

*Descriptive Statistics for Study Measures by Group*

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### Control Group

**Post-TSST Total**
- ACT Group: 79.65
- AT Group: 79.13
- Control Group: 77.0

**Recovery Total**
- ACT Group: 73.56
- AT Group: 74.3
- Control Group: 72.33

**Beck Depression Inventory II**

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**Liebowitz Social Anxiety Scale**

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**Spielberger State Trait Anxiety Inventory- Trait**

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**Anxiety Sensitivity Index**

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\*Statistically significant difference compared to ACT and Control Groups (p ≤ 0.05);
\*Statistically significant difference compared to Control Group (p ≤ 0.05)
Table 4

Descriptive Statistics for Study Measures by Gender

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*aStatistically significant difference compared to Females (p ≤ 0.05)*
Figure 1

*Autogenic Training Group: Depression and Systolic Blood Pressure*

*Figure 1*. This figure helps demonstrate the 3-way interaction (time by group by depression). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low depressive symptomatology in the Autogenic Training group.
Figure 2

Acceptance and Commitment Therapy: Depression and Systolic Blood Pressure

*Figure 1.* This figure helps demonstrate the 3-way interaction (time by group by depression). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low depressive symptomatology in the Acceptance and Commitment Therapy group.
Figure 3

*Control Group: Depression and Systolic Blood Pressure*

*Figure 3.* This figure helps demonstrate the 3-way interaction (time by group by depression). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low depressive symptomatology in the Control group.
Figure 4

*High Depressive Symptomatology: Group and Systolic Blood Pressure*

*Figure 4.* This figure helps demonstrate the 3-way interaction (time by group by depression). It represents the measure of systolic blood pressure (mmHg) over time between intervention groups in those with high depressive symptomatology.
Figure 5

Low Depressive Symptomatology: Group and Systolic Blood Pressure

Figure 5. This figure helps demonstrate the 3-way interaction (time by group by depression). It represents the measure of systolic blood pressure (mmHg) over time between intervention groups in those with low depressive symptomatology.
Figure 6

*Autogenic Training Group: Social Anxiety and Systolic Blood Pressure*

This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Autogenic Training group.
Figure 7

Acceptance and Commitment Therapy: Social Anxiety and Systolic Blood Pressure

*Figure 7.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Acceptance and Commitment Therapy group.
Figure 8

Control Group: Social Anxiety and Systolic Blood Pressure

*Figure 8.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of systolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Control group.
Figure 9

*High Social Anxiety Symptomatology: Group and Systolic Blood Pressure*

*Figure 9.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of systolic blood pressure (mmHg) over time between intervention groups in those with high social anxiety symptomatology.
Figure 10

Low Social Anxiety Symptomatology: Group and Systolic Blood Pressure

This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of systolic blood pressure (mmHg) over time between intervention groups in those with low social anxiety symptomatology.
Figure 11

Autogenic Training: Social Anxiety and Diastolic Blood Pressure

Figure 11. This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of diastolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Autogenic Training group.
Acceptance and Commitment Therapy: Social Anxiety and Diastolic Blood Pressure

Figure 12

This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of diastolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Acceptance and Commitment Therapy group.
Figure 13

*Control Group: Social Anxiety and Diastolic Blood Pressure*

*Figure 13.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of diastolic blood pressure (mmHg) over time between subjects with high and low social anxiety symptomatology in the Control group.
Figure 14

*High Social Anxiety Symptomatology: Group and Diastolic Blood Pressure*

*Figure 14.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of diastolic blood pressure (mmHg) over time between intervention groups in those with high social anxiety symptomatology.
Figure 15

*Low Social Anxiety Symptomatology: Group and Diastolic Blood Pressure*

*Figure 15.* This figure helps demonstrate the 3-way interaction (time by group by social anxiety). It represents the measure of diastolic blood pressure (mmHg) over time between intervention groups in those with low social anxiety symptomatology.