



## How does mindfulness-based cognitive therapy work?☆

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### ABSTRACT

Mindfulness-based cognitive therapy (MBCT) is an efficacious psychosocial intervention for recurrent depression (Kuyken et al., 2008; Ma & Teasdale, 2004; Teasdale et al., 2000). To date, no compelling research addresses MBCT's mechanisms of change. This study determines whether MBCT's treatment effects are mediated by enhancement of mindfulness and self-compassion across treatment, and/or by alterations in post-treatment cognitive reactivity. The study was embedded in a randomized controlled trial comparing MBCT with maintenance antidepressants (mADM) with 15-month follow-up (Kuyken et al., 2008). Mindfulness and self-compassion were assessed before and after MBCT treatment (or at equivalent time points in the mADM group). Post-treatment reactivity was assessed one month after the MBCT group sessions or at the equivalent time point in the mADM group. One hundred and twenty-three patients with  $\geq 3$  prior depressive episodes, and successfully treated with antidepressants, were randomized either to mADM or MBCT. The MBCT arm involved participation in MBCT, a group-based psychosocial intervention that teaches mindfulness skills, and discontinuation of ADM. The mADM arm involved maintenance on a therapeutic ADM dose for the duration of follow-up. Interviewer-administered outcome measures assessed depressive symptoms and relapse/recurrence across 15-month follow-up. Mindfulness and self-compassion were measured using self-report questionnaire. Cognitive reactivity was operationalized as change in depressive thinking during a laboratory mood induction.

MBCT's effects were mediated by enhancement of mindfulness and self-compassion across treatment. MBCT also changed the nature of the relationship between post-treatment cognitive reactivity and outcome. Greater reactivity predicted worse outcome for mADM participants but this relationship was not evident in the MBCT group.

MBCT's treatment effects are mediated by augmented self-compassion and mindfulness, along with a decoupling of the relationship between reactivity of depressive thinking and poor outcome. This decoupling is associated with the cultivation of self-compassion across treatment.

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Without ongoing treatment people suffering recurrent depression experience relapse/recurrence at rates as high as 80%, even

after successful acute treatment (Kupfer et al., 1992). The majority of the burden attributable to depression could be offset through interventions aimed at the prevention of relapse/recurrence (Vos et al., 2004). Mindfulness-based Cognitive Therapy (MBCT) is a promising psychosocial group-based relapse prevention program (Segal, Williams, & Teasdale, 2002). Two randomized controlled trials suggest that MBCT produces superior outcomes compared with usual care typically comprising routine monitoring in primary care (Ma & Teasdale, 2004; Teasdale et al., 2000). A more recent study suggests MBCT produces comparable outcomes to maintenance antidepressant medication (mADM) (Kuyken et al., 2008).

☆ This paper was written by Willem Kuyken and Tim Dalgleish on behalf of the Exeter MBCT Trial team. Sarah Byford, Rod Taylor & Ed Watkins were co-investigators, Emily Holden and Kat White were research staff, Alison Evans was a trial therapist, Sholto Radford completed his MSc on archival data and John Teasdale advised on the design, conduct and analysis of this study.

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Even though MBCT was developed from translational research into mechanisms of depressive relapse/recurrence (Segal et al., 2002), there are as yet no compelling studies of how it works.

MBCT's theoretical premise is that depressive relapse is associated with the reinstatement of negative modes of thinking and feeling that contribute to depressive relapse and recurrence (Segal et al., 2002). This 'reactivated' network of negative thoughts and feelings can perpetuate into a depressive episode. Laboratory studies support this model by showing that recovered depressed patients revert to a depressive information processing style following a sad mood induction (for a review see Lau et al., 2004). Following successful treatment for depression, those patients showing greater reactivation of dysfunctional thinking styles in response to a sad mood provocation are at the highest risk of relapse over an 18 month period (Segal et al., 2006). Moreover, patients who recovered with cognitive behavior therapy (CBT) showed significantly less cognitive reactivation than those recovered with ADM. Attenuating the reactivation of dysfunctional thinking styles may therefore represent one mechanism by which CBT helps prevent depressive relapse.

MBCT targets cognitive reactivation (Segal et al., 2002). Mindfulness skills are taught as a means to note distressing thoughts and feelings, hold such experiences in awareness, and cultivate acceptance and self-compassion so as to break up associative networks and offset the risk of relapse (Segal et al., 2002). This dimension of mindfulness that involves meeting distressing thoughts and feelings with kindness, empathy, equanimity and patience is woven into mindfulness-based applications and is thought to be crucial to the change process (Feldman & Kuyken, *in press*). Intentional attention is learned in the first three MBCT sessions using a range of core mindfulness practices (the body scan, mindful movement and mindfulness of the breath). As well as developing attention, these early sessions highlight habitual patterns of reactivity that arise during meditation (e.g., intrusive negative thoughts) and the associated aversion and judgments (e.g., "I am no good at this, I am just more aware of how badly I feel"). As the person learns mindfulness skills, s/he learns to give less authority to self-judgment and blame – the fuel for depressive thinking – and to respond to these states with compassion; in short to step out of habitual unhelpful patterns of thinking (Feldman & Kuyken, *in press*). Elucidating these putative mechanisms of MBCT action will improve theoretical understanding of how this relatively new treatment works and provide the opportunity to enhance efficacy via emphasis of these mechanisms.

An important first step in establishing mechanisms of action is to identify mechanism variables that *mediate* the effects of a given treatment on outcome (Kazdin, 2007). In other words, those mechanisms that are (1) differentially altered by the treatment; and (2) that explain all or part of the effect of treatment on outcome. The present study sought to identify mediators of MBCT's effects in a randomized controlled trial (RCT) of MBCT versus mADM for the prevention of depressive relapse/recurrence (Kuyken et al., 2008).

The trial has been described in full previously (Kuyken et al., 2008), but in brief we recruited 123 people with a history of three or more episodes of depression, currently in full or partial remission. Participants were all on a therapeutic dose of mADM and were interested in learning a psychosocial approach to staying well and to discontinuing their medication. Following the baseline assessment, they were randomized to groups that involved (1) continuing their medication over 15 months (the control arm) or (2) taking part in an MBCT course and tapering their ADM medication (the experimental arm). From the baseline assessment participants were followed up at 3 monthly intervals for 15 months by researchers blind to treatment condition. The trial showed that MBCT was not significantly different from mADM in terms of relapse (47% versus 60%), but produced significantly better

outcome in terms of self-reported and observer-rated depressive symptoms at 15-month follow-up (Kuyken et al., 2008).

Here, we asked three theoretically-driven questions: 1) Was better outcome in our trial mediated by greater improvement in mindfulness skills and self-compassion over the course of treatment? 2) Did MBCT, relative to mADM, attenuate the toxic relationship between reactivation of dysfunctional thinking (measured post-treatment) and later outcome (Segal et al., 2006)? 3) Was any such attenuation a function of improvement in mindfulness and self-compassion during the treatment? Prior to mediation analysis, we conducted exploratory moderation analyses for pre-randomization demographic and depression variables.

## Method

### Design

This mechanisms study was embedded in an RCT comparing MBCT (with discontinuation of ADM) to mADM (Kuyken et al., 2008). MBCT was delivered as a manualized, group-based training program designed to enable patients to learn mindfulness and other skills that prevent depression recurrence (Segal et al., 2002). The MBCT program involved a one-to-one orientation session with the therapist followed by eight weekly two hour sessions over approximately two months and four follow-up sessions spread out over approximately one year. MBCT therapists underwent an extensive training and supervision regime and high levels of competency and adherence were demonstrated by raters independent of the trial team (see Kuyken et al., 2008). MBCT participants were supported in discontinuing mADM by their primary care physician. The mADM arm involved patients remaining on a maintenance dose of ADM throughout the follow-up period, and generally this was the same ADM that they were on when they were recruited to the trial. Adherence was assessed at each follow-up point and if any adherence problems were picked up the primary care physician was alerted. Over the course of the follow-up, mADM adherence was shown to be high (see Kuyken et al., 2008).

To establish mediation requires attention to several key aspects of study design (Kazdin, 2007; Kraemer, Wilson, Fairburn, & Agras, 2002; Murphy, Cooper, Hollon, & Fairburn, 2009). First, ideally, MBCT must be compared with a treatment that works but not through the same mechanism of action, for example medication (Garatt, Ingram, Rand, & Sawalani, 2007; p. 227), thus allowing a test of effects specific to MBCT. Second, assessment of change in the hypothesized mediator must occur *during* MBCT and *before* the assessment of outcome. As MBCT targets relapse prevention, a design that tracks relapse after the end of MBCT is ideal. A robust marker for relapse is severity of depressive symptoms (Judd et al., 1999). Moreover, in the original trial MBCT produced greater change in depression severity at 15-month follow-up than did mADM (Kuyken et al., 2008). Therefore, severity of depressive symptoms and depressive relapse/recurrence over 15 months of follow-up were the dependent variables in our mediation/moderation analyses. Finally, the design requires that all those in the intervention arm received an adequate dose of the intervention to properly test the hypothesis that MBCT's impact on the hypothesized mechanisms (mindfulness, self-compassion and reactivity) mediates outcome.

Hypothesized moderators were assessed at baseline prior to randomization. Mediators were assessed as follows. Changes in self-compassion and in mindfulness skills were computed from Baseline to post-MBCT (i.e., 1 month after the end of MBCT or the equivalent time in the mADM arm). Mirroring the work of Segal and colleagues, cognitive reactivity was assessed only once, post-MBCT (Segal et al., 2006). Outcome in terms of relapse was assessed through the follow-up period up until 15 months after Baseline and

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