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Mindfulness-Based Cognitive Therapy Improves Emotional Reactivity to Social Stress: Results from a Randomized Controlled Trial

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The high likelihood of recurrence in depression is linked to a progressive increase in emotional reactivity to stress (stress sensitization). Mindfulness-based therapies teach mindfulness skills designed to decrease emotional reactivity in the face of negative affect-producing stressors. The primary aim of the current study was to assess whether Mindfulness-Based Cognitive Therapy (MBCT) is efficacious in reducing emotional reactivity to social evaluative threat in a clinical sample with recurrent depression. A secondary aim was to assess whether improvement in emotional reactivity mediates improvements in depressive symptoms. Fifty-two individuals with partially remitted depression were randomized into an 8-week MBCT course or a waitlist control condition. All participants underwent the Trier Social Stress Test (TSST) before and after the 8-week trial period. Emotional reactivity to stress was assessed with the Spielberger State Anxiety Inventory at several time points before, during, and after the stressor. MBCT was associated with decreased emotional reactivity to social stress, specifically during the recovery (post-stressor) phase of the TSST. Waitlist controls showed an increase in anticipatory (pre-stressor) anxiety that was absent in the MBCT group. Improvements in emotional reactivity partially mediated improvements in depressive symptoms. Limitations include small sample size, lack of objective or treatment adherence measures, and non-generalizability to more severely depressed populations. Given that emotional reactivity to stress is an important psychopathological process underlying the chronic and recurrent nature of depression, these findings suggest that mindfulness skills are important in adaptive emotion regulation when coping with stress.

Keywords: depression; emotional reactivity; meditation; Mindfulness-Based Cognitive Therapy; Trier Social Stress Test

MAJOR DEPRESSIVE DISORDER (MDD) is a debilitating mood disorder that affects almost 19 million adults in the United States at any given time (Narrow, 1998) and almost 20% of the U.S. population over a lifetime (Blazer, Kessler, McGonagle, & Swartz, 1994; Kessler, Chiu, Demler, Merikangas, & Walters, 2005). MDD is recurrent and progressive, with the likelihood of repeated episodes increasing

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with each subsequent episode. Approximately 60% of individuals who had experienced one episode will experience a second episode, whereas 90% of those who had experienced three episodes will have a fourth one (Judd, 1997; Mueller et al., 1999; Solomon et al., 2000). Thus, depression models attempt to explain not only why certain individuals are more likely to become depressed but also why the likelihood of recurrence increases with each subsequent episode (Hammen, 2005; Kendler, Thornton, & Gardner, 2000; Mitchell, Parker, Gladstone, Wilhelm, & Austin, 2003; Post, 2010; Segal, Gemar, & Williams, 1999; Segal et al., 2006; Segal, Williams, Teasdale, & Gemar, 1996).

Most theories posit an interaction between a latent but progressive vulnerability (diathesis) and negative life events (stress; Abramson et al., 1999; Beck, 1987; Hankin, 2008; Hankin & Abramson, 2001). The relationship between depression and stress is complex and changes over time, such that earlier episodes are more likely than later episodes to be cued by major life stressors (Post, 1992; Stroud, Davila, & Moyer, 2008). Both biological (Post, 1992) and cognitive (Segal, Williams, & Teasdale, 2002) models hypothesize that individual (intrapersonal) vulnerability risk processes are strengthened, and become more "autonomous" with each episode, such that lower levels of external provocation (stress) are needed to trigger a subsequent episode. This phenomenon was first termed "kindling"[1](Post, 1992) and later became known as "stress sensitization" (Monroe & Harkness, 2005; Morris, Ciesla, & Garber, 2010; Post, 2010; Stroud, Davila, Hammen, & Vrshek-Schallhorn, 2011). Stress sensitization explains both the inter- and intraindividual variability in response to stress, both in terms of differing thresholds of stress needed to trigger the same response, and differing magnitudes of response to the same stressor.

Cognitive theorists describe this progressive vulnerability as "cognitive reactivity," or the activation of latent negative information processing biases in response to negative affect that serve to further escalate the negative affect into an episode (Teasdale, 1988). Biological theorists describe the diathesis as insufficient modulation of the limbic system by the prefrontal cortex, resulting in prolonged activation of the amygdala and sympathetic nervous system in response to stressors (Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Drevets, 2001; Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner & Gross, 2005; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007). Both theories report the progressive potentiation or kindling of this vulnerability, whether described in biological or cognitive terms (Post, 1992; Post, Rubinow, & Ballenger, 1984; Segal, Teasdale, Williams, & Gemar, 2002; Segal et al., 1996). The common end result in both models is progressively prolonged or intensified negative affect or "emotional reactivity" in response to stress, that puts individuals at risk for a depressive episode. For example, research has shown that heightened emotional reactivity to daily stress is a hallmark of depressive tendencies (Myin-Germeys et al., 2003; Pine, Cohen, & Brook, 2001) and a predictor of future depression (Cohen, Gunthert, Butler, O'Neill, & Tolpin, 2005; Pine et al., 2001) and poor treatment response (Cohen et al., 2008). Individuals with longer durations of negative affect following daily life stressors are more likely to develop depressive symptoms than those who recover more quickly (Cohen et al., 2005). This failure to "bounce back" from transient negative affect highlights the importance of negative affect regulation and affect recovery in depression research and treatment.

Mindfulness-based therapies such as Mindfulness-Based Stress Reduction (MBSR) and Mindfulness-Based Cognitive Therapy (MBCT) are meditation-based emotion-regulation training programs that target emotional reactivity to stress in a wide range of clinical and non-clinical populations. While MBSR was created with broad clinical and non-clinical applications, MBCT was created by cognitively oriented researchers specifically for use as depression relapse prevention (Segal, Williams, & Teasdale, 2002). MBCT assumes that among people at risk for depression, life stress leads to dysphoria, which in turn activates negative thinking patterns, which further escalate the negative affect in a cycle that gradually progresses into a full-blown depressive episode (Segal, Williams, et al., 2002; Segal et al., 1996). MBCT attempts to interrupt this process by teaching depressed patients to disidentfy with or "decenter" from negative self-evaluative or ruminative thinking patterns and interrupt the stress-induced positive feedback loop between negative affect and negative thinking patterns. Thus, although the associative network activated by stress includes both negative cognitions and negative feelings (Teasdale, 1988; Teasdale, Segal, & Williams, 1995), MBCT largely emphasizes and targets the *cognitive* reactivity component of the cognition-affect cycle (Segal, Williams, et al., 2002).

In accordance with this model, previous theoretical accounts and research have focused on cognitive

[1] Increased "autonomy" in this context means "decreased reliance on stress", but it should not be confused with the "Stress Autonomy" model which states that the decoupling of stress and depression means that stressful events are no longer capable of triggering a depression. See Monroe and Harkness 2005.

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reactivity as an important change process in MBCT. Cognitive reactivity is operationalized as the magnitude of change on the Dysfunctional Attitudes Scale (Weissman, 1979) in response to negative mood provocation or affective challenge paradigms that are intended to serve "as manipulable experimental analogues for real-world environmental stressors" (Segal et al., 1999, p. 8). Segal et al. (2006) showed that greater cognitive reactivity after cognitive behavior therapy (CBT) or antidepressant medication (ADM) predicted higher rates of relapse over the course of 18 months. Moreover, patients who recovered with CBT showed significantly less cognitive reactivity to affective challenge than those who recovered with ADM. Unlike CBT, MBCT aims to help patients hold negative thoughts (and feelings) in awareness rather diminishing them. Consistent with this, Kuyken et al. (2010) found that individuals who had undergone MBCT had *higher* cognitive reactivity than those who received ADM, but the relationship between cognitive reactivity and relapse 15 months later was decoupled in the MBCT (but not the ADM) condition. In other words, depressed patients undergoing MBCT responded with increased activation in negative thinking while in a dysphoric mood, but this increase in cognitive activation did not result in affective deterioration.[2]

However, less attention has been paid to the affective component in the cognitive-affective associative network. Emotional reactivity, or the intensity and/or duration of negative affect in response to a stressor, is an important process believed to be changed in the course of MBCT (Segal, Williams, et al., 2002). As an emotion-regulation program, MBCT teaches patients to change their relationships with both negative thoughts and emotions, to hold them in awareness, and accept them with a nonjudgmental and compassionate attitude, instead of with a secondary layer of self-referential negative evaluation that serves to exacerbate them. Indeed, Kuyken et al. (2010) showed that developing a compassionate attitude toward one's own negative thoughts and feelings mediated the effect of MBCT on depressive symptoms and relapse. As a result, the program is expected to help patients regulate their emotions more effectively in response to negative affect-producing stressors (Farb et al., 2007; Grant, Courtemanche, & Rainville, 2011; Segal et al., 1999). The application of MBCT in a broader sense, as an emotion-regulation intervention, is consistent with recent efforts to integrate cognitive, behavioral, and biological models of depression and meditation through the study of emotion and integrate MBCT for depression with the prevailing affective neuroscience/emotion-regulation models of the larger field of mindfulness and meditation research (Chambers, Gullone, & Allen, 2009; Dakwar & Levin, 2009; De Raedt & Koster, 2010; Kuyken et al., 2010; Rottenberg & Johnson, 2007; Way, Creswell, Eisenberger, & Lieberman, 2010; Williams, 2010).

Recently, MBCT has expanded beyond depression relapse prevention, and also beyond depression-specific cognitive reactivity theories into broader clinical applications and broader transdiagnostic emotion-regulation models that combine cognitive, affective, and biological approaches (Chambers et al., 2009; De Raedt & Koster, 2010; Way et al., 2010). MBCT (including the present study) has expanded into the treatment of acute or residual depression (Barnhofer et al., 2009; Britton, Haynes, Fridel, & Bootzin, 2010; Eisendrath et al., 2008; Finucane & Mercer, 2006; Kenny & Williams, 2007; Kingston, Dooley, Bates, Lawlor, & Malone, 2007; Kuyken et al., 2008; Manicavasgar, Parker, & Perich, in press; Schroevers & Brandsma, 2009; Shahar, Britton, Sbarra, Figueredo, & Bootzin, 2010; Williams et al., 2008), bipolar disorder (Williams et al., 2008), anxiety disorders (Evans et al., 2008; Finucane & Mercer, 2006; Kim et al., 2009; Lovas & Barsky, 2010; Piet, Hougaard, Hecksher, & Rosenberg, 2010; Schroevers & Brandsma, 2009), and conditions where emotional reactivity and regulation (rather than depression-specific cognitive reactivity) is the common unifying feature and treatment target (Baer, Fischer, & Huss, 2005; Foley, Baillie, Huxter, Price, & Sinclair, 2010; Oken et al., 2010; Rimes & Wingrove, 2010; Sachse, Keville, & Feigenbaum, 2011; Semple, Lee, Rosa, & Miller, 2009; van der Lee & Garssen, in press). Thus, the effects of MBCT on emotional reactivity in depression may have broad implications related to the transdiagnostic applications of MBCT specifically and mindfulness-based interventions in general.

A number of studies support the relationship between mindfulness and reduced emotional reactivity to stress, including attenuated emotional responses to threatening situations or faster recovery from transient negative affect (Arch & Craske, 2006, 2010; Brewer et al., 2009; Broderick, 2005; Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Creswell, Way, Eisenberger, & Lieberman, 2007; Erisman & Roemer, 2010; Goldin & Gross, 2010; Kaviani, Javaheri, & Hatami, 2011; Kuehner, Huffziger, & Liebsch, 2009; McKee, Zvolensky, Solomon, Bernstein, & Leen-Feldner,

[2] On the other hand, see findings by Raes, Dewulf, Van Heeringen, & Williams (2009) which found less cognitive reactivity after MBCT. Clearly, these inconsistent results regarding the role of cognitive reactivity need further study but they are not the focus of the current study.

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2007; Ortner, Kilner, & Zelazo, 2007; Pace et al., 2009; Proulx, 2008; Raes et al., 2009; Tang et al., 2007; Taylor et al., 2011; Vujanovic, Zvolensky, Bernstein, Feldner, & McLeish, 2007; Weinstein, Brown, & Ryan, 2009). For example, Arch and Craske (2006) found that undergraduate students who underwent a brief mindfulness induction (breath awareness) reported less negative emotional reactivity in response to affectively valenced slides compared to controls. Broderick (2005) found that undergraduate students assigned to a brief mindfulness meditation condition showed faster recovery from a sad mood induction compared to a distraction condition. Decreased emotional reactivity and better emotion regulation in meditators has also been reported using biological measures, including greater prefrontal inhibition of the amygdala (Brefczynski-Lewis, Lutz, Schaefer, Levinson, & Davidson, 2007; Creswell et al., 2007; Farb et al., 2007; Taylor et al., 2011; Way et al., 2010) and decreased sympathetic hyperarousal (Barnes, Treiber, & Davis, 2001; Carlson, Speca, Faris, & Patel, 2007; Maclean et al., 1994; Ortner et al., 2007; Sudsuang, Chentanez, & Veluvan, 1991).

However, none of these studies examined whether MBCT directly influences the way depressed patients respond emotionally to stress. In addition, these studies suffer from other methodological limitations: most of them used brief mindfulness induction (under 10 minutes) in undergraduate students or other non-clinical samples. Only two studies have demonstrated improved emotional reactivity (using personalized scripts) following standard (8-week) mindfulness interventions in clinical samples (social anxiety and substance abuse; Brewer et al., 2009; Goldin & Gross, 2010), but neither used a depressed sample undergoing MBCT.

Within the MBCT literature, very few studies have examined emotional reactivity to stress. Ma and Teasdale (2004) attempted to assess MBCT's effect on stress-related relapse, but was unable to "examine directly the protective effects of MBCT in the face of different severities of environmental stress because the occurrence of events of those who did not relapse was not examined" (p. 39). Recently, Kaviani et al. (2011) found that MBCT reduced depression and anxiety during a natural anticipated stressor (exam period) in a non-clinical sample of university students. Thus, at present, no studies have examined how MBCT directly influences the way depressed patients respond emotionally to stress.

In order to most fruitfully examine MBCT's effects on emotional reactivity to stress with specific relevance for depression, a number of methodological issues must be considered. First, not all stressors are equal. Segal et al. (2006) warned that the negative mood provocation method that is typically used to generate negative affect (i.e., sad music or negative slides) probably does not have the ecological validity of "being rejected by a social partner" (p. 755). Indeed, the most salient and impactful stressors and the ones that most commonly precipitate depressive episodes are interpersonal and involve social evaluation, rejection, or loss (Gunthert, Cohen, Butler, & Beck, 2007; Ingram, Miranda, & Segal, 1998; Leary, 2004). While more recent studies have recognized the importance of using social evaluative threat as the stressor rather than a generic emotional film, scripts or slides, (Creswell et al., 2007; Pace et al., 2009; Weinstein et al., 2009), none were intervention studies with clinical samples. Thus, the purpose of the present study was to investigate the effects on MBCT on emotional reactivity to an ecologically valid and provocative stressor (Trier Social Stress Test [TSST]; Kirschbaum, Pirke, & Hellhammer, 1993).

Second, the time course of emotional reactions ("affective chronometry") has been recently highlighted as important in the study of depression (Davidson, 2003; Davidson, Jackson, & Kalin, 2000). Davidson argued that "Time course variables are particularly germane to understanding vulnerability to psychopathology, as certain forms of mood and anxiety disorders may be specifically associated with either a failure to turn off a response sufficiently quickly and/or an abnormally early onset of the response" (p. 658). According to Davidson, emotional reactivity and regulation can occur at three distinct temporal windows: before, during, and after the stressor. For example, he suggested less variability in emotional reactivity during exposure to a negative affect-producing stimulus (emotion generation) and more variability in emotional reactivity after exposure to the stimulus is terminated (emotion regulation). Studies from Davidson's laboratory confirmed this by showing that people with different affective styles show different patterns of emotional reactivity only following exposure to a negative stimulus. For example, Jackson et al. (2003) found that prefrontal cortex activation asymmetry mostly explained variability in eyeblink startle magnitude following a negative stimulus, not during the stimulus. One notable shortcoming in such studies is the use of stimuli that are not ecologically valid (pictures) and the short time period in which reactivity was assessed (a few seconds after exposure to the stimulus). One purpose of the current study is to expand this research line by assessing emotional reactivity to a real-world stressor and by assessing reactivity during longer time periods following the stressor.

To summarize, the primary aim of the current study was to assess the effects of MBCT on emotion

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reactivity in order to further clarify how MBCT works and to expand the cognitive focus of MBCT into a broader emotion-regulation framework. A secondary aim was to explore whether MBCT's effect on depressive symptoms are mediated by changes in emotion reactivity. In addition, we sought to address several methodological limitations that undermined previous research on emotional reactivity by (a) assessing emotion reactivity during several time points before, during, and after the stimulus; and (b) by using an externally valid, laboratory-based stressor that is known to provoke considerable stress. We examined our hypotheses in a waitlist randomized controlled trial of MBCT within a sample of individuals with recurrent depression.

Methods

PARTICIPANTS

Participants ($n = 52$) were recruited through community advertisements for a meditation-based depression-relapse prevention program. Consistent with Kuyken et al. (2008), the target population was individuals with a recurrent form of unipolar depression in partial or full remission with varying degrees of residual symptoms, as these individuals are considered at high risk for recurrence and because previous studies have shown that MBCT is effective for more recurrent forms of depression (Ma & Teasdale, 2004; Teasdale et al., 2000). A Structured Clinical Interview for Axis I (SCID-I; First, 2002) and Axis II (SCID-II) Disorders and the Hamilton Rating Scale for Depression (HRSD; Gelenberg et al., 1990) were used to determine diagnostic eligibility. Participants met DSM-IV criteria for major depression in the last 5 years and had a lifetime history of at least three episodes, but were in partial remission during the last 8 weeks with a varying degree of residual symptoms. Partial remission was defined by a subjectively reported improvement in symptoms in the last 2 months, HRSD score ≤ 20 and the exclusion of severely depressed mood (HRSD item $1 = > 2$), severe anhedonia (HRSD item $7 = > 3$), or active suicidal ideation (HRSD item $3 = > 2$). Exclusion criteria included (a) a history of bipolar disorder, cyclothymia, schizophrenia, or other psychotic disorders, persistent antisocial behavior or repeated self-harm, borderline personality disorder, or organic brain damage; (b) current panic, obsessive-compulsive disorder, eating disorder, or substance abuse/dependence; (c) inability to read/write in English; (d) current psychotherapy; or (e) a regular meditation practice. Individuals on antidepressants were permitted to participate as long as they reported no change in medication type or dose during the 3 months prior to enrollment or during the active phase of the study.

PROCEDURE

All participants were recruited through community advertisements from January 2004 through June 2005. Eligible participants participated in MBCT groups or waitlist from July 2004 through December 2005. Following a screening interview for diagnostic eligibility, participants completed a packet of self-report questionnaires and a 3-hour laboratory-based assessment that included the TSST (Kirschbaum et al., 1993).

Participants were then block randomized to either the MBCT program or waitlist control condition in a 3:2 ratio without reference (stratification) to baseline characteristics to ensure rapid and adequate enrollment in the treatment arm. In blocks of five, opaque, sealed letter-size envelopes with treatment allocation information were shuffled and placed in identical sequentially numbered containers and presented to the patients after successful completion of baseline assessments. Treatment allocation was recorded by the first author, who was also the intervention therapist, and therefore not blind to intervention allocation. Because baseline assessments were conducted before randomization, participants and research personnel were blind to treatment conditions during this phase of the project. After 8 weeks of treatment or waitlist condition, participants completed a post-treatment questionnaire packet and returned to the laboratory to repeat the TSST. Research personnel who collected or scored any post-baseline data were also blind to treatment conditions. Waitlisted subjects entered the next available wave of the MBCT program, after completing the second assessment. The sample size was calculated to have a power of .8 to detect a medium effect size for changes in depression and anxiety symptoms. A medium effect size has been found for depression and anxiety in two meta-analyses of MBCT/MBSR (Grossman, Niemann, Schmidt, & Walach, 2004; Hofmann, Sawyer, Witt, & Oh, 2010).

The study protocol was approved by the University of Arizona Institutional Review Board, and all participants provided written informed consent for research participation. The study was conducted at the University of Arizona Department of Psychology. No adverse events occurred during the trial.

LABORATORY-BASED STRESS-INDUCTION PROCEDURE

Prior to laboratory assessment, participants completed 3 weeks of sleep diaries (to establish circadian timing). Laboratory assessments were scheduled in the late afternoon (around 5 p.m.) according to subject's circadian time because circadian timing affects stress reactivity (Dickerson & Kemeny, 2004). The TSST is a procedure that

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reliably produces moderate psychological distress in laboratory settings (Kirschbaum et al., 1993). In order to prompt evaluative self-focus, subjects delivered a speech in front of a one-way mirror and were told that a panel or judges were behind the mirror, evaluating their performance. The speech was made in front of a microphone, under two tripod-mounted 1,000-watt halogen stage lights, in the presence of two video cameras with feedback to a closed-circuit television. A judge in a white lab coat with a clipboard was seated directly in front of the subject and trained to give no social feedback. Participants were told that their performance was being recorded for later analysis. Subjects were given 5 minutes to prepare their speeches but were not allowed to use the notes they had prepared. The speech lasted for a full 5 minutes and was followed by a 5-minute serial subtraction task. If the subject paused, he or she was instructed to continue. At each laboratory assessment (before and after the treatment period), the Spielberger State Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was administered at five time points: (a) upon arrival in the laboratory, before the stressor (baseline); (b) immediately after the stressor with reference to anxiety levels *during* the stressor ("How did you feel DURING the speech?"); (c) immediately after the stressor ("How are you feeling now?"); (d) 40 minutes; and (e) 90 minutes post-stressor offset. These time points were based on the chronometry of psychological and physiological reactivity to the TSST (Dickerson & Kemeny, 2004; Kirschbaum et al., 1993; Takahashi et al., 2005). Subjects were not instructed to try to alter or regulate their emotional response, although many individuals will repair negative affective states spontaneously in the absence of instruction (Forgas & Ciarrochi, 2002; Hemenover, Augustine, Shulman, Tran, & Barlett, 2008).

A detailed, scripted manual of the TSST ensured consistent administration across sessions (Payne et al., 2006). Pre- and post-treatment TSST protocols differed only in the speech topic and the starting and subtraction numbers for the arithmetic task. All laboratory sessions were conducted by staff members who were blind to the treatment allocation or phase of the study.

MEASURES

The SCID-I (First, 2002) and the SCID-II were used to assess current and past diagnostic status at entry into the study. In order to facilitate more accurate recall for past depression, participants were asked to make a list of all past depressions and bring it with them to the interview. The list included age, duration, estimated severity/impairment, and if it was associated with an identifiable stressor. Reliability for a subset of interviews (10%) had over 90% agreement with an independent clinician for the diagnosis of MDD.

Depression History

History of past depression was assessed during the diagnostic assessment. As described above, participants were asked to describe the symptom severity and duration of each possible episode. All participants had at least three prior lifetime episodes. In cases where participants were unsure about being sufficiently symptom free between episodes to identify distinct episodes, the number of episodes was determined, in some instances, based on the clinical judgment of the interviewer. Additionally, some patients reported a large number of short episodes, whereas others reported fewer but longer episodes. Given this ambiguity about the distinctiveness of episodes and the variability in number of months of depression between patients, past depression was operationalized as the number of months over the course of their lives that patients met diagnostic criteria for a depressive episode.

Depression Symptoms

Depressive symptoms were measured with the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the HRSD-24; Gelenberg et al., 1990). The BDI is a 21-item self-report measure that assesses depressive symptomatology, with an emphasis on cognitive symptoms. The BDI is a widely used measure of depressive symptoms and has excellent psychometric properties (Beck, Steer, & Garbin, 1988; $\alpha = .81$ pre-treatment, .90 post-treatment).

The HRSD is a widely used clinician-administered interview assessment of depressive symptomatology (Gelenberg et al., 1990). The HRSD and diagnostic interviews were conducted by the first author who was trained in administering the HSRD until an adequate level of reliability (>.90) with other raters of the same version was achieved. The HRSD-24 was used for screening purposes only ($\alpha = .77$).

Emotional Reactivity to Social Stress

The STAI-Y1 (Spielberger et al., 1983) is a 20-item self-report inventory where respondents rate their current levels of negative affect on a 4-point Likert scale, ranging from 1 (*not at all*) to 4 (*very much so*). The STAI was initially intended to assess anxiety, but has been more recently determined to measure a broader type of distress, a "higher-order factor of negative affect" that incorporates both anxiety (worry, distressing thoughts) and depression (dysphoric mood and negative self-appraisal; Bieling, Antony, & Swinson, 1998; Caci, Bayle,

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Dossios, Robert, & Boyer, 2003; Gros, Antony, Simms, & McCabe, 2007). The STAI-Y demonstrates good psychometric properties, including strong internal consistency (alpha coefficients range between .86 and .95) in diverse adult and adolescent samples, adequate test–retest reliability, and convergent validity. In the current sample, internal consistency ranged from .89 to .93 and the intraclass correlation (ICC = .93) was high. Sensitivity to change, as measured by effect size (η_p^2 = .44–55), and the standardized response mean (.99–1.1) for stress induction were high (see Manipulation Check section for details).

Mindfulness Meditation Practice Logs

Participants in the MBCT group kept track of their daily mindfulness meditation (MM) practice during the 8 weeks of active treatment. Diaries included information about the type of meditation, the number of minutes practiced, and use of CD/tape. Logs for the preceding week were collected at each class meeting (see Britton et al., 2010, for details).

INTERVENTION

MBCT (Segal, Teasdale, & Williams, 2004; Segal, Williams, et al., 2002; Teasdale, 2004) is an 8-week group-based intervention that combines principles from CBT (Beck, Rush, Shaw, & Emery, 1979) and MBSR (Kabat-Zinn, 1990) using a psychoeducational and client-centered format. MBCT sessions focus on cultivating mindfulness or non-judgmental present-moment awareness of mental content and everyday activities, including sitting, lying down, breathing, walking, and other simple movements. Homework assignments consisted of practicing MM exercises with the aid of a guided audio CD and completing worksheets related to stress, automatic thoughts, and common reactions to various types of events. A session-by-session description with handouts and homework assignments is available in the MBCT manual (Segal, Williams, et al., 2002). Sessions were conducted by the first author who had more than 10 years (approximately 3,000 hours) of mindfulness practice experience and has received extensive training in delivery of the program through the Center for Mindfulness MBSR Instructor Certification Program at University of Massachusetts Medical School, and through MBCT training with Dr. Zindel Segal, the first author of the MBCT manual. Although treatment fidelity and adherence were not formally assessed, we have previously demonstrated that this MBCT intervention was efficacious in reducing residual depression symptoms, and increasing mindfulness (Mindful Attention Awareness Scale scores), compared to controls (Shahar et al., 2010).

STATISTICAL ANALYSIS

Preliminary Analysis

Before analysis, all variables were examined for normality, and no outlying cases had significant influence on the results (as assessed by Cook's distance scores). Preliminary analyses were used to describe baseline characteristics, treatment retention, and TSST manipulation checks, and to investigate any baseline group differences that might affect the main analyses.

Main Analysis

In order to examine the effect of the treatment on anxiety levels before and after MBCT, a $2 \times 2 \times 5$ mixed-model ANOVA was performed. The between-groups factor was treatment (MBCT, control), and the within-groups factors were time in relation to treatment (before treatment, after treatment) and time in relation to the TSST (before TSST, during the TSST, immediately after TSST, 40 minutes after TSST, 90 minutes after TSST). In accordance with Hamilton and Dobson (2002), we controlled for initial depression in all of our analyses, by entering baseline HRSD scores as a covariate. Data were analyzed using SPSS 17.0 software. Statistical significance was set at alpha levels <0.05, two-tailed. Results are reported as mean ± standard error (*SE*) or number/percentage unless otherwise indicated. Effect sizes were reported as partial η^2 (η_p^2; small = .01, medium = .06, large = .14; Green & Salkind, 2005).

Secondary (mediational) analysis: An SPSS Macro (Preacher & Hayes, 2008) was used in order to examine whether improvements in anxiety regulation (emotion reactivity) mediated the effects of MBCT on depressive symptoms. In order to conduct this analysis we first computed a mean anxiety score for each participant based on his or her anxiety scores on all five assessments during the TSST. We then computed anxiety change scores by subtracting the pre-treatment anxiety score from the post-treatment anxiety score. We also computed depression change scores by subtracting pre-treatment BDI scores from post-treatment BDI scores. Despite concerns that difference scores are unreliable, Rogosa and Willett (1983) showed that change scores are reliable estimates of change when individual differences in true change do exist.

Preacher and Hayes's (2008) approach is based on a bootstrapping procedure that extends Baron and Kenny's (1986) regression-based causal steps approach and the Sobel test (Sobel, 1982) for the significance level of indirect effect. In short, because the distribution of the indirect effect often deviates from normality, especially in small samples (MacKinnon, Lockwood, & Williams, 2004), the

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bootstrapping approach yields more accurate 95% confidence intervals for the indirect effect.

Results

PARTICIPANT FLOW AND SAMPLE CHARACTERISTICS

Of the 52 participants who completed baseline assessments, 23 were randomized to waitlist and 29 to MBCT. Twenty-six (90%) MBCT and 19 (82%) waitlisted participants completed all assessments (total $n = 45$). Of the 29 participants randomized to MBCT, three dropped out before the third class. Of the remaining 26, 25 (96.2%) attended at least seven of the eight sessions, one person attended six sessions, and all attended the all-day retreat. Figure 1 displays the participants' flowchart.

Sample characteristics by treatment group and completion status are displayed in Table 1. Treatment groups and completers versus non-completers did not differ on any variable including age, sex, use of antidepressant medications, depression severity, or previous months of depression.

MM PRACTICE

Outside of class, the 27 completers reported engaging in formal MM practice an average of 39.94 ± 10 minutes/day, 5.2 ± 1.2 days/week. According to the goal of 45 minutes/day, 6 days/week of formal MM practice (270 minutes/week = 100%), the mean meditation minutes across all weeks was 76 ± 24% with a range of 79 to 308 minutes/week.

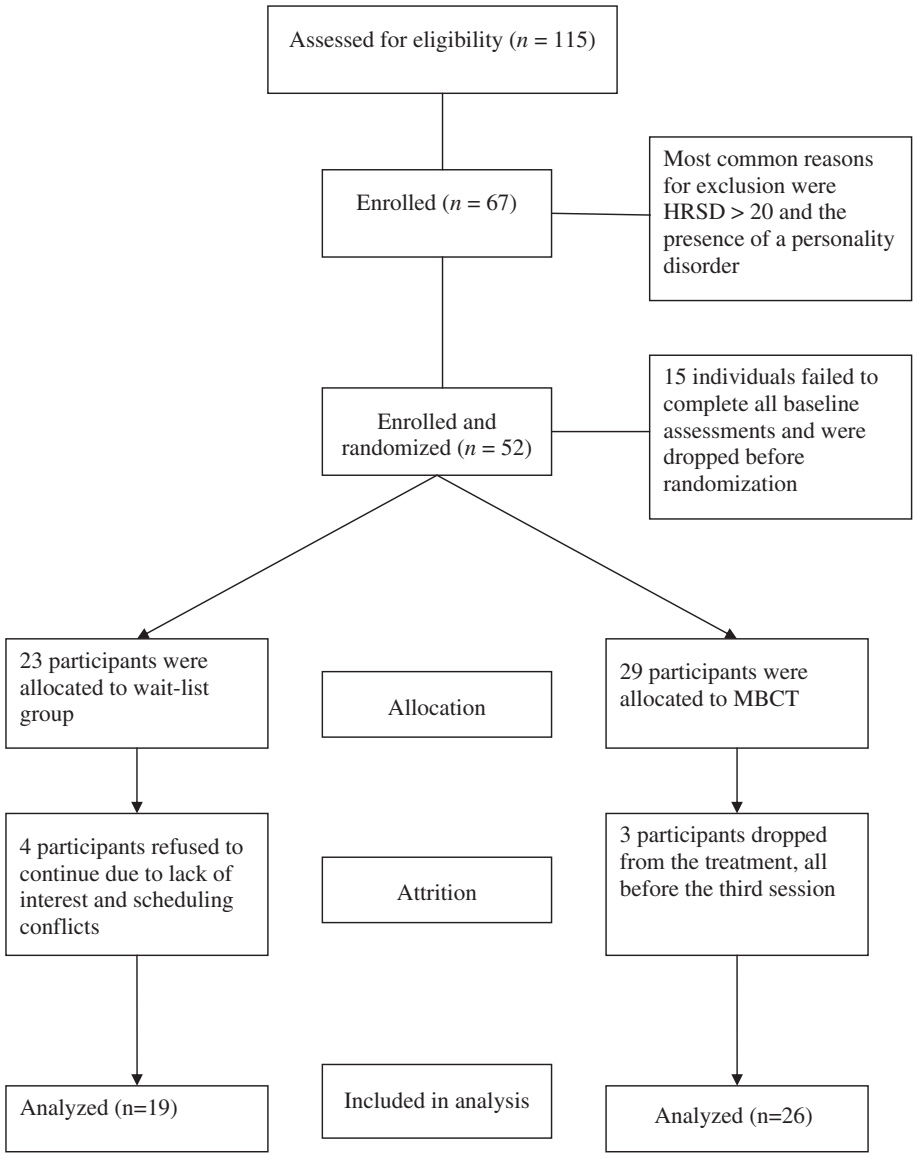

FIGURE 1 CONSORT flow diagram.

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Table 1
Baseline Characteristics of MBCT and Waitlist Samples

Characteristic	All ($n=52$)		Completers ($n=45$)	
	MBCT ($N=29$)	Waitlist ($N=23$)	MBCT ($N=26$)	Waitlist ($N=19$)
Female %	79.3	87.0	76.92	94.74
Age *M* (*SE*)	47.0 (1.39)	47.83 (2.28)	46.58 (1.52)	46.74 (2.68)
Months depressed *M*	59.55 (7.10)	61.70 (8.15)	59.85 (7.88)	61.89 (8.97)
On AD (%)	47.7	52.2	50.00	57.90
BDI *M* (*SE*)	9.09 (1.08)	9.83 (1.27)	9.10 (1.19)	10.16 (1.4)
HRSD *M* (*SE*)	10.38 (1.13)	13.17 (1.38)	10.50 (1.25)	13.32 (1.48)

Note. MBCT = Mindfulness-Based Cognitive Therapy; Months depressed = total number of months of previous depression across all episodes; AD = antidepressant medication; BDI = Beck Depression Inventory; HRSD = Hamilton Rating Scale for Depression.

MANIPULATION CHECK: TSST RELIABLY INDUCES NEGATIVE AFFECT

We conducted a series of analyses to evaluate the effectiveness of the TSST in producing negative affect/anxiety, and to assess whether repeated administration led to an attenuated response (i.e., habituation). Using the change between baseline and the report of anxiety during the speech, the TSST produced a significant increase in anxiety for all participants at both pre-, $t(44)=7.5$, $p<.001$, $\eta_p^2=.55$, and post-treatment, $t(44)=5.9$, $p<.001$, $\eta_p^2=.44$. There was no attenuation in the peak level of anxiety produced by the TSST from pre- to post-treatment assessment (STAI score during speech at pre-treatment = 53.4 ± 10.9, at post-treatment = 50.7 ± 11.12, time main effect, $F(42)=2.3$, $p=.14$).

MAIN ANALYSES: THE EFFECT OF THE TREATMENT ON EMOTIONAL REACTIVITY TO SOCIAL STRESS

The ANOVA failed to find a significant three-way interaction between treatment group, time in relation to treatment, and time in relation to task, indicating that the trajectory of anxiety scores over time was similar in both MBCT and control groups (see Figure 2).

However, a significant two-way interaction between treatment and time in relation to treatment was found, $F(1, 42)=6.20$, $p<.05$, $\eta_p^2=.13$. Simple effects analyses showed that whereas average anxiety rates (collapsing across time in relation to task) decreased in the MBCT group, they did not decrease in the control group (see Table 2).

In order to further understand the exact manner in which MBCT decreased anxiety rates, the difference between pre-treatment anxiety and post-treatment anxiety was examined at each TSST time point for the MBCT group and the control group separately. In the control group, a significant increase in pre-speech anxiety was found, followed by non-significant differences at each other measurement points. In contrast, in the MBCT group, no difference was found in pre-speech anxiety and anxiety during the speech, but significant decreases in anxiety rates were recorded at each of the other three post-speech measurement points. Means and *F* values are presented in Table 2.

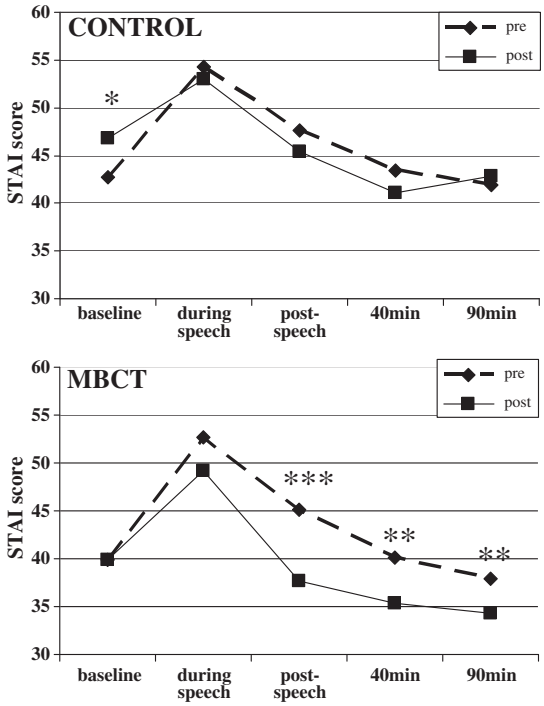

FIGURE 2 Anxiety scores pre- and post-treatment displayed separately for controls (top) and MBCT (bottom). *Note.* MBCT = Mindfulness-Based Cognitive Therapy; STAI = Spielberger State Anxiety Inventory; TSST = Trier Social Stress Test; Baseline = immediately prior to the TSST; During speech = anxiety level while the TSST was occurring; Post-speech = anxiety level at the conclusion of the TSST; 40 min. = anxiety level approximately 40 minutes after the TSST had concluded; 90 min. = anxiety level approximately 90 minutes after the TSST had concluded. *** $p<.005$, ** $p<.01$, * $p<.05$.

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Table 2
Mean (*SE*) Anxiety Levels Before, During, Immediately After, 40 Minutes After, and 90 Minutes After the TSST, as a Function of the Time of Measurement (Pre- or Post-treatment) and the Type of Treatment (MBCT, Control)

	MBCT			Control		
	Pre-Tx	Post-Tx	*F*(1, 42)	Pre-Tx	Post-Tx	*F*(1, 42)
Baseline	40.77	40.39	0.05	41.40	46.02	4.78*
	(1.54)	(1.80)		(1.90)	(2.22)	
During speech	52.88	49.53	3.01	54.07	52.37	0.51
	(2.24)	(2.13)		(2.75)	(2.62)	
Post-speech	45.72	37.92	28.36***	46.92	45.02	0.87
	(2.02)	(1.80)		(2.48)	(2.21)	
40 min.	40.94	35.44	11.19**	42.09	40.89	0.35
	(1.96)	(1.52)		(2.41)	(1.87)	
90 min.	38.55	34.46	8.34**	40.95	42.63	0.94
	(2.07)	(1.88)		(2.54)	(2.31)	
Overall	43.77	39.55	13.57***	45.04	45.39	0.06
	(1.60)	(1.53)		(1.96)	(1.89)	

Note. MBCT = Mindfulness-Based Cognitive Therapy; TSST = Trier Social Stress Test; Pre-Tx = before treatment (week 0); Post-Tx = after treatment (week 9); Baseline = immediately prior to the TSST; During speech = anxiety level while the TSST was occurring; Post-speech = anxiety level at the conclusion of the TSST; 40 min. = anxiety level approximately 40 minutes after the TSST had concluded; 90 min. = anxiety level approximately 90 minutes after the TSST had concluded. *** $p<.005$, ** $p<.01$, * $p<.05$.

SECONDARY MEDIATIONAL ANALYSIS

The effect of treatment on change in depression (*c* path) was significant ($\beta = -.42$, $p<.01$). In addition, the effect of treatment on change in anxiety (*a* path) was marginally significant ($\beta = -.27$, $p = .07$), and the direct effect of change in anxiety on change in depression (*b* path) was significant ($\beta = .39$, $p<.01$). When change in anxiety was taken into account, the direct effect of treatment on depression (*c*' path) decreased in relation to the overall effect (*c* path), although it stayed significant ($\beta = -.31$, $p<.05$). Bootstrapped .95 confidence intervals for the mediated effect did not include zero, indicating that the indirect effect of MBCT on depressive symptoms through changes in anxiety was significant. Overall, the results suggested that improvements in anxiety regulation reliably (although partially) mediated the effects of MBCT on depressive symptoms.

Discussion

This study assessed the effects of MBCT on emotional reactivity to a laboratory-based social evaluative threat in a sample with partially remitted recurrent depression. The main results were the following: MBCT was associated with an overall decrease in emotional reactivity. Overall anxiety levels (collapsed across all TSST time points) decreased significantly for the MBCT group, but not controls, when compared to pre-treatment levels. A closer examination of specific assessment points during the TSST revealed that this decreased emotional reactivity in the MBCT group was specific to the post-stressor recovery phase. The MBCT group showed an attenuation of negative affect/anxiety at all post-stressor time points compared to pre-treatment baseline, whereas post-speech anxiety levels did not change from pre-treatment among participants in the control group. Before treatment, anxiety levels in all participants remained elevated following the speech, and did not return to pre-speech baseline levels until 40 minutes after the speech had concluded. After treatment, anxiety levels in the MBCT group returned to baseline levels immediately after the speech had concluded (i.e., 40 minutes earlier than before treatment). These data suggest that MBCT training is associated with a faster affective recovery from potent negative affect-producing stressors. Furthermore, the mediational analysis showed that MBCT's positive effects on depressive symptoms was partially mediated by these improvements in anxiety regulation.

Examination of the pre-stressor time point (i.e., anticipatory anxiety before the speech) also suggests a beneficial effect of MBCT participation. The control group showed an increase in pre-speech anxiety at post-treatment compared to pre-treatment, which indicates an increased sensitization to stress in the control group in the form of more anticipatory anxiety (i.e., larger emotional response to same stressor). The MBCT group experienced similar levels of pre-speech anxiety before and after treatment, that is, they did not experience stress sensitization that the control participants experienced. These findings suggest that MBCT may help depressed patients to better regulate their anticipatory anxiety before anticipated stressors. We believe that the findings regarding better regulation of anticipatory anxiety are particularly important because anticipatory anxiety affects the intensity of response to subsequent stressors and plays an

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important role in anxiety disorders (Grillon, Ameli, Foot, & Davis, 1993) and mortality risk (Peterson, Seligman, Yurko, Martin, & Friedman, 1998).

Although MBCT was associated with an overall decrease in stress-related anxiety, these changes were specific to pre- and post-stressor anxiety and not anxiety *during* the stressor. Similarly, other studies (Farb et al., 2010; Kuyken et al., 2010) also found no difference in the magnitude of immediate emotional response to provocation following 8 weeks of mindfulness training or control treatment. Rather, increased *duration* of negative affect following a stressor, rather than the initial intensity, is associated with depression risk (Cohen et al., 2005; Gillihan et al., 2010). Similarly, Jackson et al. (2003) found that an affective style characteristic of depression (less left-side activation in the prefrontal cortex) was associated with increased emotion reactivity *after* picture presentation but not *during* picture presentation. This is consistent with the idea that emotional reactions to stress, including negative affect and physiological arousal, are adaptive (up to a point) and need not be eliminated (Mayne, 2001; McEwen & Seeman, 1999). Rapid recovery after the stressor has passed is also adaptive, as prolonged arousal and negative affect can deplete the organism's resources and increase risk for depression. Our data suggest that mindfulness training may exert a nuanced effect that is specific to the horizontal time course (chronometry) rather than a generalized suppression or blunting of the intensity or amplitude of emotional responses (Taylor et al., 2011).

These data have clinical relevance for the treatment of depression specifically, for the treatment of conditions where poor affect regulation is a central target (addictions), and for mindfulness and meditation-based interventions in general. In depression, the potentiation or prolongation of negative affect, which may be indicative of poor prefrontal control of the amygdala, and/or activation of negative cognitive schemas (cognitive reactivity; De Raedt & Koster, 2010) is associated with higher likelihood of current and future depression as well as poor treatment response (Cohen et al., 2005, 2008; Myin-Germeys et al., 2003; Pine et al., 2001) and is therefore a central treatment target. Our data suggests that MBCT can effectively target such reactivity and that this effect at least partially mediates improvement in residual depressive symptoms that reliably predicts relapse.

In addition to depression, a broad range of emotional disturbances characterized by persistent negative affect are associated with poor emotional regulation, high emotional reactivity, and/or poor prefrontal control (Baxter et al., 1989; Bench, Friston, Brown, Frackowiak, & Dolan, 1993; Clark, Chamberlain, & Sahakian, 2009; Couyoumdjian et al., 2009; Hemenover, 2003; Mayberg et al., 1999; Meyer et al., 2004; Siegle & Hasselmo, 2002). It has been proposed that the broad therapeutic effects of mindfulness meditation are mediated by strengthening prefrontal attention and emotion-regulation systems (Chambers et al., 2009; Creswell et al., 2007; Davidson & Lutz, 2008; Hofmann & Asmundson, 2008; Teasdale et al., 1995; Way et al., 2010). Davidson (2003) hypothesized that the greater prefrontal control of the amygdala (Brefczynski-Lewis et al., 2007; Creswell et al., 2007; Farb et al., 2007; Goldin & Gross, 2010; Taylor et al., 2011; Way et al., 2010), or the decoupling from self-referential elaboration (Farb et al., 2007, 2010; Grant et al., 2011; Taylor et al., 2011) would result in greater capacity to regulate negative emotion and specifically "to decrease the duration of negative affect once it arises" (p. 662), in other words, hasten affective recovery.

> Excessive identification with the negative emotion should result in a perseveration or lingering of the negative affect, following the offset of the acute elicitor. We might thus expect that the largest temporal region during which a transformation in the affective reaction might occur is in the post-stimulus recovery period following the offset of a negative stimulus. In other words, meditation training should speed the recovery following the offset of a negative stimulus. (Davidson, 2010, p. 10).

The findings from this study add to the growing support for these emotion-regulation models of mindfulness, and integrate MBCT for depression within larger affective neuroscience models of meditation.

STRENGTHS AND LIMITATIONS

This study is the first to use a potent, standardized laboratory-based social evaluative stressor before and after a randomized controlled trial of MBCT in a depressed population. This study also addressed the emotional component of MBCT's theoretical framework, which may have broader applications than a depression-specific cognitive reactivity focus. At the same time, although the current study demonstrates that MBCT reduces emotional reactivity, it does not address the mechanism of action, or how this reduction is accomplished. It is hypothesized that this therapeutic target is achieved through teaching patients to hold their thoughts and feelings in awareness while adopting a patient, compassionate, and non-judgmental attitude toward these feelings. Kuyken et al. (2010) findings that self-compassion mediates MBCT's effects on outcome support this hypothesis. Because MBCT uses a variety of meditative techniques, it is difficult to sort out which techniques are related to which outcomes, so future studies may also want to consider dismantling designs

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(Kuyken et al., 2010; Murphy, Cooper, Hollon, & Fairburn, 2009).

The present study has several other limitations, most notably the small sample size and the limited statistical power. The specificity of the sample (partially remitted recurrent depression) is both a strength and a limitation: Individuals with three or more episodes and residual symptoms represent the population that most benefits from MBCT (Ma & Teasdale, 2004; Teasdale et al., 2000) as well as those at highest risk for recurrence. However, the high number of exclusion criteria may limit the ability to generalize to more severely depressed samples or other clinical populations. The use of an 8-week mindfulness course limits the ability to speculate on the effects of other forms of meditation or the effects of longer durations of training. The classic limitations of a waitlist control design include the possibility of expectancy effects and differential attrition. While the MBCT group's previously reported increase in mindfulness and improvement in depression is consistent with other studies where high-quality MBCT was administered, checks on competency (Crane, Kuyken, Hastings, Rothwell & Williams, 2010) and adherence (Segal et al., 2002) should be included in future research to ensure treatment fidelity.

Because self-report measures, like the STAI, are sensitive to bias and demand characteristics, use of objective measures would lend convergent validity to these findings. Furthermore, use of continuous, rather than repeated, measures would yield more fine-grained information about the time course of affective responding. While the current study suggests that MBCT decreases emotional reactivity to social stress, the clinical significance is unknown. Given that prolonged negative affect in response to stress is a marker of depression vulnerability (Cohen et al., 2005; Gillihan et al., 2010) and treatment response (Cohen et al., 2008; Davidson et al., 2002), future research should investigate whether this decreased emotional reactivity predicts relapse/sustained recovery from depression, as well as the relationship between and the respective contributions of cognitive and emotional reactivity, using appropriate designs (Kazdin, 2007; Kraemer, Kiernan, Essex, & Kupfer, 2008; Kraemer, Wilson, Fairburn, & Agras, 2002).

Conclusion

In conclusion, this study suggests that mindfulness meditation training is associated with decreased emotional reactivity in the face of a negative affect-producing stressor and that this improvement in emotional reactivity is at least partially responsible for the program's effect on depressive symptoms. By providing evidence of faster affective recovery in a (stress-sensitive) chronically depressed sample, this study strengthens the empirical basis for applying mindfulness-based approaches to depressed samples, as well as other conditions with poor affect regulation.

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