PREDICTING IMPROVEMENT IN COGNITIVE BEHAVIORAL THERAPY FOR SOMATIZATION DISORDER: THE ROLE OF ALEXITHYMIA

by

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ABSTRACT OF THE DISSERTATION

Predicting Improvement in Cognitive Behavioral Therapy for Somatization Disorder:

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Alexithymia has been defined as difficulty identifying and expressing emotions and an externally oriented mode of thinking. Previous research has linked alexithymia with somatoform symptoms yet there is little prospective data examining the role of alexithymia in somatization disorder. Thus, changes in alexithymia were examined over the course of a 10-session controlled trial of cognitive behavioral therapy for somatization disorder. It was predicted that the treatment would lead to reductions in alexithymia not seen in the group whose physicians received only a psychiatric consultation letter (PCL) and that CBT participants would score significantly lower on alexithymia than PCL participants. It was also hypothesized that changes in alexithymia from pre- to post-test, assessed through the Toronto Alexithymia Scale (TAS-20), would predict improvement in somatization symptoms, as assessed through the Clinician's Global Impression Scale for Somatization Disorder (CGI-SD) at post-test and at 12-month follow-up. Daily symptom diaries and physical functioning, assessed through the MOS-PF, were also examined as outcomes. Participants were 84 individuals diagnosed with full somatization disorder according to the DSM-IV. Baseline severity and post-

treatment mental health, defensiveness, and somatosensory amplification were controlled for in regression analyses. Results partially supported hypotheses. Participants in the CBT condition decreased more in the TAS-20 and the DIF domain and marginally more in the EOT domain over the course of the study than participants in the PCL condition. They differed significantly from PCL participants at post-treatment in the EOT domain but not in the full scale TAS-20 or in any other domains. There were no significant differences between groups in alexithymia at follow-up. Decreases in alexithymia were significantly correlated with improvement in somatization symptoms and greater physical functioning. Although decreases in alexithymia significantly predicted certain outcomes at post-treatment and follow-up over and above control variables, tests for mediation yielded non-significant results. Findings from the current study support emotional functioning as a factor in somatization but do not advance the notion of alexithymia as a mediator of improvement in treatment for somatization disorder. Implications and suggestions for future areas of research are discussed.

Dedication

With love, I dedicate this thesis to my husband and to my father, who would be proud.

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Introduction

Somatization disorder is a syndrome characterized by multiple unexplained physical complaints that often results in significant functional impairment and extensive medical health care utilization. Individuals with somatization disorder exhibit higher rates of disability and experience great distress and high levels of psychiatric illness (Katon et al., 1991; Smith, Monson, & Ray, 1986). In addition, this population is characterized by a disproportionate use of health care services, often with little benefit (Smith et al., 1986). It has been suggested that persons who somatize, as a group, exhibit predominantly somatic responses to stress as opposed to cognitive or affective responses (Lipowski, 1988). This somatic response distinguishes them from individuals experiencing primary mood or anxiety disorders. Until recent efforts (e.g., Allen, Woolfolk, Escobar, Gara, & Hamer, 2006), no psychotherapeutic treatment had been shown empirically to benefit this population.

Researchers have long put forth a hypothesis that deficits in the experience of emotion played a central role in the etiology of psychosomatic illness. Individuals with psychosomatic illness were often described as exhibiting great difficulty in the verbal and symbolic representation of emotions, and as appearing unimaginative and lacking in fantasy (Alexander, 1950: Nemiah & Sifneos, 1970; Ruesch, 1948; Sifneos, 1973). Original theories of psychosomatic illness postulated that repression of dynamic conflicts led to their expression in somatic symptoms (Breuer & Freud**,** 1957; Alexander, 1950; Freud, 1997). In Studies on Hysteria, Breuer and Freud (1957) argued that the psychological mechanism responsible for bringing about hysterical (i.e., psychosomatic) symptoms involved the inhibition of affect related to trauma. Specifically, they argued

that the processes of the mind worked in equilibrium, and when mental excitations were not expressed through action or verbal expression, these excitations found release in somatic symptoms. This process was described in terms of tension, such that an overly high state of tension in the psychic system would lead to the excitation of the peripheral organs and an emotional response that would be released through somatic expression. A 'somatic compliance' (Freud, 1997) was necessary in order for psychic material to convert to somatic symptoms, however; this was what distinguished the formation of hysterical symptoms from those of a mental nature, such as obsessions or phobias (Freud, 1997). Hysterics were also described as appearing unconcerned about their symptoms, exhibiting what was called "la belle indifference" (Breuer & Freud, 1957). In order to remove the hysterical symptoms, according to this theory, the psychic origins of the physical symptoms would have to be brought out into the open through verbal expression.

Drawing on this focus on verbal expressions of emotion, Ruesch (1948) proposed that an underdeveloped personality was a defining characteristic of individuals with psychosomatic illness. He argued that individuals who had not learned means of symbolic self-expression remained at an infantile level of development in which their somatic manifestations served as their primary means of self-expression. Bringing in the Freudian notion of psychic tension, Ruesch (1948) explained that whereas mature individuals expel excess tension through various means of expression, immature individuals remain caught with pent up tension that results in a prolonged state of readiness for action, i.e., the fight or flight response. This chronic level of arousal would then result in damage to bodily systems. Alexander (1950) also argued that repressed

emotional states lead to continued arousal of the central nervous system, although he did not invoke a developmental trajectory in his explanation. In both cases, however, the mechanism through which affect became translated into physical symptoms was thought to be the continued arousal that comes along with inhibited emotional expression.

Researchers have largely abandoned the initial notion that psychological symptoms are translated to physical symptoms through repressed traumas or unreleased tension according to the earlier psychodynamic theories, due to lack of empirical support. Some of this lack of evidence is inferred from findings that are inconsistent with the original psychodynamic theory. For instance, the high rate of psychiatric comorbidity (e.g., depression, anxiety) seen in individuals with somatization disorder (Katon et al., 1991) appears to be inconsistent with the notion of somatoform symptoms conferring a defensive mechanism against negative affect (Woolfolk & Allen, 2006). Other constructs within the psychodynamic model of hysteria, such as "la belle indifference" have been found explicitly lacking in empirical support (Stone, Smyth, Carson, Warlow, & Sharpe, 2006). Finally, the decline of the psychodynamic view of psychosomatic symptoms is due at least in part to the decreasing influence of psychoanalysis in general (Woolfolk & Allen, 2006).

Considerable empirical support has mounted for a hypothesis that emotional deficits play a key role in the etiology of somatization. Sifneos (1973) described the phenomenon he saw in many psychosomatic patients as "alexithymia," meaning literally to lack a vocabulary for emotion. Alexithymia was characterized by marked difficulty in verbally expressing or describing feelings, an absent or weak fantasy life, and an externally oriented mode of thinking (Nemiah & Sifneos, 1970). The basis for the

development of this term came from examining the assessment interviews of 20 individuals with disorders then considered psychosomatic (e.g., duodenal ulcer, asthma). Nemiah and Sifneos (1970) noted that many of these patients exhibited almost a complete unawareness of emotional states and an inability to put into words their emotions. In their responses, patients often focused on the retelling of minute details while leaving out any reflections on their internal thoughts, attitudes or feelings. The authors referred to the latter phenomenon as the "pensee operatoire" (Marty and de M'Uzan, 1963), meaning that their thought processes were unimaginative, lacking in internal reflection, and focused on mundane external details of events. Nemiah and Sifneos (1970) allowed for the possibility that alexithymia resulted from mechanisms other than repressed affect, such as deficient learned associations between emotions and verbal expression, and neuropsychological mechanisms. Although the underlying cause of this emotional deficit was unclear, these researchers argued that there was a strong connection between impairment in emotional awareness and expression and somatization.

The role of impairment of emotional experiencing in somatization is invoked in a more recent cognitive-developmental theory of emotional awareness developed by Lane and Schwartz (1987). In this theory, individuals with psychosomatic illness are believed to be caught at a level of emotional development in which the affective experience is characterized by undifferentiated somatic sensations. This contrasts with the more complex blending of emotions that individuals at higher levels of the scale naturally experience. Unpleasant negative emotional arousal, according to this theory, is experienced in terms of vague and powerful somatic distress.

Recently, this connection between emotional experiencing and somatization has been conceptualized in terms of a deficit in affect regulation (Taylor, Bagby, & Parker, 1997; Waller & Scheidt, 2006). Taylor, Bagby, and Parker (1997) defined affect regulation as a self-regulatory process involving the management of affective experiences consisting of reciprocal interactions between the physiological, cognitive, and behavioral elements of emotion response systems (Taylor et al., 1997). They proposed that an unawareness of one's emotional states would necessarily lead to ineffectiveness in expressing emotions and in the inability to regulate one's emotions through behavioral responses. For example, they argued that because of unawareness of one's emotions, a somatization disorder patient might overly focus on, magnify, and misinterpret the physical sensations that accompany his emotional arousal.

A substantial body of research now supports the notion that a lack of emotional awareness and expression in alexithymia is associated with psychosomatic illness, including somatization disorder (Waller & Scheidt, 2006). Waller and Scheidt (2006) conducted a comprehensive review on emotional processes in somatization and found substantial evidence to suggest that somatoform disorders involve deficits in the cognitive processing of emotion, including impaired awareness and expression of emotion. In addition, experimental research lends empirical support to the notion of alexithymia as a core deficit in the ability to recognize and process emotional cues (Lane et al., 1996; Lane, Sechrest, Riedel, Shapiro, & Kaszniak, 2000; Mann, Wise, Trinidad & Kohanski, 1994; Parker, Taylor & Bagby, 1993). This research has found poor emotion recognition in individuals high on alexithymia, suggesting that alexithymics may have

core deficits in their awareness of emotional cues. These findings have been demonstrated in both verbal and non-verbal tasks (Lane et al., 1996; Lane et al., 2000).

There is an especially significant body of research evidence supporting the link between deficits in alexithymia and somatization (De Gucht & Heiser, 2003; Bach & Bach, 1995). In a recent review article by De Gucht and Heiser (2003), the authors surveyed 16 articles examining alexithymia and medically unexplained symptoms and found a consistently small to moderate association between medically unexplained symptoms and alexithymia. Specifically, the authors of this study found a correlation coefficient of .23 between somatic symptom reporting and total Toronto Alexithymia Scale (TAS) scores, when taking into account all study samples combined and weighted by sample size. A larger coefficient of .35 was found for the TAS dimension corresponding to difficulty in identifying feelings (DIF) when analyzed separately from the two other domains, thereby demonstrating greater support for the DIF domain. This finding suggests that different domains of the TAS may function somewhat independently from one another. Moreover, domains garnering less support such as the domain of externally oriented thinking (EOT) may obscure a larger effect of the alexithymia construct. In addition, the studies included in the review were heterogeneous and included sample of students, medical populations, psychiatric populations, and mixed medical and psychiatric samples. When considering only studies in the review that compared somatoform disorder patients to non-somatizing controls, four out of the five of these studies yielded significant effects of alexithymia, with larger effect sizes ranging from .24 to .67. Thus, the inclusion of a heterogeneous sample of studies may have deflated the average effect size.

In the only prospective study examining the relation between alexithymia and somatization, Bach and Bach (1995) showed that patients who met criteria for undifferentiated somatoform disorder at 2-year follow-up had higher pretreatment TAS scores when compared with patients who never had met criteria for a somatoform disorder or who had entered remission. De Gucht and Heiser (2003) computed a moderately large effect size of .42 based on the statistics provided in Bach and Bach's (1995) study involving undifferentiated somatoform disorder patients. Moreover, Bach and Bach (1995) found that high alexithymia scores predicted a diagnosis of undifferentiated somatoform disorder at 2-year follow-up, independent of other measures of psychopathology and illness severity. However, no significant predictive effects of alexithymia were found for individuals with somatization disorder in the study. The number of individuals with somatization disorder in the study at baseline was extremely small (n=4) out of a sample size of 30, which probably contributed to this lack of significant findings with regard to somatization disorder in this study.

It has been suggested that somatosensory amplification, a bodily hypervigilance involving increased attention to and focus on unpleasant and relatively weak bodily sensations, and the tendency to appraise these sensations as abnormal signs of disease, may account for some of the association between alexithymia and somatization (Barsky, 1992). This construct, as measured by the Somatosensory Amplification Scale (SSAS; Barsky, 1992) has been theoretically linked with somatization in general (Barsky, 1992) and has been shown to be related to hypochondriacal symptoms in individuals with clinically diagnosed hypochondriasis (Barsky, Wyshak, & Klerman, 1990). This

construct may be important in understanding the cognitive processes as they function to facilitate somatization and hypochondriasis (Barsky, 1992).

In addition, there is some evidence that somatosensory amplification is associated with alexithymia (Nakao, Barsky, Kumano, & Kuboki, 2002; Wise & Mann, 1994). For example, Nakao and colleagues (2002) studied the association between somatosensory amplification and the domains of the TAS-20 in a psychosomatic clinic in Japan and found significantly elevated somatosensory amplification and alexithymia in the psychosomatic group with respect to the control group as well as significant correlations between the SSAS and the first two domains of the TAS-20. Wise and Mann (1994) also found significant correlations between the TAS and the SSAS in a heterogeneous group of psychiatric outpatients. However, this association has not been studied explicitly in a sample with somatization disorder. Because of the potential association between somatosensory amplification and alexithymia, it is important to control for somatosensory amplification when testing the associations between alexithymia and somatization.

Defensiveness and Repressive Coping

Defensiveness is a construct related to restricted emotional processing that may be associated with alexithymia and somatization. This construct is characterized by an inability to recognize aspects of the self that threaten safety and self-esteem, i.e., high defensiveness to negative aspects of the self. The Marlowe-Crowne Social Desirability Scale (MCS; Crowne & Marlowe, 1960) was constructed to measure the tendency to present oneself in an unrealistically positive light. Many studies therefore included the MCS as a means to control for socially desirable responding (Evans, 1982).

After being used in a multitude of studies in this manner (Evans, 1982), researchers began to examine the construct of defensiveness in its own right, rather than simply as a means to control for socially desirable responding. Researchers began to use the scale in more innovative ways, such as alongside other measures as a moderator of other personality features (Evans, 1982). Perhaps most prominently, the MCS was used alongside the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953) to distinguish highly defensive individuals reporting low anxiety from truly low anxious individuals (Weinberger, Schwartz, & Davidson, 1979). Weinberger et al. (1979) examined the physiological responses during a stressful task among truly low anxious subjects, who scored low on the MCS, repressors, who scored low on reported anxiety but high on defensiveness, and high anxious subjects. They found that the repressors exhibited high levels of somatic anxiety despite low levels of reported anxiety (Weinberger et al., 1979), thereby demonstrating a dissociation between their reported levels of anxiety and their experienced somatic anxiety. This dissociation has been replicated in other studies comparing groups based on their responses on the MCS and the TMAS (Asendorpf & Scherer, 1983; Newton & Contrada, 1992). In the current study, the MCS is used to measure defensiveness as opposed to social desirability.

The MCS and other measures of defensiveness, including the Balanced Inventory of Socially Desirable Responding (BISD; Paulhus, 1984) and the Lie Scale of the Minnesota Multiphasic Personality Inventory-2, continue to be used in tandem with measures of anxiety to examine how repressive copers respond both to self-report measures in other dimensions of functioning and in experimental situations (Burns, 2000; Burns, Kubilis, Bruehl and Harden, 2001; Newton & Contrada, 1992). The MCS is also

occasionally used alone as a measure of defensiveness and is examined in relation to reporting of physical symptoms, such as in chronic pain patients (Deshields, Tait, Gfeller, & Chibnall, 1995).

A significant number of research studies have examined the role of defensiveness specifically in medical populations and have found that defensiveness is associated with negative physical health consequences (e.g., Rutledge & Linden, 2000). Moreover, repressive coping and defensiveness may be especially pertinent in pain populations given the necessity of self-report in individuals' reports of pain and the potential for outside variables to influence that reporting. In fact, although it might seem that highly defensive individuals with pain disorders would deny pain and disability as well as anxiety or other emotional problems, it appears that this is not the case. There is evidence that defensiveness is significantly and positively associated with reports of low levels of subjective distress (Gick, McLeod & Hulihan, 1997) but high levels of pain and disability (Burns, 2000; Deshields, Tait, Gfeller, & Chibnall, 1995). Moreover, this association between defensiveness and pain levels appears to hold when controlling for depression (Deshields et al., 1995). In addition, there is now evidence that repressive copers may form a unique cluster of chronic pain patients distinguished by the way they cope with their pain conditions, which is distinct from the three clusters of dysfunctional, interpersonally distressed, and adaptive copers already identified by Turk and Rudy (1988; Burns, Kubilis, Bruehl and Harden, 2001). This repressive coper group was distinguished by significantly high levels of defensiveness along with the reports of high pain and disability but low levels of emotional problems, including depression and anxiety. The finding that the repressive coper group emerged out of the dysfunctional

group rather than the adaptive group lent support for the notion that defensiveness, as it relates to pain, may deflect against certain kinds of negative self-information, specifically emotional distress, as opposed to pain and disability.

The findings obtained from chronic pain samples may differ, however, from those obtained from samples with somatoform symptoms or functional somatic syndromes. Although there have been no studies examining defensiveness or repressive coping in individuals with full somatization disorder, there is some evidence to suggest that individuals with functional somatic syndromes disproportionately exhibit the relatively rare pattern of high defensiveness concurrent with high manifest anxiety (Brosschot and Aarsse, 2001; Creswell & Chalder, 2001). For instance, two recent studies demonstrated high rates of the high defensiveness-high anxiety profile in samples with fibromyalgia (FMS) and chronic fatigue syndrome (CFS), when compared with healthy controls and other chronically ill groups (Brosschot & Aarse, 2001; Creswell & Chalder, 2001). Brosschot and Aarse (2001) also found high rates of alexithymia in women with FMS. Considering these findings, the researchers proposed that somatizers may not be typical repressors but rather may be "oversocializers" (Brosschot & Aarse, 2001), thereby exhibiting rigid control over emotions in order to meet social standards, while also experiencing high levels of distress. In a cross-sectional study using a heterogeneous behavioral medicine clinic population, Gick, McLeod and Hulihan (1997) found no significant relationship between defensiveness, as measured by the MCS, and somatization, as measured by the somatization subscale of the Brief Symptom Inventory (BSI; Derogatis, 1975). These seemingly discrepant findings are probably due to the different population and measures used in the lattermost study or possibly due to lack of

construct validity in the defensiveness measure. Studies examining alexithymia and defensiveness in full somatization disorder would be needed to understand the operation of these constructs in somatoform populations.

Although defensiveness and repressive coping were initially thought to be strongly associated with alexithymia (e.g., Nemiah & Sifneos, 1970), studies examining the relationship between defensiveness and alexithymia have offered up a complicated picture of this relationship, with some suggesting an inverse relationship between defensiveness and alexithymia (Newton & Contrada, 1992) and others suggesting a more complex relationship (Myers, 1995). Newton and Contrada (1992) showed that low alexithymics displayed a typical repressive pattern of high physiological arousal and low reported distress during a stressful laboratory task, while alexithymics showed responses typical of high anxious subjects. Myers (1995) found that repressors scored significantly lower on alexithymia than did low anxiety/low defensiveness, high anxiety/low defensiveness, and high anxiety/high defensiveness groups. Moreover, high defensiveness/high anxiety subjects scored the highest on the Toronto Alexithymia Scale. Myers (1995) concluded that the combination of high defensiveness and low anxiety was responsible for the low alexithymia scores of the repressors rather than either defensiveness or anxiety alone. These findings are consistent with the profile of FMS patients obtained from the previous study by Brosschot and Aarse (2001), who scored high on defensiveness, anxiety as well as alexithymia and suggest a profile of somatizers who are high in alexithymia, high in defensiveness, and high in reported distress.

One major difference between alexithymia and repressive coping was initially thought to be the valence of the emotions that were excluded from awareness or

experience; whereas repressors might exclude only negative emotional or personal information from awareness, alexithymics might exclude both negative and positive emotions from their experiences. However, through experimental studies examining this question, Lane and colleagues have found that these deficits may differ more in magnitude than in quality (2000). Lane and colleagues (2000) examined the effects of alexithymia and repressive coping on the performance of community-dwelling individuals on emotion-recognition tasks in which subjects matched emotional descriptions in a variety of verbal and non-verbal methods (e.g., faces with sentences). They found that both alexithymics and repressors exhibited deficits in positive and negative emotion categories, going against the prevailing notion of repression as a defense against negative emotions. The main difference between the two groups appeared to be in magnitude of the deficit; the deficits of repressors were on a lesser scale, and were found in fewer emotion categories than the deficits of alexithymics. The authors suggested that alexithymia appears to be a more fundamental deficit while repressive coping may represent a smaller scale deficit in emotional functioning. Further empirical studies are needed to elucidate the nature of the difference between alexithymia and repressive coping.

Alexithymia, Repressive Coping and Treatment Outcome

Deficits in emotional functioning such as alexithymia and repressive coping may be important predictors of treatment outcome. Alexithymia was traditionally thought to pose a significant barrier to the success of insight-oriented therapy (Krystal, 1979). More recently, Porcelli and colleagues (2003) studied the predictive value of alexithymia in individuals with gastrointestinal functional somatic symptoms prospectively. In a study

assessing outcome following routine treatment for functional gastrointestinal disorders, Porcelli et al. (2003) found that baseline alexithymia scores predicted recovery status and reduction in gastrointestinal symptoms after controlling for baseline gastrointestinal symptoms, depression, and anxiety symptoms. Specifically, high levels of alexithymia were associated with worse recovery and more gastrointestinal symptoms at post-treatment. In addition, repressive coping has been found to predict poor response to psychotherapy for chronic pain (Burns, 2000). Previous studies have not examined how changes in these constructs may predict improvement in somatization symptoms, however.

There is some research evidence that psychotherapy targeting emotional awareness and expression may have a significant impact on alexithymia. Berenesvaite (2000) compared the effects of a 4-month long group psychotherapy program with a 2-session educational intervention program on TAS scores in 20 post-myocardial infarction patients with moderate or high baseline alexithymia (TAS $\geq$ 63). The psychotherapy program targeted emotional understanding and expression and relaxation techniques whereas the education intervention covered information regarding coronary heart disease. TAS scores were assessed at post-treatment and at 6-month, 1-year, and 2-year follow-up. Results showed a significant reduction in TAS scores in the psychotherapy group that was maintained over the 2-year follow-up, whereas no such reduction in TAS occurred in the group that received the educational intervention. Thus, alexithymia appears to be amenable to change through psychotherapeutic intervention, although no such research has yet been conducted in individuals with somatization disorder.

Yet the task of improving alexithymia may be especially important considering that impaired emotional functioning has been associated with negative consequences for both psychological and physical health. For example, inhibition of emotional material has been shown to be related to increased reports of pain (Cioffi & Holloway, 1993), increased somatic activation of the cardiovascular system (Gross & Levenson, 1997), and higher skin conductance (Pennebaker, Hughes, & O'Heeron, 1987). Suppression of thoughts in an attempt to regulate emotions has been associated with effects on the immune system (Petrie, Booth & Pennebaker, 1998) and on psychological distress in psychiatric patients (Lynch, Robins, Morse & Krause, 2001) and borderline personality patients (Cheavens et al., 2005; Rosenthal, Cheavens, Lejuez, & Lynch, 2005). Conversely, it appears that expression of emotionally meaningful material may be positively associated with both mental and physical health (Pennebaker & Seagal, 1999).

An Illustrative Example of the Experience of Emotion

Let us examine an instance of an experience of emotion in greater detail to understand how difficulty in the ability to recognize and express emotions may play a role in somatization. In a non-somatizing individual, a physiological experience of an emotion (e.g., stomach upset), leads to a cognitive process of recognizing and labeling the emotion (e.g., "I am feeling quite nervous right now"), and an appropriate behavioral response or action (e.g., engaging in calming self-talk). This behavioral response will lead to a reduction in experienced nervousness and is dependent on the adequate recognition and labeling of the emotional experience as nervousness.

An individual with a somatoform disorder who lacks the requisite awareness of his emotions, in contrast, might focus on the somatic aspect of this experience of fear and

label it in somatic terms rather than in emotional terms (e.g., “My stomach problems are really acting up”). This illustrates the process by which individuals with somatoform disorders may misattribute the physical aspects of emotional states as signs of physical illness and thus amplify the somatosensory experiences (e.g., physical sensations; Barsky, 1992). Similar to panic disorder patients whose arousal-inducing cognitions spawn further panic (Salkovskis & Clark, 1990), somatization patients may also experience continued physiological arousal as a result of their anxiety-provoking thoughts (Martin & Pihl, 1985).

Moreover, such a failure in the ability to identify and label one’s emotions will naturally lead to ineffective coping behaviors (Lane & Schwartz, 1987). For example, in contrast to the first individual who coped effectively with his emotion of nervousness, the somatizing individual in the previous example may take antacids and call the doctor to schedule an appointment for the stomach upset rather than engage in behaviors aimed at reducing the stress or nervousness he or she is experiencing. Thus, somatization disorder patients who exhibit difficulties in emotional awareness and expression may engage in illness behaviors such as seeking medical care for their symptoms or may avoid behaviors they feel may put them at risk for injury, such as intense exercise (Sharpe, Peveler, & Mayou, 1992). They may not reach out to a loved one when feeling sad or change their circumstances when feeling frustrated. Moreover, because of the lack of effective coping behaviors, these individuals’ negative emotional states continue unabated; their sadness, frustration, anger or anxiety go unnoticed and therefore persist.

While speculative, this emotional deficit hypothesis of psychosomatic illness may help explain the mechanism by which various somatic symptoms arise under the umbrella

of somatization disorder. More explicitly, somatization disorder is characterized not by a uniform set of symptoms but of a heterogeneous group of symptoms that may vary across individuals and within an individual over time. The multitude of possible somatic manifestations of emotions, such as elevated heart rate, impaired gastrointestinal functioning, and muscular tension, are reflected in the many possible somatic symptoms that somatizers experience. A person who lacks the ability to recognize and label his or her emotional states may focus on any one of these physical sensations, resulting in a variety of possible somatoform symptoms.

The Current Study

Despite the theoretically and empirically grounded link between impairment in emotional awareness and expression and somatization, there have been few treatments targeting emotional functioning in somatoform disorder patients. Because of the lack of empirical treatments addressing this concern, researchers have pushed for an increase in psychotherapy approaches that address emotional deficits in somatoform disorders populations (Waller & Scheidt, 2006). It is not known whether such a treatment would have a significant effect on emotional functioning in individuals with a somatoform illness like somatization disorder. Moreover, based on the association between alexithymia and somatization, improvements in the ability to identify and express emotions may lead to a reduction in somatization symptoms. Yet this hypothesis has not been tested.

The current study begins to address these questions. Specifically, this study asks whether participation in a randomized, wait-list controlled trial of cognitive behavioral treatment would have a significant effect on alexithymia in somatization disorder patients

and moreover, whether improvements in alexithymia over the course of treatment would predict improvement in somatization symptoms and functioning. This study also examines repressive coping by comparing groups according to their responses on the MCS and the short form of the Taylor Manifest Anxiety Scale (Bendig, 1956). The results of prior analyses, published in Allen and colleagues' (2006) study, suggest that participants in the 10-session CBT treatment condition experience improvements in objective and self-report measures of somatization symptoms and physical functioning when compared with a group whose physicians received a contact letter regarding somatization disorder. These positive findings demonstrate that the treatment was helpful in evoking positive change in the participants with somatization disorder treated with the CBT protocol, yet the mechanisms behind this positive change are not clear.

Recently, much attention has been given to the pursuit of examining and understanding mediators and moderators of treatments being tested in randomized controlled trials (RCTs; Kendall, 2006; Kraemer, Wilson, Fairburn, & Agras, 2002). Kendall (2006) has strongly encouraged the practice of conducting additional analyses on the data collected from RCTs toward the goal of understanding of the mechanisms of change in outcomes reported in clinical trials. Understanding how these treatments work and in whom they work is critical to the design of improved, targeted psychotherapies as well as to the understanding of the etiology and maintenance of clinical disorders (Kraemer et al., 2002). For example, understanding that catastrophic cognitions are central to the elimination of panic disorder in treatment lends support for the cognitive theory of panic (Kraemer et al., 2002; Salkovskis & Clark, 1990). Similarly, research evidence confirming the notion that emotional functioning predicts improvement in

somatization disorder would support a hypothesis of emotional functioning as an important factor in the development of somatization symptoms. Moreover, such positive findings might lead to the development of enhanced psychotherapeutic approaches targeting emotional functioning in this population.

There are two primary aims for the current study. The first aim of this study is to examine whether a psychotherapy treatment program consisting of cognitive, behavioral and affective components significantly affects alexithymia in somatization disorder patients. The second aim is to examine whether changes in alexithymia predict the improvement in somatization symptoms experienced as a result of participating in the treatment. I have three main hypotheses for the current study: 1) individuals in the treatment group will exhibit a significant decrease in alexithymia scores over the course of treatment whereas individuals in the waiting list will not undergo a change in alexithymia scores and this effect will be maintained over the 12-month follow-up period; 2) alexithymia scores will be lower in the treatment group than in the control group at post-treatment and 12-month follow-up; and 3) decreases in alexithymia through the course of treatment, as assessed through scores on the TAS-20, will to some degree predict improvement in somatization when controlling for baseline severity of somatization symptoms, mental health, defensiveness, and somatosensory amplification.

Method

Participants

Participants in the current study were men and women between the ages of 18 and 70 who met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria for somatization disorder. A total of 367 individuals were recruited from medical clinics and through advertisements in the community. In order to be included in the study, potential participants had speak English fluently and receive a Clinical Global Impression for Somatization Disorder (CGI-SD) score of 4 (moderate severity or higher). Patients with a psychotic disorder, organic brain syndrome, active suicidal ideation, unstable medical condition, or psychoactive substance dependence were excluded. In order to control for the effects of the psychotherapeutic treatment, individuals on medication that was not stabilized for the previous two months were excluded. For the same reason, individuals who had plans to engage in additional psychotherapy during the first 3 months after baseline were also excluded. Out of the 367 individuals who completed a telephone screening interview, 142 reported at least 6 possibly unexplained physical symptoms and agreed to participate in a face-to-face screening interview. Thirteen individuals declined to participate in the study, 45 were ineligible, leaving 84 individuals randomized to either treatment condition or the PCL. Out of the 45 individuals deemed ineligible for participation, 26 failed to meet DSM-IV criteria for somatization disorder, 10 reported unstable medication regimens, 6 were ineligible due to psychiatric comorbidity, 2 reported plans for pregnancy, and 1 reported a medical exclusion. In all, 41 individuals were assigned to PCL condition and 43 were assigned to the CBT condition.

Measures

Descriptions of the full battery of measures used in the CBT treatment study can be found in the appendix. Because we were specifically interested in examining the contribution of particular variables in the current study, only measures whose data were involved in the analyses conducted in the current study will be described in detail below.

CGI-SD

The primary outcome used in this study was the score on Clinical Global Impression for Somatization Disorder (CGI-SD). The CGI-SD consists of an independent evaluator's judgment of the severity of somatization as informed by a series of questions about the patient's frequency of, intensity of, and impairment caused by the 33 somatic symptoms that are assessed in assigning a DSM-IV diagnosis of somatization disorder. At baseline, the CGI-SD consisted of one item, a global severity item. At the other 3 assessment points, the CGI-SD consisted of two items, the global severity item and an improvement item. Severity was rated on a 7-point Likert-type scale ranging from 1 (no somatization) to 7 (very severe, among the most extreme cases of somatization), whereas improvement is rated on a 7-point scale ranging from 1 (very much improved) to 7 (very much worse).

Daily Symptom Diary

Daily symptom diaries have been widely used in research on functional somatic syndromes and have been shown to exhibit adequate psychometric properties (e.g., Allen et al., 2001; Payne & Blanchard, 1995). The daily symptom diary used in this study was a self-report measure in which patients recorded the maximum severity of somatoform symptoms experienced each day. Symptoms were rated from a 0 (no discomfort at all; no

impairment of functioning) to 5 (extremely severe discomfort; extreme impairment of functioning). Mean severity scores are computed from participants' ratings for the 7 consecutive days immediately prior to the evaluation sessions.

Physical Functioning

Physical functioning was assessed through the physical functioning scale of the Mental Health scale of the MOS 26-Item Short-Form Health Survey (SF-36; Ware & Sherbourne, 1992). This scale consists of 10 items assessing the ability to perform activities that vary in difficulty, including lifting or carrying groceries, climbing several flights of stairs, or walking more than a mile. This scale has consistently demonstrated high reliability and validity (Ware & Sherbourne, 1992).

Mental Health

To examine whether improvements in alexithymia are significant over and above improvements in psychological functioning, a variable assessing mental health was included in the analyses. Mental health was measured through the Mental Health scale of the SF-36 (Ware & Sherbourne, 1992). The Mental Health scale consists of 5 items assessing mood and nervousness, with low scores indicating nervousness and depression and higher scores indicating peacefulness, calm, and happiness. The mental health scale has been consistently been found to yield high ratings of internal reliability and validity and has been used extensively as a separate scale capable of assessing mental health (Berwick, 1991; Ware, Kosinski, & Keller, 1994; Ware & Sherbourne, 1992). Because the mental health scale does not incorporate somatic symptoms in assessing depression as in other depression scales, it is not expected to overlap with the assessment of somatization symptoms.

Alexithymia

The Toronto Alexithymia Scale-20 (TAS-20; Bagby, Parker, & Taylor, 1994) is a self-report measure that involves rating each of 20 items using a five-point Likert scale, yielding a maximum score of 100. The twenty items are divided into three subscales: 1) difficulty identifying feelings; 2) difficulty describing feelings; and 3) externally oriented thinking. The first subscale (DIF) consists of 7 items such as, "I am often confused about what emotion I am feeling." The second subscale (DDF) consists of 5 items such as, "It is difficult for me to find the right words for my feelings." The third subscale (EOT) consists of 8 items such as the following, "I prefer talking to people about their daily activities rather than their feelings." The TAS-20 has been used in a multitude of studies examining the alexithymia construct in somatization populations (see DeGucht & Heiser, 2003 for a review).

Defensiveness

In the current study, the Marlowe-Crowne Social Desirability Scale was included in analyses to control for defensiveness and for any possible confounding relationship between alexithymia and defensiveness. The Marlowe-Crowne Social Desirability Scale (MCS; Crowne & Marlowe, 1960) was used to measure defensiveness. This scale is a 33-item true/false scale.. Fifteen items keyed false are probably true but socially undesirable (e.g., "I am sometimes irritated by people who ask favors of me", and 18 items keyed true are improbably true but socially desirable (e.g., "No matter who I'm talking to, I'm a good listener.")

Somatosensory Amplification

The SSAS is a 10-item scale that assesses the construct of amplification of visceral and somatic sensations that has shown adequate reliability and validity in discriminating between hypochondriacs and normal controls (Barsky et al., 1990). This measure includes items such as the following: "Sudden loud noises really bother me" and "I can't stand smoke, smog, or pollutants in the air." Participants are instructed to indicate how characteristic each of the ten statements is of them, on a 5-point Likert scale, where higher scores signify higher levels of somatosensory amplification.

Procedure

The current study examines data from a randomized, controlled study of the efficacy of cognitive behavior therapy for somatization disorder conducted by Allen et al. (2006). Briefly, the study was a randomized, controlled treatment trial in which patients diagnosed as having SD were assigned to one of two treatment conditions: 1) a 10-session cognitive behavioral therapy (CBT) added to the psychiatric consultation letter (PCL; CBT + PCL) or 2) the PCL alone. The PCL was based on Smith et al. (1986). Individuals who were deemed possibly eligible for the study through a telephone screening participated in an in-person screening assessment in which somatization pathology and the presence of comorbid Axis I disorders were evaluated. Eligible participants returned for a baseline evaluation 1 to 2 weeks later. Patients were evaluated immediately after finishing the 10 week treatment (3 months post-baseline) and follow-up assessments were conducted at 6 and 12 months after completing treatment (9 and 15 months after baseline).

Treatment Conditions and Therapists. The CBT condition consisted of 10 one-hour individual therapy sessions following a manualized protocol with the primary aim of

assisting the patient with coping with stress and physical discomfort. The protocol focused on the following goals and activities: reduce physiological arousal through relaxation techniques, enhance activity regulation through increasing exercise, pleasurable activities, and meaningful activities; pace activities; increase awareness of emotions; modify dysfunctional beliefs; enhance communication of thoughts and emotions; reduce spousal reinforcement of illness behavior.

The PCL was a standardized letter sent to the principal treating physician of every study participant and stated that the participant met DSM-IV criteria for SD and made recommendations for the participant's ongoing medical treatment. The letter stated the following recommendations: schedule appointments with patients on a regular basis instead of as-needed; perform brief physical examinations on the area of discomfort at each visit; avoid unnecessary diagnostic procedures, invasive treatments, and hospitalizations; avoid explaining symptoms with statements such as "Your symptoms are all in your head."

Therapists were master's- or doctoral-level psychologists with several years of supervised training in CBT who had been trained in CBT for SD and who received weekly supervision during the study. Adherence and competence ratings were made throughout the study.

Analytic Plan

The analytic plan for this study consisted of four parts. First, I computed a correlation matrix among all predictor and outcome variables. Analyses were run on specific domains of the TAS in addition to the full scale. Next, I examined whether only the treatment group exhibited a significant change in alexithymia scores over the course

of therapy by comparing baseline TAS scores with TAS scores at post-treatment and at follow-up in the treatment and control groups. I also conducted a repeated measures mixed Analysis of Variance (ANOVA) comparing the treatment group with the PCL group over the three time points of pre-treatment, post-treatment and 12-month follow-up on measures of alexithymia, mental health, physical functioning, defensiveness, and somatosensory amplification. In addition, I conducted an ANOVA comparing the responders (rated as "much improved" or "very much improved" at post-treatment) with the nonresponders (receiving a rating any of 3, meaning "somewhat improved" or greater) on the following variables at post-treatment and follow-up: alexithymia, defensiveness, somatosensory amplification, mental health, physical functioning, and daily symptom diary scores.

Finally, I conducted a series of regression analyses, in order to determine which, if any**,** variables significantly mediate the association between treatment and outcome. These analyses were conducted in a similar manner to recent analyses on data obtained from randomized controlled trials of a psychotherapy (e.g., Cinciripini et al., 2003; Nock & Kazdin, 2005; Smits, Powers, Cho, & Telch, 2004). According to the Baron and Kenny (1986) model, mediation occurs when the following four conditions are met: a) the relation between treatment condition (independent variable) and improvement in CGI-SD (dependent variable) is significant; b) the hypothesized mediators (change in TAS full scale and subscale scores) are significantly related to treatment condition; c) the hypothesized mediators (change in TAS full scale and subscale scores) are significantly related to global improvement in CGI-SD; and d) the relation between treatment condition and CGI-SD is weakened statistically when controlling for the hypothesized

mediators. According to Baron and Kenny (1986), full mediation exists when the association between treatment and outcome is no longer significant after controlling for the mediator variable whereas partial mediation exists if this association is weakened, but still significant, after controlling for the mediator. In addition, the indirect effect of the predictor variable on the outcome variable via the mediating variable should be tested through the Sobel test (Sobel, 1982). The Sobel test assesses the extent to which the mediator accounts for the influence of an independent variable and is distributed as a Z-statistic. A statistically significant result demonstrates that the indirect effect of the predictor on the outcome works through the effects of the mediator on the outcome. Change scores for the TAS were computed by subtracting post-treatment TAS scores from TAS scores at baseline change scores**.** Additional change scores were computed by subtracting follow-up TAS scores from post-treatment scores and by subtracting follow-up TAS scores from baseline scores.

The following control variables were taken from post-treatment: defensiveness, somatosensory amplification, and mental health. I also controlled for baseline severity in the regression analyses. I also conducted similar sets of regression analyses, to determine whether changes in TAS scores predict improvement in somatization symptoms at 12-month follow-up, and in physical functioning and daily symptom diaries at post-treatment and follow-up, when controlling for the same variables. In addition to the main effects of treatment on TAS and CGI-SD, there may be interactive effects between the treatment condition and each mediating variable that may help explain differences in response to treatment (Kraemer et al., 2002). Thus, these interaction effects were tested in the regression analyses. It is expected that the effects of the predictor variables proposed in

this model will hold when controlling for demographic and other related variables. All analyses were conducted examining the three factors of the TAS separately, in addition to the total TAS-20 score.

It should be noted that although mediational analyses were initially intended to determine the causal relationships among factors (Baron and Kenny, 1986), in non-experimental study designs such as the one used in the current study, the regression analyses that are performed to determine mediator status cannot determine a causal relation between the treatment and the mediator or between the mediator and the outcome of interest. Kraemer et al. (2002) argued that demonstrating the causality of a mechanism of change in an RCT is more difficult than establishing mediator status and that further studies, such as component analyses examining the mechanisms of change through enhanced treatments, are needed to help illuminate more substantial relationships between treatment components and outcomes. Only experimental studies in which an independent variable is directly manipulated and all treatment groups begin the study as identical can determine if a particular variable is responsible for causing change in another variable.

Power Analysis

The following power calculation was based on a sample size of 84 individuals using six variables to be added into a regression analysis (treatment condition, alexithymia, mental health, baseline severity on the CGI, defensiveness, somatosensory amplification). The effect size used to calculate power was derived from a study by Porcelli et al. (2003) in which the authors measured the predictive value of TAS scores in predicting change in functional gastrointestinal symptoms over the course of treatment as

usual. They found that adding TAS scores to a regression equation after controlling for depression, anxiety and baseline symptoms resulted in an R^2 change of .19. Using an R^2 of .19, an effect size was found to be .2346, $\lambda = .2346 \times 84 = 19.7$. Considering a significance criterion (α) of .05, the power of this study is calculated at .91.[1] This power analysis assumes that collinearity between the six predictor variables is distributed normally. Thus, there is adequate power in the study's regression analysis to find a significant effect of alexithymia on the outcome of somatization symptoms.

[1] The power analysis described was conducted using the full sample size. Another power analysis was conducted using only the data from completers ($N = 77$). Using the sample size including only completers of post-treatment evaluation, six predictor variables and an alpha criterion of .05, the power of the study was calculated to be .88.

Results[2]

Baseline characteristics of study participants are shown in Table 1. Participants ranged in age from 22 to 65, with a mean age of 46.6 (*SD* = 9.79). More than half of the participants were employed, with most working either full-time (42.9%) or part-time (11.9%) or were retired (4.8%), although many were either unemployed (21.4%) or on paid disability (19%). The duration of somatoform symptoms ranged from 2 years to 55 years, with a mean of 25 years (*SD* = 13.32).

Table 2 shows means and standard deviations for all key predictor, mediating and outcome variables at baseline. Participants in the study overall revealed a high level of severity of somatization, with a mean CGI-SD of 5, corresponding with "marked somatization disorder," (*SD* = .83). At post-treatment, the global improvement ratings in the full sample ranged from 2 (much improved) to 5 (minimally worse), with a mean of 3.3 (minimally improved; *SD* = .98).

Completer Analysis and Missing Data

There were seven participants (8.3%) who dropped out of treatment prior to completing the post-treatment evaluation and 12 (14.3%) who did not complete the 12-month follow-up evaluation. The group of non-completers (at post-treatment) was compared with completers on all demographic and key predictor variables. Non-completers were equally likely to come from the CBT or the PCL condition ($p = .75$). There were no significant differences between completers and non-completers in age,

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[2] Because of the exploratory nature of the analyses in this study, the significance levels were not corrected using Bonferroni corrections. Therefore, some of the findings may be inflated due to the high number of analyses.

gender, marital status, work status, education level, baseline CGI-SD, or baseline mental health or physical functioning. The groups significantly differed on duration of symptoms, F (1, 82) = 6.76, p = .01, such that non-completers had an average shorter duration of symptoms (M = 12.86, SD = 11.61) than did completers (M = 26.08, SD = 12.98). There were no significant differences between these groups in baseline TAS full scale (p = .63) or domain scores (p = .62, .44, and .88) for the DIF, DDF, and EOT domain scores, respectively. Completers also did not differ from non-completers in defensiveness at baseline (p = .88). In addition, individuals who did not complete the follow-up assessment did not differ from those who did complete the follow-up on post-treatment CGI-SD (p = .07). There were non-significant trends toward completers at follow-up being older (M = 47.42, SD = 9.64) and having longer duration of symptoms (M = 26.08, SD = 13.32) than non-completers (M for age = 41.92, SD = 9.77, p = .07; M for duration = 18.33, SD = 11.73, p = .06) at follow-up. Analyses utilize data from completers only, unless noted as intent-to-treat analyses. In intent-to-treat analyses, missing data from these participants were entered into analyses through carrying their last completed data points forward. This strategy is consistent with an intention-to-treat approach, in which data from all randomized participants are examined from entrance into the study until the end of the study (Hollis & Campbell, 1999).

Correlations

Correlation coefficients were calculated among treatment condition and demographic variables to check for adequate randomization of demographic

characteristics in treatment conditions.[3] There were no significant correlations between group condition and the following variables: age, race, education, marital status, duration of somatoform symptoms, or CGI-SD at baseline. Correlation coefficients were also calculated between demographic variables and the outcome variable, i.e., global improvement in somatization symptoms on CGI-SD, as assessed at post-treatment. There was no significant correlation between improvement in somatization symptoms and gender, race, marital status, work status, education level, or duration of symptoms.

Table 3 shows a correlation matrix between predictor and control variables at baseline and at post-treatment. Although the DIF and DDF domains were highly correlated with one another, the EOT domain had the smallest correlations with other domains and with the full scale score. Baseline alexithymia scores appeared highly stable across the length of the study, as indicated by high correlations between scores at baseline and follow-up (for TAS-20, $r = .76$, $p < .0001$; for DIF domain, $r = .70$, $p < .0001$; for DDF domain, $r = 72$, $p < .0001$, for EOT domain, $r = .80$, $p < .0001$). The MCS correlated significantly with the DIF and DDF domains of the TAS, but not with the full scale or the EOT. Defensiveness was negatively correlated with the DIF and DDF domains of the TAS. Mental Health was significantly negatively correlated with the total TAS score ($r = -.32$, $p < .01$) and with all three domains of the TAS-20 at baseline (for DIF, $r = -.27$, $p < .05$; for DDF, $r = -.28$, $p < .05$; for EOT, $r = -.22$, $p < .05$) such that higher alexithymia scores were associated with worse mental health. However, TAS scores tended to be positively correlated with reports of physical functioning. Defensiveness was significantly positively correlated with mental health at baseline but

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[3] Correlation coefficients were calculated using only complete data. Missing data were excluded from these

not at post-treatment, such that higher defensiveness was associated with greater mental health. Baseline severity of somatization symptoms, assessed through the CGI-SD, did not correlate significantly with global improvement in symptoms at post-treatment. The DDF domain was significantly correlated with somatosensory amplification at baseline but not at post-treatment, and defensiveness was significantly correlated with the DIF and DDF domains at baseline but not at post-treatment.

As shown in Table 5, there were significant correlations between improvement in somatization symptoms and TAS change scores on the TAS full scale, the DIF domain and the EOT domain. No significant correlations were found between improvement in somatization symptoms and change on the TAS DDF domain or on post-treatment MCS or SSAS scores. Lastly, there were no significant correlations between improvement in somatization symptoms and TAS scores at post-treatment.

To examine whether demographic variables were associated with change in alexithymia, correlation coefficients were calculated between TAS change scores and demographic variables. There were significant correlations between work status and changes on the TAS full scale score ($r = .26$, $p = .02$), and on the DIF ($r = .23$, $p = .04$) and DES ($r = .32$, $p = .003$) domains, such that working was associated with a greater decrease in alexithymia scores from baseline to post-treatment. There was also a significant correlation between change on the EOT domain and duration of symptoms ($r = -.31$, $p = .005$), such that a longer duration of symptoms was associated with a decrease in externally oriented thinking from baseline to post-treatment. No other correlations were significant.

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analyses.

Analyses Comparing CBT vs. PCL Groups

One-way analysis of variance was conducted to examine the relationship between treatment condition and global improvement in somatization symptoms from baseline to post-treatment using the Clinician's Global Impression Scale as the primary outcome measure. The ANOVA's were significant, F (1, 82) = 36.06, p <.0001. Participants in the CBT group experienced significantly greater improvement in somatization symptoms from baseline to post-treatment, as evidenced by lower scores on the Global Improvement measure (M = 2.69, SD = .73), than did participants in the PCL group (M = 3.74, SD = .79). The groups remained significantly different on improvement scores at 12-month follow-up, F (1, 82) = 27.85, p < .0001.

Repeated measures analyses of variance were used to compare treatment conditions on secondary outcome measures and other predictor variables. For the outcome of diary scores, participants in the CBT condition showed significantly greater decreases in diary scores over the course of the study than those in the PCL condition, as shown by a significant interaction between time and condition, F (2, 69) = 5.52, p = .005. Participants in the CBT group had significantly lower diary scores at post-treatment (M = 2.72, SD = 1.15) and at 12-month follow-up (M = 2.50, SD = 1.15) than did those in the PCL condition (M = 3.26, SD = .87 for post-treatment; M = 3.21, SD = 1.05 for follow-up, when controlling for baseline diary scores. Repeated measures analyses of variance were conducted to compare treatment conditions across time on the following variables: defensiveness, somatosensory amplification, mental health, and physical functioning. There was a significant interaction between time and condition for physical functioning, F (2, 69) = 5.39, p = .007, such that participants in the CBT condition improved

significantly more on physical functioning from baseline to follow-up. There were no significant interactions between time and condition for defensiveness, somatosensory amplification or mental health from baseline to 12-month follow-up, indicating that CBT participants did not change significantly more from baseline to follow-up on those measures.

Analyses Comparing Participants Scoring High and Low on the TAS-20

Two groups were coded based on pre-established cut-off scores on the TAS-20 (Taylor et al., 1997). Participants scoring 61 or above on the TAS-20 were considered alexithymic whereas those scoring 51 or below were considered non-alexithymic (Taylor et al., 1997). Twenty participants scored in the alexithymic range while 49 scored in the non-alexithymic range. Twenty-four percent of the participants scored in the alexithymic range at baseline, while 58% of the participants scored in the non-alexithymic range and 18% scored in the middle range for alexithymia and were excluded from the analyses. Alexithymics were then compared with non-alexithymics on baseline characteristics and improvement on somatization symptoms. Alexithymics did not differ significantly from non-alexithymics on age, gender, race, work status, marital status, or duration of symptoms. There was a significant difference between groups on baseline mental health, F (1, 67) = 9.74, p = .003, with non-alexithymics scoring significantly higher on mental health (M = 62.53, SD = 18.65) than alexithymics (M = 48.00, SD = 14.39). At post-treatment and follow-up, this difference was no longer significant. Alexithymics scored significantly higher on manifest anxiety at baseline (M = 13.5, SD = 4.31) and at follow-up (M = 10.35, SD = 5.93) than non-alexithymics (M = 8.41, SD = 5.24; M = 6.91, SD = 5.13; F (1, 67) = 14. 73, p = .0003 for baseline; F (1, 59) = 5.07, p = .02, for follow-up.

The groups did not differ on baseline CGI-SD or on improvement in CGI-SD at post-treatment when controlling for baseline CGI-SD. Although the groups did not differ significantly on physical functioning at baseline, at post-treatment the groups differed significantly when controlling for baseline physical functioning, with alexithymics reporting greater physical functioning (M = 74.72, SD = 23.73) than non-alexithymics (M = 55.00, SD = 26.31; F (1, 62) = 10.29, $p < .002$. Alexithymics scored significantly lower on defensiveness at baseline (M = 15.50, SD = 4.88) than non-alexithymics (M = 18.82, SD = 6.67), F (1, 67) = 4.04, $p < .05$. Alexithymics did not differ significantly from non-alexithymics in somatosensory amplification.

Change in Alexithymia

Table 4 shows the means and standard deviations for TAS full scale and domain change scores from baseline to post-treatment. A repeated measures analysis of variance comparing treatment groups across all three time points (baseline, post-treatment and 12-month follow-up) yielded a significant effect for the interaction between time and condition on TAS full scale scores, F (2, 68) = 3.36 , p = .04 and a marginally significant main effect of time F (2, 68) = 3.20, p = .05 (see Figure 1). In the repeated measures analysis of variance comparing treatment groups across all three time points on DIF domain scores of the TAS, there was a significant time by condition interaction, F (2, 68) = 4.23, p = .02 but no main effect for time, signifying that participants in the CBT group changed significantly more on the DIF domain over time whereas participants in the PCL group stayed relatively the same[4] (see Figure 2). There was no significant time by

[4] The sphericity assumption was violated for this analysis. Therefore, results for the multivariate analyses are presented here rather than the univariate tests which are not appropriate when the assumption of sphericity is violated.

condition interaction for change in the DDF domain of the TAS. There was a marginally significant time by condition interaction in change in EOT scores, F (2, 138) = 3.04, p = .05 but no main effect for time in change in EOT scores (see Figure 3).

Separate analyses of variance were conducted to evaluate between-groups differences on change scores at two time points. Significant differences were found between treatment groups in change from baseline to post-treatment in TAS full scale score, F (1, 75) = 5.59, p = .02, in the DIF domain of the TAS, F (1, 75) = 6.19, p = .02, and in change in the EOT domain of the TAS, F (1, 75) = 4.73, p = .03.

Treatment conditions were also compared on their post-treatment scores on the TAS full scale and domain scores. There was a significant effect for treatment condition on the EOT domain of the TAS, F (1, 75) = 4.62, p = .03, such that CBT participants (M = 16.28, SD = 4.19) had lower EOT scores at the end of treatment than PCL participants (M = 18.26, SD = 3.89). This effect remained significant when controlling for baseline EOT. There were no other significant differences between treatment groups on TAS scores at post-treatment. There were also no significant differences between treatment groups on TAS scores at 12-month follow-up.

An additional change score was calculated by subtracting post-treatment scores on the TAS full scale and domains from 12-month follow-up scores. Treatment groups were then compared on these change scores using one-way ANOVA's. There were no significant differences between treatment groups on change in the TAS full scale score or on any TAS domains from post-treatment to 12-month follow-up.

Finally, a change score was calculated by subtracting baseline scores on the TAS full scale and domains from 12-month follow-up scores. Treatment groups were then

compared on these change scores using one-way ANOVA's. There was a significant difference between treatment conditions for change on the DIF domain of the TAS from baseline to follow-up, F (1, 69) = 5.41, p = .02. There were no significant differences between groups on change between baseline and follow-up on full scale TAS scores or change in the DDF or EOT domains of the TAS.

Correlations were calculated among TAS change scores and outcome measures at post-treatment and 12-month follow-up (see Table 5). Overall, change in alexithymia on the full TAS, and the DIF and DDF domains, correlated significantly with improvement in somatization symptoms on the CGI-SD and with physical functioning on the MOS at post-treatment and slightly less consistently at 12-month follow-up. The full scale and the DIF domain were more strongly correlated with outcomes than the other domains. Change in the EOT was significantly correlated with improvement in the CGI-SD at post-treatment but not at follow-up. Correlations among TAS change scores and mental health at post-treatment and at 12-month follow-up were not significant.

Intent-to-Treat Analyses

In these analyses, missing data were imputed using a last-observation-carried-forward approach. A one-way analysis of variance was conducted to examine the relationship between treatment condition and global improvement in somatization symptoms from baseline to post-treatment using the Clinician's Global Impression Scale as the outcome measure. The ANOVA was significant, F (1, 82) = 30.51, p <.0001. Participants in the CBT group experienced significantly greater improvement in somatization symptoms from baseline to post-treatment, as evidenced by lower scores on the Global Improvement measure (M = 2.81, SD = .79), than did participants in the PCL

group (M = 3.76, SD = .77). Participants in the CBT condition also had significantly greater improvement ratings than participants in the PCL condition at 12 month follow-up, F (1, 82) = 27.85, p < .0001. Individuals in the CBT group received an average rating between minimally improved and much improved (M = 2.79, SD = .89) whereas participants in the PCL group generally stayed the same (M = 3.80, SD = .87).

Univariate analyses of variance were conducted using all data from all randomized participants on changes in alexithymia over the course of treatment by treatment condition. Baseline data were imputed for participants missing the post-treatment assessment in these analyses. Significant differences were found between treatment groups in change in TAS full scale score, F (1, 82) = 5.50, p =.02, in the DIF domain of the TAS, F (1, 82) = 5.93, p = .02, and in the EOT domain of the TAS, F (1, 82) = 4.78, p = .03. Treatment conditions did not differ on change scores in the DDF domain of the TAS from baseline to post-treatment.

Treatment conditions were also compared on their post-treatment scores on the TAS full scale and domain scores. There was a marginally significant effect for treatment condition on the EOT domain of the TAS, F (1, 82) = 3.67, p = .06, such that CBT participants (M = 16.42, SD = 4.17) had lower EOT scores at the end of treatment than PCL participants (M = 18.17, SD = 4.21). There were no other significant differences between groups on TAS scores at post-treatment. There were also no significant differences between treatment groups on TAS scores at 12-month follow-up.

An additional change score was calculated by subtracting post-treatment scores on the TAS full scale and domains from 12-month follow-up scores. This change score demonstrates change in alexithymia experienced from the end of treatment until the 12-

month follow-up assessment. Treatment groups were then compared on these change scores using one-way ANOVA's. An ANOVA was not significant for the full scale score or any domain except for the EOT domain, which was significant, F (1, 81) = 4.08, p < .05. Participants in the control group decreased more on externally oriented thinking from post-treatment to follow-up (M = -.73, SD = 2.63) than did participants in the experimental condition, who experienced a slight increase in externally oriented thinking (M = .52, SD = 3.01).

Responder Analyses

Separate analyses of variance were conducted to evaluate the relationship between response to treatment and demographic and predictor variables. As defined by a global improvement rating of 1 or 2 ("much improved" or "very much improved"), there were a total of 20 responders and 57 non-responders.[5] No significant differences were found between responders and non-responders on any demographic variables, including gender, race, work status, educational level, marital status or duration of symptoms. Responders were significantly younger than non-responders, F (1, 75) = 12.40, p < .001, with a mean of 40.85 years (SD = 8.36) as opposed to a mean of 49.12 years (SD = 9.26) in the non-responder group. A significant difference was found between responders and non-responders on their baseline severity on the CGI-SD, F (1, 75) = 4.28, p = .04, such that responders (M = 5.30, SD = .92) had a slightly higher severity at baseline than did non-responders (M = 4.88, SD = .73). Responders did not differ from non-responders in baseline mental or physical functioning. An ANOVA comparing responders with non-

[5] Because these analyses were concerned with the status of improvement of individuals who completed the study, these analyses were conducted using only the complete sample of 77 individuals at post-treatment and of 72 individuals at follow-up.

responders on the three TAS change scores, from baseline to post-treatment, from post-treatment to follow-up, and from baseline to follow-up, yielded non-significant findings for total TAS score and for all domains of the TAS. In addition, there were no significant differences between responders and non-responders on their scores on all of the following predictor variables, taken at post-treatment: TAS total score, all domains of the TAS, defensiveness, SSAS, MOS-EF, or MOS-PF. Responders had significantly lower daily symptom diary scores at post-treatment (M = 2.31, SD = 1.09) than non-responders (M = 3.22, SD = .93; F (1, 75) = 12.98, p = .0006,).

Responders and non-responders were also compared based on their responses on all measures at 12-month follow-up. At follow-up, there were 17 responders and 55 non-responders. Five individuals who were non-responders at post-treatment became responders at follow-up, while six individuals who were responders at post-treatment became non-responders at follow-up. Being a responder at post-treatment was highly correlated with being a responder at follow-up (r = .59, p < .0001). A one-way ANOVA comparing responders and non-responders on their scores on the Somatosensory Amplification Scale (SSAS) was significant, such that responders had significantly more amplification at 12-month follow-up (N = 16, M = 34.69, SD = 6.65) than non-responders (N = 51, M = 28.78, SD = 6.97), as indicated by higher scores on the scale, F (1, 65) = 8.93, p = .004. There were no significant differences between responders and non-responders on their follow-up scores on the total TAS, TAS domains of DIF, DDF or EOT, MOS-EF, MOS-PF, MCS, or daily symptom diary scores.

As reported previously (Allen et al., 2006), treatment condition was a significant predictor of responder status at post-treatment (OR = 9.02, 95% CI, 2.37-34.37, p = .001)

and at 12-month follow-up (OR = 13.12, 95% CI, 2.72-63.36, $p = .001$) using logistic regression. There were no significant effects of change in TAS in the full scale or any domains or in group based on defensiveness and anxiety scores when entered into a regression analysis with treatment condition. Change in defensiveness from baseline to post-treatment also did not predict responder status. Somatosensory amplification (SSAS) scores at follow-up significantly predicted responder status at follow-up (OR = 1.10, 95% CI, 1.01-1.22, $p = .03$).

Analyses Based on Extreme MCS and MAS Scores

Based on previous findings, a score greater than or equal to 19 on the MCS has been considered indicative of high defensiveness, whereas a score below or equal to 11 has been considered indicative of low defensiveness (Weinberger et al., 1979; Newton & Contrada, 1992). In this study, quartile ranges were used to distinguish high anxiety from low anxiety participants. The upper quartile on the TMAS was characterized by a score of 14 or higher and the lower quartile was characterized by a score of 6 or lower on the short form of the TMAS. Using these criteria, 36 individuals were classified into four groups: high defensiveness, low anxiety (repressors; $n = 16$); high defensiveness, high anxiety ($n = 11$); low defensiveness, high anxiety ($n = 6$); and low defensiveness, low anxiety ($n = 3$). Forty-eight participants were not classified into these groups because their scores on the MCS and the TMAS fell into the middle range for participants.

Analyses of variance were conducted to examine whether there were any differences among the groups in demographic characteristics. A chi-square test was marginally significant for marital status, $x^2 = 7.7$, $df = 3$, $p = .05$, with high defensiveness/high anxiety participants exhibiting the highest percentage of married

participants. There were no significant differences among groups in terms of age, gender, work status, level of education, duration of symptoms or baseline severity on the CGI-SD.

Table 6 shows mean scores for each of the groups distinguished by extreme anxiety and defensiveness scores on measures which revealed significant between-groups differences using analysis of variance, on baseline CGI-SD, and on improvement in CGI-SD at post-treatment and 12-month follow-up. Significant pairwise differences were analyzed using Tukey's post-hoc tests. No significant differences were found between these four groups when compared on baseline symptom severity on the Clinician's Global Impression rating scale, on global improvement ratings at post-treatment and 12-month follow-up, or on responder status. Groups also did not differ significantly on daily symptom diary scores at any of the three time points. An ANOVA for baseline total TAS score was significant, F (3, 32) = 4.13, p = .01, with one significant difference between repressors and high defensiveness/high anxiety groups such that high defensiveness/high anxiety participants had significantly higher alexithymia scores than repressors. An ANOVA for baseline DIF domain of the TAS was even more highly significant, F (3, 32) = 7.22, $p < .001$ such that high anxiety participants who scored either high or low on defensiveness, scored significantly higher on alexithymia than repressors. There were no significant differences among the groups on the DDF or EOT domains of the TAS. An ANOVA was significant for mental health, F (3, 32) = 24.13, $p < .0001$, such that repressors reported significantly greater mental health than both groups that were high in anxiety (high defensiveness and low defensiveness), and that the low defensiveness/low anxiety group had greater mental health at baseline than the low defensiveness/high

anxiety group. No significant differences were found among groups based on their TAS change scores. There were no differences among groups in baseline scores on physical functioning or somatosensory amplification.

Similar analyses were conducted using post-treatment and follow-up scores on the TAS.[6] An ANOVA was significant at post-treatment for TAS full scale score, F (3, 32) = 6.04, p = .002, with both high defensiveness/high anxiety participants and low defensiveness/high anxiety participants scoring higher on alexithymia than repressors. At follow-up, there was only one significant difference between high defensiveness/high anxiety individuals and repressors, with the former group scoring more highly on alexithymia than the latter group, F (3, 31) = 3.36, p = .03. An ANOVA for post-treatment scores on the DIF domain of the TAS was also significant, F (3, 32) = 9.26, p < .0001, such that again, repressors scored lower on alexithymia than both high defensiveness/high anxiety participants and low defensiveness/high anxiety participants. At follow-up, there was only a significant difference between high defensiveness/high anxiety individuals and repressors, F (3, 31) = 5.50, p = .004. Lastly, an ANOVA for the DDF domain was significant at post-treatment, F (3, 32) = 3.71, p = .02, with repressors scoring lower in this domain of alexithymia than low defensiveness/high anxiety participants. This difference was no longer significant at follow-up. There were no significant differences among groups on the EOT domain at post-treatment. There were

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[6] Analyses presented here use data from all randomized participants. Missing data were imputed using the last-observation-carried-forward approach. Analyses were also conducted using only completers at post-treatment. Analyses yielded similar results for between-groups tests of post-treatment TAS full scale score, DIF domain, and MOS-EF scores with the exception of the analyses of the DDF domain of the TAS, which no longer significantly differentiated among repressiveness groups at post-treatment. Analyses conducted using only completers at 12-month follow-up found no significant differences among repressiveness groups in full scale TAS score but similar findings in comparing the DIF domain scores and scores on the MOS-EF.

significant differences among groups in mental health at post-treatment, F (3, 32) = 7.52, $p < .001$, and at follow-up, F (3, 32) = 5.87, $p = .003$, with repressors reporting better mental health at post-treatment than both groups distinguished by high TMAS scores (high defensiveness/high anxiety and low defensiveness/high anxiety).

Change in Defensiveness

In order to examine whether defensiveness was amenable to change and whether it distinguished among the repressiveness or treatment groups, change in defensiveness was computed by subtracting baseline scores on the MCS from the post-treatment score. The four groups were then compared on this variable. An ANOVA was significant, F (3, 32) = 4.48, $p < .01$, such that did high defensiveness/high anxiety individuals experienced a significantly greater decline in defensiveness from baseline to post-treatment than low defensiveness/low anxiety individuals. In a repeated measures analysis of variance, there was no significant effect of time or of the interaction between time and condition on change in defensiveness over the three time points of baseline, post-treatment and follow-up.

Mediational Analyses

Post-Treatment Improvement in Somatization Symptoms as Outcome Variable. The results of the mediational analyses for the outcome of improvement in somatization symptoms at post-treatment with full scale TAS scores as mediator are shown in Table 7a. The first step of the mediational analysis was significant; treatment condition significantly predicted improvement in somatization symptoms at post-treatment, F (1, 75) = 36.06, ß = -.57, $p < .0001$, such that being in the treatment condition predicted greater improvement in symptoms (a lower rating on the global improvement scale). In

step 2, change in TAS scores on the full scale and each domain were regressed separately onto treatment condition to determine which, if any, of these variables would be predicted by treatment condition. Treatment condition significantly predicted change in the TAS full scale score, F (1, 75) = 5.59, ß = -.26, p = .02, in the DIF domain of the TAS, F (1, 75) = 6.19, ß = -.28, p = .02, and in the EOT domain of the TAS, F (1, 75) = 4.73, ß = -.24, p = .03. Change in the DDF domain of the TAS was not significantly predicted by treatment condition and cannot therefore not serve as a mediator of improvement in somatization symptoms.

In step 3, improvement in somatization symptoms at post-treatment was regressed onto the change scores for the TAS full scale and each domain of the TAS separately to examine which, if any, of these variables would predict global improvement ratings. Improvement in somatization symptoms at post-treatment was significantly predicted by change in TAS full scale score, F (1, 75) = 9.25, ß = .33, p = .003, change in TAS DIF domain score, F (1, 75) = 5.61, ß = .26, p = .02, and change in TAS EOT domain score, F (1, 75) = 9.68, ß = .34, p = .003.

In step 4, when entered into the regression equation with treatment condition, change in TAS full scale score significantly predicted improvement at post-treatment, F (2, 74) =20.81, ß = .19, $p < .05$, and weakened the effect of treatment condition, although not to a non-significant level. Treatment condition alone accounted for 32% of the variance in improvement in somatization symptoms at post-treatment while adding change in TAS full scale score accounted for an additional 4% of the variance. In the final model, change in TAS full scale score no longer significantly predicted improvement in somatization symptoms when accounting for post-treatment mental

health scores, baseline severity of somatization symptoms, and defensiveness at post-treatment, and somatosensory amplification. In the final model, somatosensory amplification approached significance levels. A Sobel test was performed using the final regression model and was not significant (z = -1.29, p = .20). Mental health was not a significant predictor of improvement at post-treatment. When entered alone into the regression equation with treatment condition, none of the individual domains of the TAS significantly predicted global improvement at post-treatment and the full regression models are therefore not presented for those variables.

Interaction Effects. Entered alone in a regression model, the interaction between condition and change in full scale TAS score was significant in predicting improvement in somatization symptoms at post-treatment, F (1, 75) = 5.48, p = .02. The interaction alone accounted for 7% of the variance in improvement in somatization symptoms at post-treatment. This effect dropped below significance levels when added to the equation with condition and treatment separately along with it (p = .32), and was therefore not included in the final regression model. The interaction between condition and change in DIF domain of the TAS was not statistically significant when entered alone into the regression model, p = .08. The interaction between condition and change on the EOT domain of the TAS was significant when entered alone into a regression model predicting improvement at post-treatment, F (1, 75) = 8.38, p = .005, accounting for 10% of the improvement in somatization symptoms at post-treatment. This interaction was no longer significant when added to a model with condition and change in EOT domain separately (p = .89) and was therefore eliminated from the final model. The interaction between treatment condition and change in DDF domain of the TAS was not significant.

12-Month Follow-up Improvement as Outcome Variable. The first step of the regression analysis was significant; treatment condition significantly predicted improvement in somatization symptoms at 12-month follow-up, F (1, 70) = 28.89, ß = -.54, $p < .0001$, such that being in the treatment condition was a predictor of greater improvement in symptoms. Step 2, in which change in TAS scores on the full scale and each domain were regressed separately onto treatment condition, was identical to step 2 in the previous set of regression analyses. In step 3, improvement in somatization symptoms at 12-month follow-up was regressed onto change scores of the TAS full scale and each domain of the TAS separately. Improvement in somatization symptoms at 12-month follow-up was significantly predicted by change in TAS full scale score, F (1, 70) = 9.06, ß = .34, p = .004, in the TAS DIF domain score, F (1, 70) = 8.21, ß = .34, p = .006, and in the DDF domain score, F (1, 70) = 5.33, ß = .27, p = .02. Change in the TAS EOT domain score was not a significant predictor of improvement ratings at 12-month follow-up. In step 4, when added to a regression equation with treatment condition alone, change in TAS full scale score was no longer significant, ß = .20, p = .06. Change in the DIF also lost significance when added to a regression equation with treatment condition in predicting improvement at follow-up (p = .12). Sobel tests were not conducted because change in alexithymia was not significant when entered with condition alone.

Although treatment condition did not significantly predict change in the DDF, disabling it from being a mediator of improvement in somatization symptoms, change in this domain of the TAS did, however, significantly predict improvement in somatization symptoms at 12-month follow-up, even when controlling for baseline severity, mental health at post-treatment, and defensiveness at post-treatment, ß = .23, p = .03. When

somatosensory amplification was added to this equation, however, change in this domain no longer significantly predicted improvement at follow-up, ß = .20, $p = .08$ for DDF. When entered with treatment condition alone predicting improvement ratings at 12-month follow-up, condition accounted for 29% of the variance while change in the DDF domain of the TAS accounted for an additional 6% of the variance. None of the control variables significantly predicted improvement at follow-up in the final regression equation.

Interaction Effects. Entered alone in a regression model, the interaction between condition and change in full scale TAS score was significant in predicting improvement in somatization symptoms at 12-month follow-up, F (1, 70) = 6.09, $p = .02$. The interaction alone accounted for 8% of the variance in improvement in somatization symptoms at 12-month follow-up. This effect dropped below significance levels when added to the equation with condition and treatment separately along with it ($p = .47$), and was therefore eliminated from the final model. The interactions between condition and change in all domains of the TAS were not significant predictors of improvement in somatization symptoms at follow-up.

Physical Functioning as Outcome Variable. Tables 7b and 7c presents the regression data from hierarchical regression analyses conducted in examining mediational status of change in alexithymia in the relationship between treatment condition and physical functioning at post-treatment. The first step of the regression analysis was significant; treatment condition significantly predicted physical functioning at post-treatment when controlling for baseline physical functioning, such that being in the treatment condition was a predictor of greater physical functioning. Step 2, in which

change in TAS scores on the full scale and each domain were regressed separately onto treatment condition, was identical to step 2 in the previous set of regression analyses. In step 3, physical functioning at post-treatment was regressed onto change scores of the TAS full scale and each domain of the TAS separately. Physical functioning at post-treatment was significantly predicted by change in TAS full scale score and change in the TAS DIF domain score. As shown in these tables, change on both the TAS full scale and on the DIF domain of the TAS significantly predicted physical functioning at post-treatment when controlling for baseline physical functioning, post-treatment defensiveness, post-treatment mental health and post-treatment somatosensory amplification. However, the mediational status of both the TAS full scale and the DIF domain were not significant according to Sobel tests ($z = 1.40$, $p = .16$, and $z = 1.41$, $p = .16$, respectively). Because change in the DDF domain of the TAS was not significantly predicted by treatment condition, this variable did not meet criteria for a mediating variable. In addition, although treatment condition significantly predicted change on the EOT domain of the TAS, change on the EOT domain of the TAS did not significantly predict physical functioning at post-treatment and thus failed to meet criteria for a mediating variable.

12-Month Follow-up Physical Functioning as Outcome Variable. The first step of the regression analysis was significant; treatment condition significantly predicted improvement in physical functioning at 12-month follow-up when controlling for baseline physical functioning, F (2, 69) = 48.92, ß = .20, $p = .01$, such that being in the treatment condition was a predictor of greater physical functioning at follow-up. Step 2 was conducted in previous analyses. In step 3, physical functioning at 12-month follow-

up was regressed onto change scores of the TAS full scale and each domain of the TAS separately. When controlling for baseline physical functioning, physical functioning at 12-month follow-up was significantly predicted by change in TAS full scale score, F (2, 69) = 50.84, ß = -.23, p = .01 and in the DIF domain score, F (2, 69) = 52.79, ß = -.25, p = .002. Change in the TAS DDF or EOT domains did not significantly predict physical functioning at 12-month follow-up. In step 4, when added to a regression equation with treatment condition and baseline physical functioning, change in TAS full scale score was still a significant predictor of the outcome, while condition was no longer a significant predictor. In the final model, when controlling for baseline physical functioning, and post-treatment defensiveness, mental health and somatosensory amplification, change in the full scale score was marginally significant as a predictor of physical functioning at follow-up (p = .06). When controlling for baseline physical functioning, and post-treatment defensiveness, mental health and somatosensory amplification, change in the DIF domain significantly predicted physical functioning at follow-up, while condition was no longer significant (see Table 7d). The Sobel test for mediation was just above significance (z = 1.75, p = .08).

Interaction Effects. Entered with baseline physical functioning in a regression model, the interaction between condition and change in full scale TAS score was significant in predicting physical functioning at post-treatment, F (2, 74) = 147.25, p < .0001, p = .02. This effect dropped below significance levels when added to the equation with treatment condition and baseline physical functioning along with it (p = .18), and was therefore eliminated from the final model. The interaction between treatment

condition and change in TAS DIF domain was not significant in predicting post-treatment physical functioning. No other interactions were analyzed.

Daily Symptom Diary as Outcome Variable. The first step of the mediation analysis was significant; treatment condition significantly predicted diary scores at post-treatment when controlling for baseline diary scores, F (2, 74) = 11.45, ß = -.29, p = .006. Step 2 was identical to that conducted in previous analyses. In step 3, when controlling for baseline diary scores, neither the full scale TAS-20 nor any of the domain scores significantly predicted diary scores at post-treatment or follow-up, with one exception. Change in the EOT domain significantly predicted diary scores at post-treatment when controlling for baseline diary scores, ß = .26, p = .02. When added to the equation with condition and baseline diary scores, change in the EOT domain became marginally significant, ß = .20, p = .06. In the final model, however, when controlling for baseline diary scores, and post-treatment defensiveness, mental health and somatosensory amplification, change in EOT was a significant predictor of diary scores at post-treatment, ß = .30, p = .01 (see table 7e). The Sobel test for this was non-significant, however (z = -1.74, p = .08).

12-Month Diary Scores as Outcome. The first step of the mediation analysis was significant; treatment condition significantly predicted diary scores at follow-up when controlling for baseline diary scores, F (2, 69) = 17.53, ß = -.31, p = .002. Step 2 was identical to that conducted in previous analyses. In step 3, none of the change scores significantly predicted diary scores at follow-up when controlling for baseline diary scores.

Discussion

The current study was concerned with examining factors associated with improvement in somatization symptoms through participation in a cognitive behavioral treatment for somatization disorder. Specifically, this study examined whether a cognitive behavioral treatment for somatization disorder would affect emotional experiencing as assessed through the Toronto Alexithymia Scale-20 and moreover, whether decreases in alexithymia would predict and/or mediate improvement in somatization symptoms over the course of treatment and the 12-month follow-up period. There were three major hypotheses in the current study: 1) Participants in the CBT group would change significantly more on alexithymia than those in the PCL group; 2) Participants in the CBT group would score significantly lower on alexithymia than those in the PCL group at post-treatment and follow-up, and 3) changes in alexithymia would predict and possibly mediate improvements in somatization symptoms and related outcomes, including physical functioning and daily symptom diary scores.

The first hypothesis was fairly well supported. In repeated measures analyses of variance, significant time by condition interactions for the TAS-20 full scale scores and the DIF domain and a marginally significant time by treatment interaction for the EOT domain revealed that participants in the CBT condition changed significantly more over the course of treatment and 12-month follow-up in alexithymia than participants in the PCL condition in the hypothesized direction. These changes were evident despite significant correlations among alexithymia scores at all time points, indicating that alexithymia was relatively stable over the course of the study. Moreover, participants in the current study began the study at a range of alexithymia that was similar to those found

in several previous studies using somatoform samples (Bach & Bach, 1996; Waller & Scheidt, 2004). Thus, despite stability in the TAS scores, these findings suggest that alexithymia may decrease in response to a CBT protocol addressing cognitive, affective, and behavioral strategies to improve medically unexplained physical symptoms. Perhaps because it has generally been accepted that alexithymia is a relatively stable trait (De Gucht, 2003), there has been little research directly examining the possibility that a psychotherapy could directly influence alexithymia scores (e.g., see Berenesvaite, 2000 for an exception). Thus this study provided preliminary support for the notion that a cognitive behavioral treatment aimed targeting somatization disorder may significantly influence alexithymia in a sample with somatization disorder.

The importance of change in alexithymia in somatization disorder may be questioned when considering the relatively low baseline rate of alexithymia in the current study sample. Specifically, approximately one-quarter of the participants in this study were classified as alexithymic at baseline according to pre-established cut-off scores (Bagby et al., 1997). However, although this initial level of alexithymia may seem low, it is consistent with the rates of alexithymia reported in other studies, including a recent study of heterogeneous psychosomatic patients (Waller & Scheidt, 2004). An explanation for the relatively low baseline level of alexithymia in the current study sample concerns the intervention setting; specifically, in volunteering for a psychosocial intervention for their medically unexplained physical symptoms, individuals in the current study may have been more amenable to a psychosocial explanation of and treatment for their symptoms, and hence may have been less alexithymic than the population of somatization disorder patients in general. It is possible that focusing only on alexithymic

individuals with somatization disorder would yield more significant results than were found in the current study. However, in the current study, decreases in alexithymia were significantly correlated with certain outcomes including improved global severity of somatization symptoms, suggesting that even for the majority of study participants scoring in the non-alexithymic range, this construct may nonetheless be associated with improved outcomes. Therefore, it is possible that change in alexithymia may be important even for individuals with somatization disorder who fall below the cut-off score for being considered alexithymic.

Because of the previously well-documented association between alexithymia and mental health (Deary et al., 1997; Katon et al., 1991; Rief, Heuser, & Fichter, 1996; Waller & Scheidt, 2004), it might be suggested that decreases in alexithymia reflected improvements in overall mental health rather than increased identification and differentiation of emotions and decreased externally oriented thinking. Indeed, the previously documented relationship between alexithymia and mental health was replicated in the current study, as alexithymia was significantly correlated with poorer mental health. This was found for the full scale score as well as for the individual subscales. Moreover, the association between alexithymia and somatization symptoms in this study persisted even when controlling for mental health, as has been previously found in similar studies (De Gucht, Fischler & Heiser, 2004; Porcelli et al., 1999; Porcelli et al., 2002; Waller & Scheidt, 2004). Yet participants in the CBT condition did not change significantly more than those in the PCL condition on mental health, and therefore the change in alexithymia does not appear to be attributable to an overall improvement in mental health. In addition, although alexithymia scores were

significantly correlated with poorer mental health, change in alexithymia was not significantly correlated with mental health. It is thus unlikely that improvements in mental health are responsible for the change in alexithymia scores.

The second hypothesis, that participants in the CBT group would be significantly lower on alexithymia than those in the PCL condition following treatment was also partially supported. Despite the significant changes in the full scale TAS-20 and in the DIF and EOT subscales, treatment conditions differed significantly at post-treatment on the EOT only, while no significant differences between groups emerged at follow-up. Surprisingly, treatment groups did not differ significantly on the DIF at either endpoints, despite a significant time by treatment interaction for change in this dimension of alexithymia. While it follows that a CBT protocol emphasizing awareness of one's inner feelings and thoughts would be associated with a cognitive shift away from externally oriented thinking, the lack of significant differences between groups in DIF and in change in DDF domains is unclear but could be due to a lack of statistical power. Although treatment in the current study aimed at increasing participants' awareness and expression of their feelings in relation to their symptoms, perhaps not enough time was spent in distinguishing among various emotional states to evoke change in the DDF domain of the TAS. It would be interesting to examine whether a more extensive intervention targeting the range of emotional functioning would bring about broader decreases in alexithymia in somatization disorder patients. Considering the evidence that alexithymia is a fairly entrenched mode of emotional processing (Lane et al., 2000), more extensive and possibly more direct methods of increasing emotional functioning may be necessary to evoke clinically meaningful change in alexithymia.

The fact that decreases in alexithymia were significantly lower in the CBT condition at post-treatment in the EOT subscale only differs from the more extensive effects of treatment found in Berenesvaite's (2000) study. Perhaps the fact that the treatment in Berenesvaite's (2000) study directly targeted alexithymia resulted in a greater decrease in alexithymia in that study. In contrast with the broad focus in the current CBT protocol, which targeted stress reduction and behavioral engagement as well as affective experience, the foregoing study included training in reporting dreams and enhancing fantasies among other emotion-oriented exercises. It is also possible that some of the reduction in alexithymia in that study may reflect a treatment targeted at a specific measure, much as a school-based intervention might specifically teach items known to be tested in standardized examinations.

The third hypothesis stated that improvements in alexithymia, as evidenced by decreases in the TAS-20, would, to some extent, account for improvement in outcomes as a result of participation in the treatment study. This hypothesis was tested in a variety of ways, including responder analyses, correlational analyses, and regression analyses testing change in alexithymia as a mediator of the effect of treatment on outcomes.

Responders to treatment did not differ from non-responders in terms of amount of change experienced in alexithymia at any time point. Nor did change in alexithymia predict responder status at post-treatment or follow-up. This would appear to disconfirm the hypothesis. However, change in alexithymia was significantly correlated with improvement in somatization symptoms and physical functioning at both endpoints, suggesting a positive association between decreases in alexithymia and improved outcomes. In addition, results of regression analyses demonstrated that decreases in the

EOT and the DIF domains of the TAS predicted lower diary scores and greater physical functioning at post-treatment and at follow-up, respectively, over and above baseline functioning, and mental health, defensiveness, and somatosensory amplification. In addition, adding change scores to the regression equations lowered the significance of condition in predicting outcomes, especially in the regression equations predicting physical functioning. This provides some support for the notion that change in alexithymia may account for some of the effects of treatment condition on outcomes. Yet, for the primary outcome of improvement in somatization symptoms, although change in alexithymia significantly predicted improvement in somatization symptoms at post-treatment and follow-up, these effects were not significant when controlling for baseline functioning and post-treatment control variables. Moreover, results of Sobel tests for the significance of mediation effects, performed when the mediating variable significantly predicted outcomes in the final model, were not significant. The most highly significant Sobel test was that assessing change in EOT as a mediator in predicting daily symptom diary scores at post-treatment, which was slightly above significance levels. These findings provide some support for the hypothesis in that they contribute to the association between alexithymia and outcomes in somatization disorder but ultimately do not support change in alexithymia as a mediator in the association between treatment and outcomes in this study.

One possible explanation for the loss of the statistical significance of change in alexithymia in predicting outcomes is the inclusion of somatosensory amplification in the final regression models. Previous researchers have suggested that somatosensory amplification might be a mechanism by which faulty emotional processing leads to

somatization (De Gucht & Heiser, 2003). The current study offers mixed some support for this notion. At baseline, difficulty in distinguishing feelings (DDF subscale of the TAS-20) was significantly correlated with somatosensory amplification. This is consistent with the notion that difficulty in distinguishing one's emotional experiences is associated with amplification of somatic symptoms and with some prior findings (Nakao et al., 2002; Wise & Mann, 1994). Yet somatosensory amplification was not significantly correlated with other subscales or the full scale TAS. In addition, although somatosensory amplification was not a significant predictor of improvement in somatization symptoms at post-treatment when entered alongside condition, baseline severity and change in TAS-20 scores, it did reduce the contribution of change in alexithymia to all outcomes except diary scores. In contrast, the other control variables, including defensiveness and mental health, generally did not detract from the significance of change in alexithymia in predicting outcomes. Interestingly, alexithymics did not score significantly higher in somatosensory amplification than non-alexithymics, which would have been predicted according to this association, in contrast the findings reported by Wise and Mann (1994). Also interestingly, 12-month follow-up somatosensory amplification was the only variable to distinguish responders from non-responders at that time point, with responders reporting significantly greater somatosensory amplification at follow-up than non-responders. This seemingly surprising finding may suggest the presence of a different variable not being measured that may account for the enhanced somatosensory amplification at follow-up. Lastly, the SSAS was generally not significantly associated with baseline levels of somatization severity, as assessed through the CGI-SD or the diary scores or with these measures at post-treatment. Nor did SSAS change significantly

across the study, while severity of somatization symptoms did, indicating a lack of association between somatosensory amplification and somatization symptoms. However, SSAS scores were significantly negatively correlated with mental health at post-treatment. This finding is consistent with prior research (Barsky et al., 1990) and suggests that continued amplification of somatic symptoms is associated with worse mental health. It is interesting that this finding emerged at post-treatment and not at baseline.[7] Clearly, additional studies are needed to elucidate the relationships among somatosensory amplification and mental and physical health outcomes in populations with somatization disorder. In sum, this study offers some support for the notion that increased amplification of somatic experiences, at least as it is captured by the SSAS, may be associated with alexithymia but offers little support for the association between somatosensory amplification and somatization. Perhaps hypochondriasis is better typified by the cognitive processes in somatosensory amplification while somatization disorder is more appropriately characterized by disruption in affective functioning. Such a hypothesis needs to be clarified through further research examining both hypochondriacal and somatization symptoms in somatization disorder patients. Experimental studies would be useful in examining whether actual arousal levels in response to physical symptoms differ among alexithymics and non-alexithymics and further, whether such arousal leads to increased symptom reporting and severity.

The non-significance of the Sobel tests for mediation may disconfirm the hypothesis of alexithymia as a possible mechanism of change in improvement through the CBT treatment program, or it may point to a sample size that was simply too small to

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[7] SSAS was also not correlated with mental health scores at 12-month follow-up.

elicit significant findings. Perhaps a larger sample size would have provided enough power to push the statistical finding toward significance. Given the large power obtained through the power analyses, it might seem contradictory to state that the sample was not large enough to yield significant findings. However, the power analysis was based on a study that may have overestimated the association between alexithymia and somatization (Porcelli et al., 2003), as the TAS-20 scores in that population were substantially larger than in the current and other similar studies (Bach & Bach, 1996; Waller & Scheidt, 2004). Indeed, although that study was similar to the current one in terms of procedures, population and statistical analyses, it may not have been an appropriate study on which to base an estimate of sample size, due to the somewhat high level of alexithymia in that study sample.

Although much previous research has supported the link between alexithymia and somatization (DeGucht & Heiser, 2003), in the current study, baseline correlations between alexithymia and somatization symptoms, as assessed through the CGI-SD, were not significant. It is therefore even more striking that change in alexithymia predicted improvement in somatization symptoms using a similar measure at post-treatment and follow-up. Alexithymia in the current study was correlated with physical functioning at baseline, but in a seemingly contradictory direction. Specifically, higher alexithymia was associated with greater physical functioning and alexithymics exhibited significantly greater physical functioning than non-alexithymics. However, a decrease in alexithymia full scale score and DIF subscale over the course of treatment was nonetheless associated with better physical functioning at post-treatment in regression analyses. It is interesting that despite the negative correlation between physical functioning and alexithymia at

baseline, decreases in alexithymia nonetheless predicted better physical functioning at post-treatment and follow-up. The positive association between alexithymia and physical functioning is inconsistent with certain studies that found alexithymia to be predictive of worse physical symptoms (Porcelli et al., 2002). Differences in findings may reflect differences in the means of assessment. For instance, physical functioning, as measured broadly through a subscale of the MOS, may differ from a scale used to assess gastrointestinal symptoms. Studies should continue to examine this association explicitly in samples with somatization disorder and to compare this association with that of alexithymia and other outcomes.

As one of the few studies directly examining alexithymia in a sample with full somatization disorder, the current study sheds light on the functioning of the full scale of the TAS-20 and its subscales. Findings from this study demonstrate that although the subscales all correlated significantly with one another, the EOT subscale correlated less well with other subscales. This is consistent with prior findings concerning the discrepancy between the EOT and the other subscales (Deary et al., 1997; De Gucht & Hesier, 2003). However, although it has been suggested that the EOT may not reflect the construct of alexithymia as well as the other two domains (De Gucht & Heiser, 2003), this study shows that the EOT subscale is useful to include in studies with alexithymia and somatization. In the current study, the EOT subscale significantly predicted diary symptom diary scores at post-treatment, showing that the EOT subscale was a unique predictor of a self-report measure of somatization symptoms. In addition, participants in the CBT group scored significantly lower on only the EOT subscale at post-treatment than the PCL group. These findings, in conjunction with prior research findings

correlating the EOT subscale with alternative observer-rated measures of alexithymia, suggest that the EOT is related to both somatoform outcomes and to alexithymia. Waller and Scheidt (2004) showed that the EOT was correlated with observer-rated measures of alexithymia while the DIF and DDF domains were not (Waller & Scheidt, 2004). They proposed that the EOT subscale may tap into the cognitive component of alexithymia while the DIF and DDF domains may reflect a purely affective component of alexithymia (Waller & Scheidt, 2004). In future research, it would be interesting to examine the associations between the affective and cognitive components of alexithymia. For instance, just as certain cognitions are known to correspond to the physical and emotional components of the panic experience (Salkovskis & Clark, 1990), perhaps certain cognitions accompany alexithymic affective experiences and related behaviors.

Of interest in the current study was an examination of defensiveness, as measured by the Marlowe-Crowne scale (Crowne & Marlow, 1960). In contract with some prior research (Rutledge & Linden, 2000), defensiveness was not correlated with negative physical outcomes in the current study. Repressive copers in the current study, however, did differ from other groups in certain ways that echoed previous findings. Specifically, the finding that repressors reported better mental health than non-repressor counterparts was consistent with past research (Gick, McLeod & Hulihan, 1997). In contrast with prior research (Burns, 2000; Burns et al., 2001), repressive copers did not exhibit poorer physical functioning or worse somatization symptoms than the other groups. However, those studies were conducted in chronic pain populations that may differ in important ways from samples with full somatization disorder.

The current study supports the finding of a relatively high rate of individuals with both high manifest anxiety and high defensiveness in samples with functional somatic syndromes (Brosschot and Aarsse, 2001; Creswell & Chalder, 2001). Out of the groups coded according to extreme TMAS and MCS scores, the group with the largest number of individuals in the current study was the high defensiveness/high anxiety group. However, this group still amounted to only 20% of the full sample. Thus, this rate was not as high as that in the study of fibromyalgia (FMS) patients found in Brosschot and Aarsse's (2001) study, in which almost all of the FMS patients scored in this group, or in Creswell & Chalder's (2001) study, in which almost half of the sample with chronic fatigue syndrome fell into this group. These differences may also be due to a slightly less inclusive method of classifying these groups in the current study.[8] Individuals in this study in the high defensiveness/high anxiety group were characterized by high alexithymia, whereas repressors were characterized by low alexithymia. In addition, the high defensiveness/high anxiety group was characterized by poor mental health. These findings are consistent with prior findings (Creswell & Chalder, 2001; Myers, 1995) and suggest that somatization may be linked with both high rates of alexithymia as well as high rates of defensiveness and emotional distress. In light of Lane et al.'s (2000) findings, it may be that the high defensiveness/high anxiety group, in scoring as significantly more alexithymic than other groups, may be the group exhibiting the most

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[8] When defensiveness and anxiety groups were coded exactly as coded in the Creswell & Chalder (2001) study, 34.5% of the study sample fell into the high defensiveness/high anxiety group, 24% fell into the repressive coper group, 32% fell into the low defensiveness/high anxiety group, and 9.5% fell into the low defensiveness/low anxiety group.

significant emotional deficit, with repressive copers exhibiting a less substantial emotional deficit. Interestingly, alexithymics scored lower on defensiveness than non-alexithymics, suggesting that alexithymia may differ from defensiveness in ways that are not currently understood. Continued examination of both alexithymia and defensiveness will be necessary to parse out the relationship between alexithymia and repressive coping in individuals with somatization disorder.

There are several possible limitations to the current study. Firstly, as mentioned previously, a small sample size may have contributed to the lack of significant findings for mediation effects for change in alexithymia.[9] Secondly, the CBT condition was not compared with another form of treatment and so it is unclear if changes in alexithymia may be attributable to the content of the CBT protocol administered in the present study. It may be that individuals with somatization disorder taking part in any kind of psychotherapy would decrease in alexithymia. Thirdly, because mediational analyses do not, by nature, assess causation, it is not possible to conclude from these study findings that participation in the CBT program decreased alexithymia, which then decreased somatization symptoms. Such inferences of causality are not possible from the current

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[9] Part of this problem may have been due to using completers in most analyses. Intent-to-treat analyses generally yielded similar findings to those obtained using the full sample. However, in the intent-to-treat analysis comparing change scores from post-treatment to follow-up between treatment conditions, those in the CBT condition appeared to increase slightly in the EOT subscale of the TAS while those in the PCL condition appeared to decrease in this subscale. This contrasts with the analysis conducted using the completers only, in which no significant difference was found. This difference goes in the opposite direction as other findings regarding greater reductions in the EOT subscale in the CBT condition, suggesting that substituting the last data point carried forward may have biased this analysis.

study design. Additional experimental studies would be useful in examining the relationship between change in emotional awareness and consequent somatization symptoms. Recent experimental studies that are beginning to be conducted in healthy subjects (e.g., Kano, et al., in press) support the pathway between alexithymia and elevated sensitivity to physical sensations, thereby possibly suggesting a pathway to somatoform symptoms. However, such studies will need to be conducted in individuals with somatization symptoms in order to make these findings applicable to populations with somatization disorder.

Despite the limitations to the current study, this study has contributed to current knowledge surrounding the association between somatization and emotional functioning by examining alexithymia in somatization disorder patients involved in a randomized controlled trial of CBT. Findings lent fairly strong support for the notion that a CBT protocol emphasizing stress management, behavioral strategies, and affective components decreased alexithymia in comparison to those whose physicians received a psychiatric letter only. Findings partially supported change in alexithymia as a predictor of outcomes but did not support change in alexithymia as a mediator of improvement in symptoms or of other outcomes as a result of participation in the treatment. Because the present study was the only known study to examine this hypothesis in participants of a CBT study with full somatization disorder, the findings must be replicated in additional studies using somatization disorder. It would also be advisable to extend the findings from the current study here in future studies with larger sample sizes, in which significant mediation effects may emerge that support improved emotional functioning as a mediator of improvement in treatment. In addition, because the study did not directly manipulate

alexithymic characteristics, a causal relationship between the CBT intervention and reductions in alexithymia could not be made. A future study that tests the effects of a CBT intervention in a sample specifically scoring high in alexithymia would make a causal hypothesis more plausible. Despite limitations of the current study, the findings of the current study corroborate the notion of impaired emotional functioning in those with somatization disorder and support increasing research efforts aimed at improving emotional functioning in this population.

Appendix

List of All Measures Administered in CBT Study for Somatization Disorder

Diagnostic and Structured Interviews:

Clinician's Global Impression Scale (CGI-SD; GI-SD)

Global Assessment of Functioning Scale (GAF)

Hamilton Depression Inventory

Hamilton Anxiety Inventory

Structured Interview for Diagnosis for the DSM-IV-TR

Target Somatic Symptom Index

Self-Report Questionnaires:

Affect Intensity Measure

Beck Depression Inventory

Beck Anxiety Inventory

Daily Symptom Diary

Differential Emotions Scale-IV-Revised

Expectation Rating Scale

Life Experiences Survey

Life Satisfaction Scale

Marlowe-Crowne Social Desirability Scale

Minnesota Multiphasic Personality Inventory (MMPI)

MOS 26-Item Short-Form Health Survey (SF-36)

Patient Health Questionnaire (PHQ-15)

Psychosomatic Symptom Checklist

Self-Efficacy Scale

Severity of Symptoms Scale

Somatosensory Amplification Scale

Taylor Manifest Anxiety Scale

Tellegen Absorption Scale

Toronto Alexithymia Scale

Stoicism Questionnaire

Whitely Index for Hypochondriasis

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Table 1. Baseline Characteristics of the Participants.

Characteristic	CBT Group (N = 43)	PCL Group (N = 41)	P Value
Age, mean (SD), y	45.47 (8.45)	47.85 (10.99)	.27
Female	36 (84)	39 (95)	.09
Race/ethnicity			.94
White	36 (84)	33 (80)	
African American	1 (2)	2 (5)	
Hispanic	5 (12)	5 (12)	
Other	1 (2)	1 (2)	
Education			.46
Graduate degree	6 (14)	7 (17)	
College degree	16 (37)	9 (22)	
Some college	9 (21)	14 (34)	
High school degree	11 (26)	9 (22)	
Some high school	1 (2)	2 (5)	
Married	21 (49)	22 (54)	.90
Employed	23 (53)	23 (56)	.81
Receiving disability	9 (21)	7 (17)	.65
Duration of symptoms, Mean (SD), y	24.95 (11.54)	25.00 (15.12)	.99

Abbreviations: CBT, cognitive-behavioral therapy, PCL, psychiatric consultation letter.
*Data represent frequencies followed by percentage of patients in parentheses unless otherwise indicated.

Table 2. Baseline Mean Scores on Predictor and Outcome Variables.

Variable	Total Sample (N = 84)
TAS Total Score	49. 31 (12.91)
TAS Difficulty in Identifying Feelings (DIF)	19.10 (6.53)
TAS Difficulty Describing Feelings (DDF)	12.38 (4.70)
TAS Externally Oriented Thinking (EOT)	17.83 (4.66)
Marlowe Crowne Social Desirability Scale (MCS)	17.90 (6.03)
Taylor Manifest Anxiety Scale (TMAS)	9.93 (5.32)
Somatosensory Amplification Scale (SSAS)	29.06 (7.19)
Clinician's Global Impression Scale for Somatization Symptoms (CGI-SD)	5.01 (.83)
Medical Outcomes Survey-Short Form Physical Functioning Subscale (MOS-PF)	53.87 (29.03)
Medical Outcomes Survey-Short Form Mental Health Subscale (MOS-EF)	57.33 (19.18)
Daily Symptom Diary	3.38 (.86)

Note: Means are presented followed by standard deviations in parentheses. Missing data for SSAS scores left an N of 76 for this variable only.

Table 3. Correlation Matrix Among all Variables at Baseline and Post-treatment.

Post-treatment											
GI2	.12	.06	.09	.17	.02	.07	-.19	-.13	-.18	.49***	**-.17**
Diary	.09	-.02	.06	.22	.05	-.00	-.06	-.24*	-.38***	**.39*** **	.61***
MOS-PF	.25	.27**	.17	.15	.01	-.02	.22	.09	**.89*** **	-.44***	-.59***
MOS-EF	-.14	-.22	-.18	-.01	-.03	-.65***	-.26*	**.68*** **	.06	-.16	-.20
SSAS	.04	.07	.07	-.06	-.22	-.36**	**.69*** **	-.13	.22	-.06	-.03
TMAS	.26*	.30**	.28*	.00	-.15	**.81*** **	.28*	-.70***	-.01	-.01	-.06
MCS	-.21	-.26*	-.10	-.11	**.92*** **	.-35***	-.24*	.24*	-.01	-.01	-.05
TASEOT	.75***	.39***	.58***	**.74*** **	.12	.19	-.01	-.22*	.05	.06	-.02
TASDDF	.86***	.59***	**.72*** **	.42***	-.25*	.31**	.24*	-.28*	.25*	-.03	-.02
TASDIF	.85***	**.77*** **	.62***	.39***	-.26*	.41***	.20	-.27*	.29**	-.14	-.04
TAST	**.81*** **	.87***	.83***	.71***	-.18	.39***	.19	-.32**	.26*	-.06	-.02
Baseline	TAST	TASDIF	TASDDF	TASEOT	MCS	TMAS	SSAS	MOS-EF	MOS-PF	Diary	CGI

* p<.05, ** p<.01, *** p<.001
Baseline correlation coefficients are presented on the bottom right half of the table and post-treatment correlation coefficients are presented on the top left half of the table.
Correlation coefficients in bold represent the coefficient between that variable at baseline and at post-treatment.
GI2=global improvement in somatization symptoms at post-treatment; MOSPF=SF-36 Physical Functioning scale; MOSEF=SF-36 Mental Health scale; SSAS=Somatosensory Amplification Scale; TMAS=Taylor Manifest Anxiety Scale; MCS=Marlowe-Crowne Social Desirability Scale; TASEOT=Externally Oriented Thinking Subscale of TAS-20; TASDDF=Difficulty Distinguishing Feelings Subscale of TAS-20; TASDIF=Difficulty Identifying Feelings Subscale of TAS-20; TAST=TAS-20 full scale score.

Table 4. Change in Alexithymia Scores from Baseline to Post-treatment in the Total Sample and Each Treatment Group.

Toronto Alexithymia Scale Change Scores	Total (N = 77)	CBT Group (N = 39)	PCL Group (N = 38)
TAS Total Score	-1.88 (7.80)	-3.90* (7.76)	.18* (7.38)
TAS Difficulty in Identifying Feelings (DIF)	-.62 (4.33)	-1.79* (4.13)	.58* (4.24)
TAS Difficulty Describing Feelings (DDF)	-.66 (3.42)	-.74 (3.57)	-.58 (3.31)
TAS Externally Oriented Thinking (EOT)	-.59 (3.19)	-1.36* (3.23)	.18* (2.99)

Note: Means are presented followed by standard deviations in parentheses.
Table values are based on analyses conducted with treatment completers.
Means with a * within each row are statistically different from one another at a .05 level of significance.

Table 5. Correlation Matrix among TAS Change Scores (from baseline to post-treatment) and Outcomes.

	TAS Full Scale	DIF Domain	DDF Domain	EOT Domain
Post-treatment MOS-PF	-.26*	-.27*	-.23*	-.01
MOS-EF	-.13	-.12	-.13	.00
Gl-SD	.33**	.26+	.11	.34**
Daily Symptom Diary	.21	.17	.07	.21
12-month Follow-up MOS-PF	-.32**	-.37**	-.20	-.09
MOS-EF	-.16	-.15	-.06	-.13
GI-SD	.34**	.32**	.27*	.13
Daily Symptom Diary	.12	.12	.06	.05

Note: Table values are based on analyses conducted with treatment completers.
Abbreviations: GI-SD, Global improvement in somatization symptoms based on the Clinician's Rating Scale.
* $p<.05$, ** $p<.01$

Table 6. Baseline and Post-Treatment Scores on Variables by Groups Distinguished by High and Low Scores on TMAS and MCS.

Variable	High defensiveness, high anxiety (N = 11)	High defensiveness, low anxiety (Repressors) (N = 16)	Low defensiveness, high anxiety (N = 6)	Low defensiveness, low anxiety (N = 3)
Baseline TMAS	16.27[a]	3.25 [b]	17.17 [a]	4.3 [b]
Baseline MCS	21.36[a] (2.11)	24.06[a] (3.28)	6.17[b] (3.60)	10.33[b] (.58)
Baseline TAS Total Score	57.28[a] (10.67)	43.75[b] (8.04)	55.50 (13.90)	49.67 (15.89)
Post-treatment TAS Total Score	53.18[a] (7.73)	42.00[b] (8.33)	54.67[a] (6.22)	48.33 (9.45)
12 Month Follow-up TAS Total Score	54.00 [a] (8.28)	42.93 [b] (9.03)	51.67 (10.93)	43.00 (15.10)
Baseline TAS-DIF domain	23.91[a] (4.83)	15.19[b] (5.27)	23.00[a] (6.23)	22.67 (5.51)
Post-treatment TAS-DIF domain	21.91[a] (4.61)	14.94[b] (4.11)	22.50[a] (2.51)	21.33 (2.89)
12 Month Follow-up TAS-DIF domain	22.45[a] (3.88)	15.13 [b] (5.71)	21.33 (3.39)	19.67 (5.77)
Post-treatment TAS-DDF domain	13.00 (2.90)	10.31[a] (3.22)	15.17[b] (2.79)	10.67 (6.03)
Baseline MOS-EF	44.00[a] (14.09)	76.00[b] (11.31)	34.67 [ac] (12.31)	61.33[ab] (4.62)
Post-treatment MOS-EF	46.91[a] (22.13)	76.25[b] (11.91)	53.33[a] (17.28)	64.00 (12.00)
12 Month Follow-up MOS-EF	55.64[a] (27.68)	80.25[b] (11.43)	48.00[a] (18.42)	56.00 (14.42)
Baseline CGI	5.09 (.94)	5.00 (.82)	5.00 (1.10)	5.33 (.58)
Post-treatment Improvement on CGI	3.45 (.93)	3.25 (.93)	3.33 (1.21)	4.00 (1.00)
12 Month Follow-up Improvement on CGI	3.27 (.90)	3.25 (1.18)	3.33 (1.21)	3.00 (1.00)

Note: Standard deviations are in parentheses below the means.
Values with different letters are significantly different from one another at the $p < .05$ level of significance.
Post-treatment and follow-up values include the last observation carried forward for participants missing evaluations at those time points.

Table 7a. Results from analysis testing mediator status of change on the TAS full scale in the relationship between treatment condition and global improvement in somatization symptoms at post-treatment.

Step	Outcome Variable	Predictor Variable(s)	R^2	F	df	B	SE	ß	p <
Step 1	Global Improvement	Condition	.32	36.06	1, 75	-1.04	.17	-.57	.0001
Step 2	TAS Full Scale Change	Condition	.07	5.59	1, 75	-4.08	1.73	-.26	.05
Step 3	Global Improvement	TAS Full Scale Change	.11	9.25	1, 75	.04	.01	.33	.01
Step 4	Global Improvement	Condition				-.95	.18	-.52	.0001
		TAS Full Scale Score				.02	.01	.19	.05
		Model	.36	20.81	2, 74				.0001
Final Model		Condition				-.91	.19	-.49	.0001
		TAS Full Scale Score				.02	.01	.16	ns
		Post-treatment Mental Health				-.01	.01	-.20	ns
		Baseline CGI				-.15	.10	-.13	ns
		Post-treatment Defensiveness				.00	.02	.02	ns
		Post-treatment Somatosensory Amplification				.02	.02	-.20	ns
		Model	.40	9.39	5, 71				.0001

Note: Table values are based on analyses conducted with treatment completers.

Table 7b. Results from analysis testing mediator status of change on the TAS full scale in the relationship between treatment condition and physical functioning symptoms at post-treatment.

Step	Outcome Variable	Predictor Variable(s)	R^2	F	df	B	SE	ß	p <
Step 1	MOS-PF at Post-Treatment	Condition							
		Baseline MOS-PF							
		Model	.81	158.53	2, 74				

Table 7c. Results from analysis testing mediator status of change on the DIF Domain of the TAS in the relationship between treatment condition and physical functioning symptoms at post-treatment.

Step	Outcome Variable	Predictor Variable(s)	R^2	F	df	B	SE	ß	P <
Step 1	MOS-PF at Post-Treatment	Condition				8.55	2.67	.16	.01
		Baseline MOS-PF				.82	.05	.90	.0001
		Model	.81	158.53	2, 74				.0001
Step 2	TAS Change in DIF Domain	Condition	.08	6.09	1, 75	-2.37	.95	-.28	.05
Step 3	MOS-PF at Post-Treatment	TAS Change in DIF Domain	.08	6.09	1, 75	-1.68	.68	-.27	.05
Step 4	MOS-PF at Post-Treatment	Condition				6.76	2.72	.13	.05
		TAS Change in DIF Domain				-.72	.32	-.12	.05
		Baseline MOS-PF				.80	.05	.88	.0001
		Model	.82	113.33	3, 73				.0001
Final Model		Condition				7.50	3.04	.14	.05
		TAS Change in DIF Domain				-.64	.35	-.11	ns
		Baseline MOS-PF				.80	.05	.87	.0001
		Post-treatment Mental Health				.08	.07	.05	ns
		Post-treatment Defensiveness				.13	.27	.03	ns
		Post-treatment Somatosensory Amplification				.19	.25	.05	ns
		Model	.81	44.56	6, 63				.0001

Note: Table values are based on analyses conducted with treatment completers.

Table 7d. Results from analyses testing mediator status of change on the DIF Domain of the TAS in the relationship between treatment condition and physical functioning symptoms at follow-up.

Step	Outcome Variable	Predictor Variable(s)	R^2	F	df	B	SE	ß	p <
Step 1	MOS-PF at Follow-up	Condition				11.13	4.23	.20	.01
		Baseline MOS-PF				.70	.07	.76	.05
		Model	.59	48.92	2, 69				.0001
Step 2	TAS Change in DIF Domain	Condition	.08	6.09	1, 75	-2.37	.95	-.28	.05
Step 3	MOS-PF at Follow-up	TAS Change in DIF Domain	.14	11.07	1, 70	-2.41	.73	-.37	.01
Step 4	MOS-PF at Follow-up	Condition					4.36	.13	ns
		TAS Change in DIF Domain					.53	-.20	.05
		Baseline MOS-PF					.07	.72	.0001
		Model	.62	37.07	3, 68				.0001
Final Model		Condition				8.73	4.77	.16	ns
		TAS Change in DIF Domain				-1.31	.57	-.21	.05
		Baseline MOS-PF				.64	.08	.69	.0001
		Post-treatment Mental Health				.02	.13	.02	ns
		Post-treatment Defensiveness				.22	.42	.04	ns
		Somatosensory Amplification				.30	.38	.07	ns
		Model	.62	15.62	6, 58				.0001

Note: Table values are based on analyses conducted with treatment completers.

Table 7e. Results from analyses testing mediator status of change on the EOT Domain of the TAS in the relationship between treatment condition and post-treatment daily symptom diary scores (DSD).

Step	Outcome Variable	Predictor Variable(s)	R^2	F	df	B	SE	ß	P <
Step 1	DSD at Post-Treatment	Condition				-.60	.21	-.29	.01
		Baseline DSD				.51	.13	.41	.0001
		Model	.24	11.45	2, 74				.0001
Step 2	TAS Change in EOT	Condition							

Figure 1. Toronto Alexithymia Scale-20 scores over the course of the study by treatment condition.

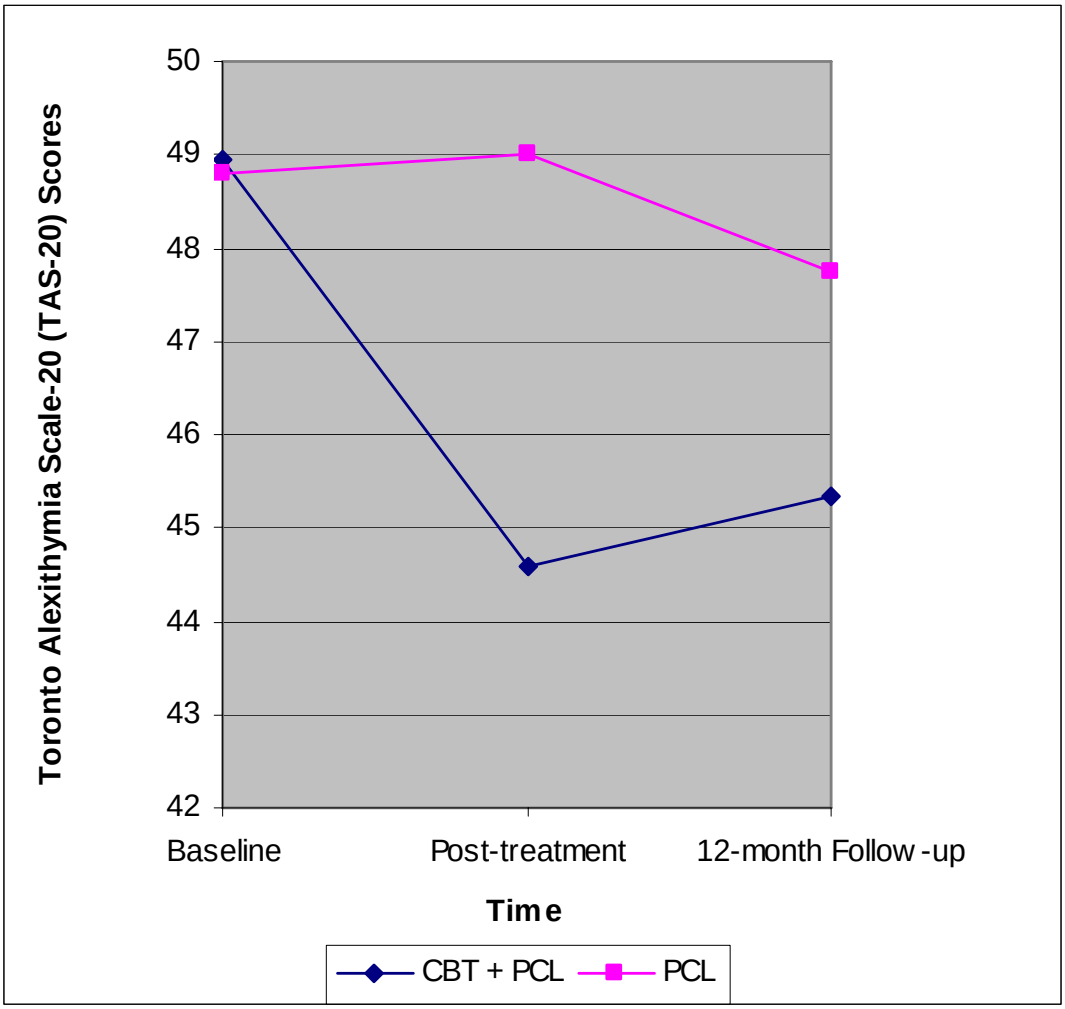


Figure 2. Toronto Alexithymia Scale-20 DIF domain scores over the course of the study by treatment condition.

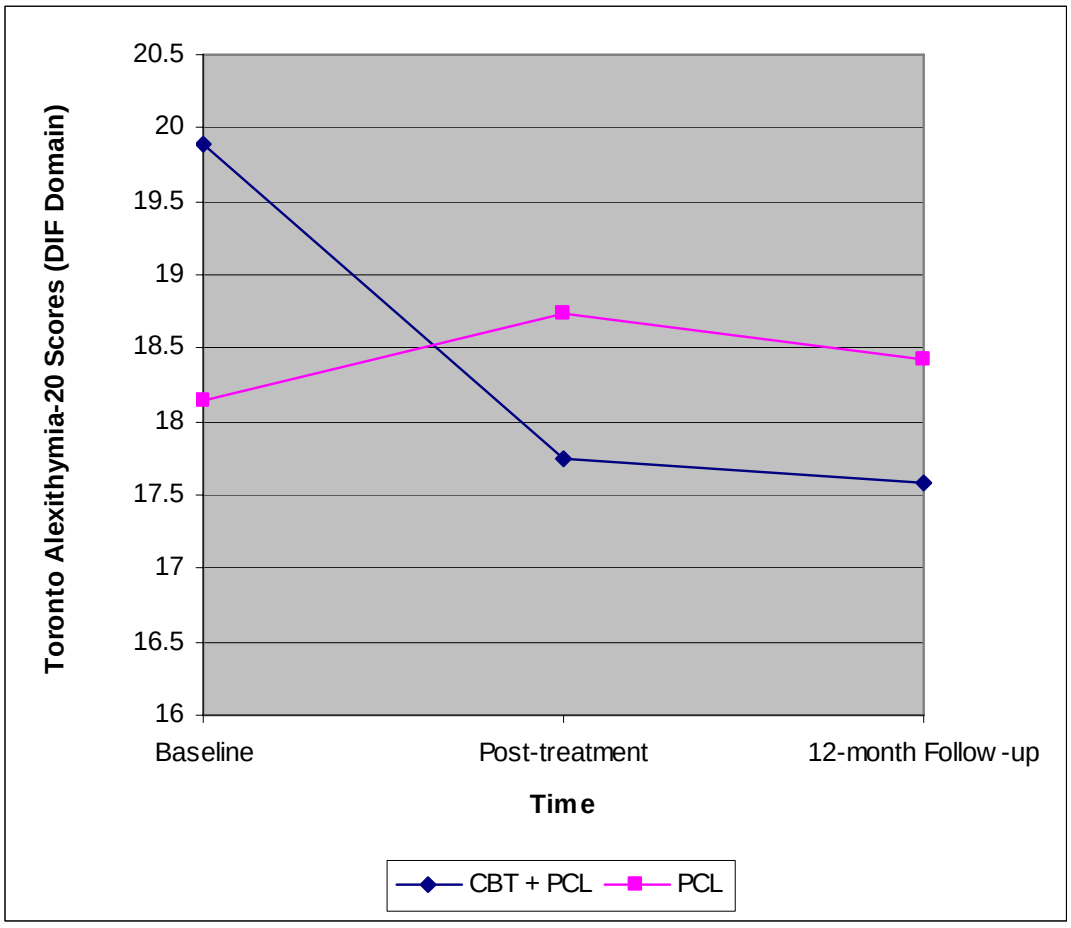
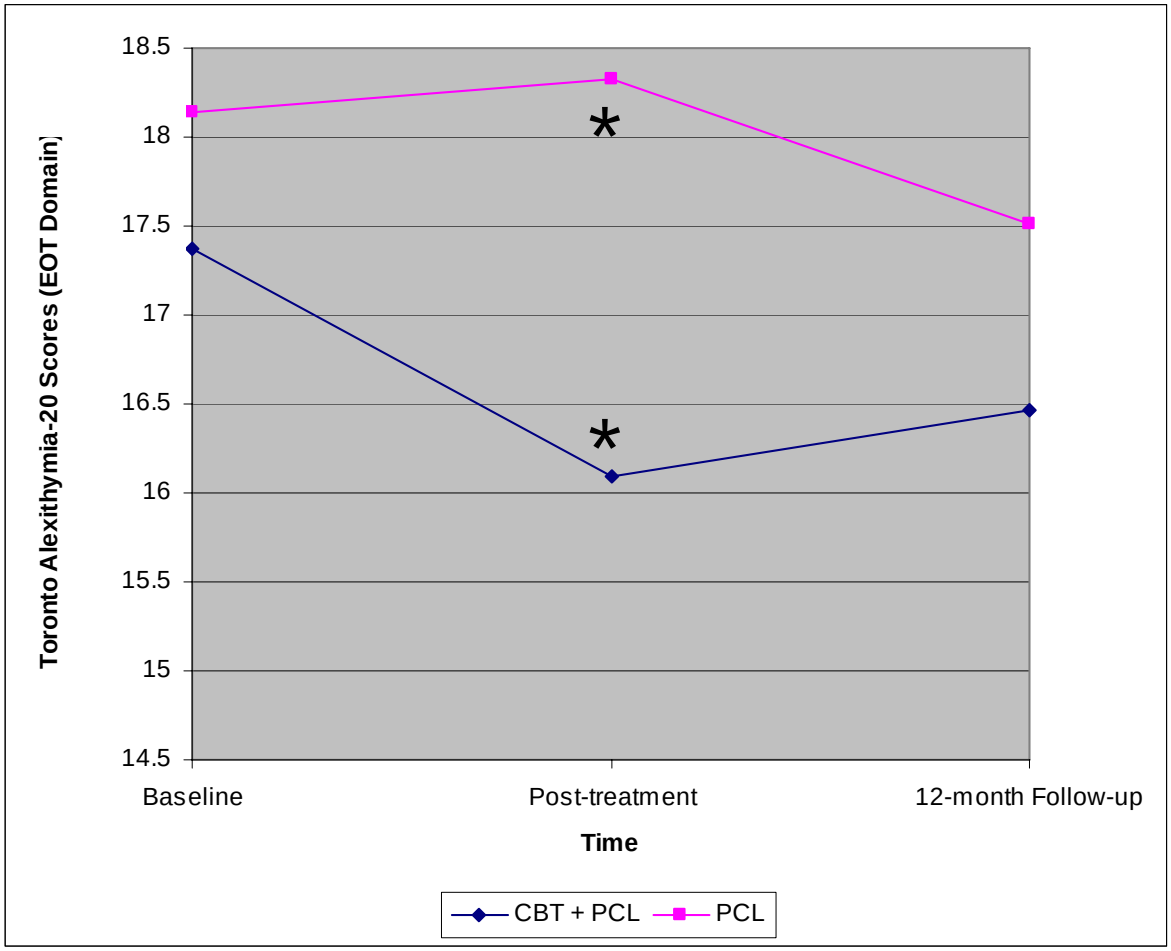


Figure 3. Toronto Alexithymia Scale-20 EOT domain scores over the course of the study by treatment condition.



Note: Asterisks denote differences significant at $p < .05$ level of significance.

Curriculum Vita

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