Metacognitive Awareness and Prevention of Relapse in Depression: Empirical Evidence

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Metacognitive awareness is a cognitive set in which negative thoughts/feelings are experienced as mental events, rather than as the self. The authors hypothesized that (a) reduced metacognitive awareness would be associated with vulnerability to depression and (b) cognitive therapy (CT) and mindfulness-based CT (MBCT) would reduce depressive relapse by increasing metacognitive awareness. They found (a) accessibility of metacognitive sets to depressive cues was less in a vulnerable group (residually depressed patients) than in nondepressed controls; (b) accessibility of metacognitive sets predicted relapse in residually depressed patients; (c) where CT reduced relapse in residually depressed patients, it increased accessibility of metacognitive sets; and (d) where MBCT reduced relapse in recovered depressed patients, it increased accessibility of metacognitive sets. CT and MBCT may reduce relapse by changing relationships to negative thoughts rather than by changing belief in thought content.

Growing evidence suggests that cognitive therapy (CT; Beck, Rush, Shaw, & Emery, 1979) reduces relapse and recurrence in major depression. Outpatients who recover following treatment of depression by CT show less relapse or need for treatment than do patients who recover with antidepressant medication and are then withdrawn from pharmacotherapy (Blackburn, Eunson, & Bishop, 1986; Evans et al., 1992; Shea et al., 1992; Simons, Murphy, Levine, & Wetzel, 1986). CT following recovery with pharmacotherapy can also reduce relapse and recurrence (Fava, Grandi, Zielezny, Rafanelli, & Canestrari, 1996; Fava, Rafanelli, Grandi, Conti, & Belluardo, 1998). In patients responding only partially to antidepressant medication, the addition of CT to clinical management and continuing antidepressant medication can significantly reduce relapse (Paykel et al., 1999).

Although it is clear that CT can reduce depressive relapse, the processes through which this is achieved are not well understood. The original cognitive model underlying CT for depression suggested that vulnerability to major depression depended on certain persistent underlying dysfunctional attitudes or assumptions, particularly those that involved a dependence of self-worth on approval from others or on the success of activities (e.g., Beck, Epstein & Harrison, 1983). From this perspective, reduction in relapse following CT would be seen as the result of CT reducing those dysfunctional attitudes. This hypothesis has received little support (Barber & DeRubeis, 1989); when CT has produced significantly better long-term outcomes than pharmacotherapy, the two treatments often do not differ on posttreatment measures of dysfunctional attitudes such as the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978). (See Simons, Garfield, & Murphy, 1984.)

In response to such negative findings, it has been suggested that vulnerability to depression, and relapse, depend not so much on relatively enduring trait-like dysfunctional attitudes as on the patterns of negative thinking that become accessible and activated in mildly depressed mood (Persons & Miranda, 1992; Teasdale,
1983, 1988). Increasing empirical evidence supports this view (e.g., Ingram, Miranda, & Segal, 1998). On the basis of this more dynamic approach to understanding cognitive vulnerability to depression, Teasdale and colleagues (Teasdale, 1997a, 1997b; Teasdale & Barnard, 1993, pp. 225–245; Teasdale, Segal, & Williams, 1995) have suggested an alternative account of the way that CT reduces depressive relapse and recurrence.

This account suggests that CT leads to changes in the patterns of negative thinking that are activated by depressed mood. As a result of these changes, those patterns of negative thinking are less likely, following CT, to produce further depression and lead to relapse. Consistent with this suggestion, Segal, Gemar, and Williams (1999) reported that, when the DAS was administered following a dysphoric mood induction, posttreatment DAS scores were significantly less following CT than pharmacotherapy. Such scores also predicted relapse.

The account proposed by Teasdale and colleagues (Teasdale, 1997a, 1997b; Teasdale & Barnard, 1993; Teasdale et al., 1995) suggests that CT leads to changes in the cognitive sets patients use to interpret and relate to depressive symptoms, thoughts, and feelings at times of potential relapse. According to this analysis, patients, in implementing the behavioral and cognitive strategies of CT, learn to relate differently, more functionally, to depressive symptoms and problematic situations. Frequently repeated, this creates a store of representations in memory encoding new relational links to depression. The creation and storage in memory of cognitive sets encoding such alternative perspectives and relationships to depressive experience, it is suggested, mediates the therapeutic effects of CT. At times of potential relapse, access to these more functional sets preempts access to depressogenic sets and so reduces the risk that mild dysphoric states will escalate to major depression.

Particular importance has been attached (Moore, 1996; Teasdale, 1997b; Teasdale et al., 1995) to the shift in cognitive set known as “decentering” or “disidentification,” in which, rather than simply being their emotions, or identifying personally with negative thoughts and feelings, patients relate to negative experiences as mental events in a wider context or field of awareness. For example, a patient’s perspective on thoughts and feelings of worthlessness might change from one in which they are experienced as the “reality by which I am condemned” to one in which they are experienced more as “passing thoughts and feelings that may or may not have some truth in them.” It is suggested that, in CT, as a result of repeatedly identifying negative thoughts as they arise and standing back from them to evaluate the accuracy of their content, patients shift to a decentered perspective on negative thoughts and feelings. Experienced and interpreted through such a decentered cognitive set at times of potential relapse, negative thoughts and feelings will have different cognitive, emotional, and behavioral consequences than if they are experienced and interpreted as “me” or “reality.” In particular, reinstatement of a decentered set at such times will mean that negative thoughts and feelings reactivated by dysphoria will be less likely to lead to the escalation of dysphoria to major depression and relapse.

The importance of distancing or decentering has previously been recognized in discussions of CT (e.g., Beck et al., 1979), but usually as a means to the end of changing thought content rather than, as the above analysis suggests, the primary mechanism of therapeutic change. Others have, however, suggested a more central role for decentering (e.g., Ingram & Hollon, 1986). We refer to the process of experiencing negative thoughts and feelings within a decentered perspective as metacognitive awareness and suggest that CT creates a store of memories in which depressive phenomena have been experienced within a metacognitive perspective. We propose that, following CT, the increased accessibility of metacognitive sets in response to negative thoughts and feelings at times of potential relapse makes it more likely that those thoughts and feelings will be experienced simply as passing events in the mind and that this shift in relationship mediates the effects of CT in preventing relapse.

Our aim in the present studies was to test the hypotheses, emerging from the above analysis, that reduced metacognitive awareness is associated with vulnerability to further major depression and that CT reduces depressive relapse by increasing metacognitive awareness, so reducing vulnerability. A new measure of metacognitive awareness was developed and used in three studies:

1. Study 1 focused on the hypothesis that vulnerability to further major depression is associated with reduced accessibility of metacognitive sets in relation to negative thoughts and feelings. Patients known to be at high risk of depressive relapse were compared with nondepressed controls on a specially developed measure of metacognitive awareness in autobiographical memories.

2. Study 2 investigated further the hypothesis that vulnerability to onset of depression is associated with reduced accessibility of metacognitive sets in response to depression-related cues. In it we examined the extent to which baseline metacognitive awareness predicted risk of relapse in depressed patients. Study 2 also examined the hypothesis that CT, when it reduces relapse, increases accessibility of metacognitive sets to negative thoughts and feelings.

3. Study 3 examined the hypothesis that psychological interventions (other than CT) that increase metacognitive awareness will also reduce relapse. We tested the prediction that a treatment program primarily directed to changing patients’ relationships to their negative thoughts and feelings (rather than to changing patients’ belief in the content of their negative thoughts) will both increase metacognitive awareness and reduce relapse.

Study 1

The above analysis suggests that vulnerability to recurrence of major depression, and relapse, is related to the relative accessibility of different cognitive sets from memory in response to depression-related negative thoughts and feelings. Specifically, it predicts that, compared with less vulnerable individuals, patients who are known to be at high risk for further onset of depression will be less likely to access a metacognitive perspective on depressive experience. Patients still experiencing residual symptoms of depression following treatment with antidepressant medication are known to be at high risk for future episodes (Paykel et al., 1995). Study 1 tested the prediction that patients with residual depression would show lower levels of metacognitive awareness than age- and gender-matched nondepressed controls.

As no satisfactory measure already existed, we developed a new measure of metacognitive awareness. To explain the rationale of
this measure, it may be helpful to clarify further the concept of metacognitive awareness. In particular, it is important to distinguish metacognitive awareness from metacognitive belief, as the latter has figured prominently in recent theorizing on emotional disorders and their psychological treatment (e.g., Wells, 1999). Metacognitive belief refers to how much individuals believe particular thoughts about cognition to be true (e.g., “worrying is dangerous for me”) and is concerned with thoughts about thoughts or feelings. The extent of belief in such thoughts could, in principle, be rated using appropriate questionnaire items.

By contrast, metacognitive awareness refers to the way that negative thoughts and feelings are experienced as they arise. In particular, metacognitive awareness refers to the extent to which thoughts, for example, are experienced as thoughts (mental events) rather than as aspects of self or direct reflections of truth. As such, metacognitive awareness may not be easily measured by questionnaire items. For this reason, we developed a measure of metacognitive awareness by adapting paradigms previously used in autobiographical memory research. We assumed that the way individuals’ thoughts and feelings are experienced would be determined by the cognitive sets they access from memory; accessing a metacognitive set would make it more likely that thoughts and feelings are experienced as mental events. For an individual at a given time, the accessibility of metacognitive sets in response to negative thoughts and feelings is a function of the availability of such sets in memory and the prevailing context. From the point of view of understanding recurrence and relapse of depression and their prevention, the accessibility of metacognitive sets in the context of dysphoria is of central importance. To measure this, we created appropriate contexts using emotive vignettes, asked for recall of autobiographical memories cued by the dysphoric feelings evoked by the vignettes, and, for each memory, obtained detailed descriptions of how the individual had related to negative thoughts and feelings in that memory. From those descriptions, we could infer the cognitive set that prevailed in relation to negative thoughts and feelings at the time of the remembered event. We assumed that the extent to which the inferred cognitive sets demonstrated metacognitive awareness in the memories most readily accessed by dysphoric vignettes reflected the accessibility of metacognitive sets to negative thoughts and feelings in the context that was of most interest to us.

Method

Participants

Depressed patients. The patient sample in Study 1 was a subset of the sample in Study 2 (see below). Forty psychiatric outpatients (20 consecutive recruits at each of two treatment sites in a clinical trial of CT for residual depressive chronicity; Paykel et al., 1999), ages 20 to 65 years, had satisfied Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.; DSM–III–R; American Psychiatric Association, 1987) major depression criteria within the past 18 months but not within the past 2 months, with residual symptoms scoring at least 8 on the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and at least 9 on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Patients with a history of bipolar disorder, cyclothymia, schizoaffective disorder, drug or alcohol dependence, persistent antisocial behavior or repeated self-harm, DSM–III–R dysthymia with an onset before age 20 years, borderline personality, learning disability, organic brain damage, or any other primary axis I disorder at the time of the index illness were excluded, as were patients currently receiving formal psychotherapy or who had previously received more than 5 CT sessions.

Patients had to have been taking antidepressant medication for 8 or more weeks, with 4 or more weeks at a daily dose at least equivalent to 125 mg of amitriptyline.

Nondepressed controls. Twenty participants, ages 21–65 years, with BDI score <10, similar in age and gender distribution to patients, were recruited from a volunteer panel.

Measures

HRSD. The 17-item HRSD, an interview measure of depressive symptoms, has good reliability and validity (Rabkin & Klein, 1987).

BDI. The BDI is a 21-item self-report measure of depressive symptoms with good psychometric properties (Rabkin & Klein, 1987).

Measure of Awareness and Coping in Autobiographical Memory (MACAM). The MACAM (Moore, Hayhurst, & Teasdale, 1996) measures metacognitive awareness (the cognitive set in which negative thoughts and feelings are seen as passing mental events rather than aspects of self) in the autobiographical memories accessed by depression-related cues. The MACAM is intended to analogize the situation identified as critical, in our analysis, in determining whether mildly depressing experiences escalate to more persistent and severe depressions. It is assumed that the cognitive sets in relation to negative thoughts and feelings that can be inferred in the autobiographical memories most easily accessed by vignettes of mildly depressing situations reflect the cognitive sets most likely to be accessed in real world situations preceding onset of major depression. Descriptions of eight mildly depressing situations, recorded in a flat, depressing tone of voice, are presented by tape recorder (e.g., “You are out shopping in town at the weekend and you have arranged to meet a friend for a cup of tea in the cafe in a department store. You get there in good time and wait outside. It is very busy and the crowd is spilling out of the door, which makes you feel a bit edgy. You wish your friend would hurry up and arrive. The time you arranged to meet comes and goes. You feel rather slighted that your friend has not made the effort to show up for you and you start to feel a bit low.”) Respondents are asked to put themselves into the situations and to feel the feelings described. After each description, respondents are asked to recall and briefly describe a specific occasion brought to mind by the feeling elicited by the vignette. After all eight vignettes are presented, the interviewer uses semistructured interviewing to elicit detailed descriptions of occasions and when they occurred; asks participants to rate the bad feelings at the worst point of each occasion, using a 0–100 scale anchored at 0 = not at all bad and 100 = as bad as you can imagine feeling; and gets detailed descriptions of participants’ responses to those feelings at the time of the original event. These descriptions are tape recorded. The interviewer rates the meta-awareness shown at the time of the original event using, supplemented by a coding manual, the following scale: 1 is minimal discrimination of different negative thoughts and feelings—being immersed in undifferentiated bad feelings. This may be reflected in the use of global descriptions of thoughts and feelings (e.g., “bad”, “crap”, “awful”) or of less global terms being used interchangeably. The participant may report being “swamped” by the thoughts and feelings, which they may attribute directly to an event or circumstance. They report no distance from their thoughts and feelings, no wider perspective. 2 is discrimination of different negative thoughts and feelings. The participant is able to report different strands of negative feelings and the different thoughts associated with them. A single but precisely described feeling may be taken as evidence of such discrimination (i.e., the participant was aware of feeling more x than y). Precision of description is more important than is the presence of two or more different feelings. 3 is some discrimination of self and own reactions from...
the situation. The participants have a sense of their thoughts and feelings as being distinct from the external situation. They would thus potentially be able to question or consider the appropriateness of their feelings or their perspective (e.g., “I’m too upset to think straight”). This may be reflected in some acknowledgement of internal generation of feelings (e.g., “I felt that I was being lazy”). The participants may report knowing that their views might be different once the mood changes or that other people might have a different view. They may arrive at this new perspective by talking to someone else. 4 is discrimination of self from thoughts and feelings. At some point, the participants can stand back and see their thoughts and feelings in a wider perspective. They may report being able to “stand back from the depression,” to “catch it,” or to “see what was going on.” Participants may report being able to link their thoughts and feelings to personal characteristics (e.g., “I’m being self-critical”) or may intentionally adopt an attitude toward those thoughts and feelings (e.g., “I’ll see if that’s right” or “I’ll try to let go of this”). 5 is persistent or extensive distancing from thoughts and feelings. The level of awareness obtained at Score 4 may be reached more quickly, more clearly, or may persist continuously once it has been reached. An independent rater also rates the tape recorded descriptions. Both raters receive extensive prior training in use of this scale. If the interviewer’s and independent rater’s ratings of a memory are more rapidly, more clearly, or may persist continuously once it has been reached. The interrater agreement was satisfactory. The conventional measure of internal validity, Cronbach’s alpha, is not readily applicable to MACAM measures as too many participants failed to retrieve a memory to one or more cues. Instead, for the sample of 40 patients, we calculated the odd–even split-half reliability for MAWARE. This yielded a correlation of .47 that, although significant (p = .003), was relatively modest.

Comparison of Depressed Patients and Controls

Depressed patients scored significantly lower than did nondepressed controls on each of the three MACAM measures (Table 3), indicating lower levels of metacognitive awareness in the memories accessed by depression-related cues in patients. Effect sizes for the differences between patients and controls were large for MAWARE (1.08) and MAWARE \( \leq 5 \) (1.18) and medium for MAWARE > 5 (0.69; Cohen, 1988, p. 26). Within the 40 patients, correlations between depression severity and MACAM measures were low and generally nonsignificant. For MAWARE, MAWARE \( \leq 5 \), and MAWARE > 5, correlations with HRSD were, respectively, −.15 (p > .1), −.02 (p > .1) and −.17 (p > .1), and with BDI, respectively, −.26 (p > .1), −.33 (p < .05), and −.17 (p > .1). These correlations suggest that, in general, MACAM measures did not simply reflect current severity of depression and that differences between patients and controls could not generally be attributed merely to differences between them in current depression.

Discussion

As predicted, compared with age- and gender-matched nondepressed controls, patients at high risk for relapse and recurrence of major depression showed significantly less evidence of a metacognitive set in relation to negative thoughts and feelings in the autobiographical memories most easily accessed by depression-related cues. These findings are consistent with the hypothesis that vulnerability to relapse and recurrence of major depression is related to a relative difficulty in accessing a metacognitive perspective with respect to depressive experience at times of low mood.

Study 2

Study 2 had two aims. The first was to test a further prediction from the hypothesis that the level of metacognitive awareness in relation to negative thoughts and depressed feelings at times of lowering mood is inversely related to the likelihood that such thoughts and feelings will persist and escalate to states of syndromal depression. This hypothesis predicts that patients in whom metacognitive sets are relatively more accessible are less likely to suffer further onsets than are patients in whom metacognitive sets are relatively less accessible. Further, the relationship of metacognitive awareness to onset should be over and above the relationship of onset to baseline depression level. The second aim was to investigate the hypothesis that the effects of CT in reducing relapse are achieved, at least in part, through its effects in increasing metacognitive awareness at times of dysphoric mood. This hypothesis predicts that, in situations where CT reduces relapse compared with a control treatment condition, post-
treatment measures of metacognitive awareness should be greater following CT than following the control treatment.

These predictions were tested in a clinical trial of CT in the prevention of relapse and recurrence in patients with recent major depression, partially remitted with treatment by antidepressant medication. In this trial, described in detail elsewhere (Paykel et al., 1999), CT significantly reduced relapse and recurrence. As in Study 1, the MACAM was used to measure accessibility of metacognitive sets in relation to negative thoughts and feelings.

Method

Design

At two treatment sites (Cambridge and Newcastle, England), 158 patients with recent major depression, partially remitted with antidepressant medication, were randomized to receive antidepressant medication and clinical management either alone (CM group) or together with CT (CT group). In a 20-week treatment phase all patients received drug continuation and clinical management, and the CT Group received, additionally, 16 CT sessions. Over a 48-week follow-up all patients received continuation and maintenance antidepressant medication. Clinical state was assessed before treatment, monthly during treatment, and bimonthly during follow-up. Metacognitive awareness was assessed before treatment (MACAM Version 1) and after 20 weeks treatment (MACAM Version 2).

Participants

Participants were psychiatric outpatients meeting selection criteria for the patient sample in Study 1; the latter were a subsample of the present sample. In Study 2, 10 patients not conforming to treatment protocol were eliminated from analyses of metacognitive awareness, leaving a maximum sample size of 148; in some analyses, sample size was less because of missing data.

Measures

HRSD and BDI. These were described in Study 1.

MACAM. This was described in Study 1. As in that study, separate measures were derived for memories from the 5 months preceding the MACAM assessment and for memories from before then. The reason for this is as follows. In examining predicted effects of CT on the MACAM measure of metacognitive awareness, it is important to be able to distinguish effects of CT on the encoding of depressing experiences (hypothesized to mediate effects of CT on relapse prevention) from effects of CT merely on the way participants talk about depressing experiences (which might, artifically, affect MACAM scores). Effects of CT on encoding can only occur after the start of CT, which was 20 weeks (5 months) before the posttreatment MACAM assessment. Consequently, effects of CT resulting from the creation of alternative (meta) cognitive sets at the time depressing events were experienced and encoded would only be apparent on posttreatment MACAM measures based on memories encoded in the preceding 5 months (MAWARE ≤ 5) and not on posttreatment measures based on memories encoded before the start of CT (MAWARE > 5). Conversely, effects of CT on the way depressing experiences are described at recall should be shown on memories encoded both before and after the beginning of CT. The creation of separate MAWARE ≤ 5 and MAWARE > 5 measures was primarily motivated by this need to clarify treatment effects on the MACAM, but this distinction having been made, it was also used in predictive analyses (and in Study 1).

Table 1
Study 1: Baseline Characteristics of Samples

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Control group (n = 20)</th>
<th>Depressed patients (n = 40)</th>
<th>Control vs. patient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Age</td>
<td>42.5</td>
<td>10.2</td>
<td>42.0</td>
</tr>
<tr>
<td>BDI</td>
<td>3.3</td>
<td>2.7</td>
<td>22.4</td>
</tr>
<tr>
<td>Female, %</td>
<td>65</td>
<td></td>
<td>58</td>
</tr>
</tbody>
</table>

Note. BDI = Beck Depression Inventory.

* df for unequal variances.

Table 2
Study 1: Interrater Agreement on MACAM Measures (Patient Sample)

<table>
<thead>
<tr>
<th>Measure</th>
<th>r</th>
<th>n</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAWARE</td>
<td>.77</td>
<td>40</td>
<td>.01</td>
</tr>
<tr>
<td>MAWARE ≤ 5</td>
<td>.64</td>
<td>40</td>
<td>.01</td>
</tr>
<tr>
<td>MAWARE &gt; 5</td>
<td>.83</td>
<td>32</td>
<td>.01</td>
</tr>
</tbody>
</table>

Note. MAWARE = Measure of Awareness and Coping in Autobiographical Memory; MAWARE ≤ 5 = metacognitive awareness (all memories); MAWARE > 5 = metacognitive awareness (memories in past 5 months).

Table 3
Study 1: Measures of Metacognitive Awareness in Controls and Patients

<table>
<thead>
<tr>
<th>Measure</th>
<th>Control group (n = 20)</th>
<th>Depressed patients (n = 40)</th>
<th>Control vs. patient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>MAWARE</td>
<td>2.80</td>
<td>0.36</td>
<td>2.32</td>
</tr>
<tr>
<td>MAWARE ≤ 5</td>
<td>3.00</td>
<td>0.54</td>
<td>2.37</td>
</tr>
<tr>
<td>MAWARE &gt; 5</td>
<td>2.69</td>
<td>0.38</td>
<td>2.25</td>
</tr>
</tbody>
</table>

Note. MAWARE = metacognitive awareness (all memories); MAWARE ≤ 5 = metacognitive awareness (memories in past 5 months); MAWARE > 5 = metacognitive awareness (memories before past 5 months).
Relapse. Two alternative relapse criteria were used: (a) Participants met DSM–III–R criteria for major depressive disorder for at least 1 month (2 weeks longer than DSM–III–R criteria require) and, at two successive face-to-face assessments at least 1 week apart, met both the severity criteria for major depression and scored 17 or more on HRSD or (b) participants’ residual symptoms during follow-up persisted between two successive ratings 2 months apart and reached HRSD scores of 13 or more on both occasions, and caused distress or dysfunction for which the withholding of additional active treatment was no longer justified.

Assessments

Interview assessments of depression and clinical state were made by experienced research psychiatrists at baseline, every 4 weeks through 20 weeks, and every 8 weeks thereafter. The MACAM was administered at baseline and at 20 weeks by master’s- or doctoral-level psychologists. Assessors were unaware of treatment group and patients were asked to conceal their treatment assignment.

Treatment

Clinical management and drug continuation (all patients). Patients were seen every 4 weeks during the main treatment phase and every 8 weeks in follow-up. Interviews were based on the clinical management described by Elkin et al. (1989) and lasted about 30 min. Symptoms were rated, drugs prescribed, and limited support provided. Specific CT techniques were prohibited.

CT. Patients received 16 sessions of CT during the 20-week main treatment phase, plus 2 booster sessions during follow-up. CT was as described by Beck et al. (1979), modified as described in a manual (cf. Scott, 1998). Elaborations of CT (Beck & Freeman, 1994), including techniques for engagement in CT and schema-focused approaches to core unconditional beliefs, were permitted. Therapists had diplomas in CT and at least 4 years CT experience. One therapist at each site provided CT for all CT patients at that site. In Cambridge, the therapist was a male clinical psychologist; in Newcastle, the therapist was a female nurse therapist.

Independent raters assessed audiotapes of randomly selected CT sessions on the Cognitive Therapy Rating Scale (CTRS; Young & Beck, 1990) to ensure fidelity and competence. Median CTRS score was 54, with no rating below 39, the accepted threshold level. Randomly selected psychiatrists’ clinical management sessions rated on the CTRS yielded ratings high on nonspecific items (e.g., empathy) but low on CT strategies; median score was 19 with no tape rated higher than 24.

Results

Baseline Characteristics of Treatment Groups

CT and CM groups were similar on initial variables (Table 4); none of the differences were statistically significant on conventional between-group tests.

Clinical Outcome: Relapse and Symptom Ratings

As reported by Paykel et al. (1999), survival analysis showed that CT significantly ($p = .02$) increased survival (nonrelapse). CT reduced risk of relapse by about 40% of the risk in CM (e.g., in an intent-to-treat analysis, cumulative relapse rates at 68 weeks were 47%, CM, and 29%, CT). This difference yields an $h$ value of 0.37, described by Cohen (1988, p. 185) as a small-to-medium effect size.

As previously reported (Paykel et al., 1999), CT and CM did not differ significantly on 20-week HRSD and BDI scores. Adjusted

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CM group ($n = 78$)</th>
<th>CT group ($n = 80$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>M = 43.2, SD = 11.2</td>
<td>M = 43.5, SD = 9.8</td>
</tr>
<tr>
<td>Female (%)</td>
<td>53</td>
<td>46</td>
</tr>
<tr>
<td>Marital status (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>Married</td>
<td>73</td>
<td>66</td>
</tr>
<tr>
<td>Formerly married</td>
<td>9</td>
<td>20</td>
</tr>
<tr>
<td>HRSD</td>
<td>12.2, 2.9</td>
<td>12.1, 2.7</td>
</tr>
<tr>
<td>BDI</td>
<td>22.3, 8.0</td>
<td>21.9, 7.7</td>
</tr>
<tr>
<td>Median length of index episode, months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(first and third quartile)</td>
<td>13.0 (9.0, 21.0)</td>
<td>14.5 (9.0, 18.0)</td>
</tr>
<tr>
<td>Severity of index episode, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild or moderate</td>
<td>35, 45</td>
<td>39, 49</td>
</tr>
<tr>
<td>Severe</td>
<td>43, 56</td>
<td>41, 51</td>
</tr>
<tr>
<td>No. of episodes, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>11, 14</td>
<td>10, 13</td>
</tr>
<tr>
<td>2</td>
<td>27, 35</td>
<td>29, 36</td>
</tr>
<tr>
<td>3+</td>
<td>22, 29</td>
<td>21, 26</td>
</tr>
<tr>
<td>Doses at inclusion, mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tricyclic (amitriptyline equivalent)</td>
<td>188, 45</td>
<td>186, 45</td>
</tr>
<tr>
<td>Selective serotonin reuptake inhibitor</td>
<td>36, 15</td>
<td>31, 11</td>
</tr>
</tbody>
</table>

Note. CM = clinical management; CT = cognitive therapy; HRSD = Hamilton Rating Scale for Depression; BDI = Beck Depression Inventory.
20-week means in CM and CT were, respectively, 9.40 and 8.58 for HRSD and 16.06 and 13.46 for BDI. CT reduced relapse, but it did not significantly reduce posttreatment depression.

**Prediction of Relapse From Baseline Measures of Metacognitive Awareness**

We used Cox proportional hazards regression analyses to examine prediction of relapse from baseline MACAM measures, for each measure entered alone and together with concurrent BDI or HRSD scores. Prediction from BDI and HRSD scores alone were also examined. Preliminary analyses showed that the interaction with treatment (CT vs. CM) provided no significant prediction beyond that of the variable alone for any predictor variable. Patients from both treatment groups were, accordingly, pooled for these analyses. The results are shown in Table 5.

Baseline BDI scores and baseline HRSD scores predicted hazard of relapses high levels of depression predicting early relapse. MACAM measures based on memories from the 5 months preceding baseline assessment (MAWARE ≤ 5) predicted hazard of relapses lower metacognitive awareness predicting earlier relapse. This prediction remained significant when either BDI or HRSD was also entered in the regression. Prediction from MAWARE ≤ 5 was substantial, 59% (19/32) with mean scores < 2 relapsed, compared with 34% (36/107) with mean scores ≥ 2.

Neither the MACAM measure based on memories more than 5 months prior to assessment (MAWARE > 5) nor the MACAM measure based on all memories (MAWARE) significantly predicted hazard of relapse.

**Timing of MACAM Memories in Relation to Clinical State**

Timing of recalled events in relation to the course of depression was examined. By selection, all patients experienced major depression in the 18 months before baseline assessment, but for at least 2 months before assessment, they experienced residual symptoms falling short of major depression. Mean duration of residual symptoms was 5.0 months (SD = 3.5 months).

Memories for MAWARE ≤ 5 almost all fell within the period of residual symptoms. 139 of the total 148 patients had one or more memories from the 5 months before baseline assessment, and for 116 (84%) of these all such events fell in the period of residual symptoms. By contrast, memories for MAWARE > 5 were mainly from the period of major depression or before: Of 122 patients with memories more than 5 months before baseline assessment, 25 (21%) had only memories from within the index episode, 54 (44%) had only memories outside episode, and 43 (35%) had memories from both inside and outside episode. Memories for MACAM > 5 often happened long ago; mean age of memory was 59 months (SD = 81) compared with a mean of 15 months since the start of the index episode.

**Prediction From Mood at the Time of the Remembered Events**

Metacognitive awareness ratings might have been related to the severity of depressed mood originally experienced at the time of the remembered events. If those levels of depression, themselves, predicted time to relapse, the prediction of relapse from MAWARE ≤ 5 might have been artifactual based on the correlation of MAWARE ≤ 5 with depression. To investigate this possibility, we examined the relationships between 0–100 ratings of negative mood during the remembered events, time to relapse, and MAWARE ≤ 5. For events from the 5 months before assessment, mean negative mood was not significantly correlated with mean MAWARE ≤ 5 scores, r(137) = −.12, p > .1, and prediction of relapse from MAWARE ≤ 5 remained significant when these mood scores were also included in the regression, Wald $\chi^2(1, N = 139) = 7.27$, p < .01. Thus, there was no evidence that prediction of relapse from MAWARE ≤ 5 depended on the correlation of both with levels of negative mood during the remembered events.

**Effects of Cognitive Therapy on Posttreatment MACAM Measures**

Table 6 shows mean MACAM scores before and after 20 weeks of CT and CM, together with results of analyses of covariance on posttreatment scores with pretreatment scores as covariates. Measures of metacognitive awareness based on all memories were significantly higher after CT than after CM. However, this difference was accounted for entirely by memories encoded during the treatment phase. Posttreatment MAWARE ≤ 5 measures were significantly higher for CT, but MAWARE > 5 measures were very similar for the two treatments. A repeated measures analysis of variance of MAWARE ≤ 5 and MAWARE > 5 measures with treatment (CT vs. CM) as between-subject factor and age of memory (within 5 months vs. before 5 months) as within-subject factor yielded a significant Treatment × Age of Memory interaction, $F(1, 108) = 8.35$, p < .01, confirming that memories encoded during treatment were more sensitive to CT than were those encoded before treatment. Memory age gave a significant main effect, $F(1, 108) = 27.51$, p < .01, with more recent memories showing higher metacognitive awareness (see Table 6).
The treatment effect on MAWARE ≤ 5 remained significant when a dummy variable indicating whether patients had relapsed before the posttreatment MACAM score was added as a covariate, \( F(1, 117) = 8.12, p < .01 \), and when the posttreatment BDI score was also included, \( F(1, 114) = 5.63, p < .05 \). Thus, effects of CT on MAWARE ≤ 5 were not secondary to effects on relapse or depression. The treatment effect size for MAWARE ≤ 5 (0.56) was medium (Cohen, 1988, p. 26).

In summary, as predicted, CT increased metacognitive awareness. The finding that CT affected memories encoded during treatment but not those encoded before treatment indicates that CT changed the encoding of depressive experiences rather than merely changing the way those experiences were described at recall, their encoding having been unchanged.

**Discussion**

Study 2 examined two predictions derived from the analysis of relapse and its prevention by CT presented in the introduction. The first was that the likelihood of relapse is inversely related to the ease with which depression-related cues access from patients’ memory metacognitive sets with respect to negative thoughts and feelings. This prediction was confirmed: A measure of metacognitive awareness in the memories of depressing experiences accessed from the 5 months before baseline assessment significantly predicted time to relapse; lower levels of metacognitive awareness predicted earlier relapse. This finding is consistent with the hypothesis that the ability to relate to depressive thoughts and feelings within a wider, decentered, perspective affects whether mild states of depression will escalate to more severe and persistent syndromal states characteristic of relapse. This finding also provides evidence for the discriminative validity of the MACAM. As reported elsewhere (Teasdale et al., 2001), the DAS and Attributional Style Questionnaire (Peterson et al., 1982) were also included in this treatment trial as measures of depressive thinking. Baseline values of dysfunctional attitudes and depressive attributional style derived from these measures failed to significantly predict increased risk of relapse; in contrast with the significant prediction from the MACAM reported here. Such evidence suggests that the MACAM has predictive validity over and above that shared with existing questionnaire measures of depressive thinking.

Prediction of relapse from MACAM measures was shown for memories of events from the 5 months prior to baseline assessment but not for memories of events occurring before that period. Two explanations can be suggested for the difference in prediction from recent and more distant memories. The first is that high MACAM scores require definite evidence of “discrimination of self from thoughts and feelings—seeing thoughts and feelings in a wider perspective,” and that the detailed recall necessary to provide such evidence is more likely to occur in recent rather than remote events. The second possible explanation is that the accessibility of metacognitive sets for depressing events occurring in the context of residual symptoms of depression, specifically, affects whether relapse will occur. This explanation is particularly interesting in relation to the established relationship between the presence of residual symptoms of depression and increased risk of relapse (e.g., Paykel et al., 1995). Consistent with this explanation, almost all the memories contributing to the predictive MAWARE ≤ 5 measure came from the period when patients experienced residual symptoms, whereas few of the memories contributing to the non-predictive MAWARE > 5 measure came from this period.

The second prediction examined was that if CT reduced relapse compared with a comparison treatment, CT would also increase accessibility of metacognitive sets with respect to negative thoughts and feelings compared with the comparison treatment. This prediction was confirmed, consistent with the hypothesis that CT reduced relapse through its effects in increasing metacognitive awareness. Differences between CT and CM were shown only on memories encoded during the treatment phase and not on memories encoded before then. This finding suggests that changes in the MACAM as a result of CT reflected CT’s effects on the encoding

<table>
<thead>
<tr>
<th>Measure</th>
<th>Clinical management (CM)</th>
<th>Cognitive therapy (CT)</th>
<th>CM vs. CT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>n</td>
</tr>
<tr>
<td>MAWARE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>2.23</td>
<td>0.49</td>
<td>74</td>
</tr>
<tr>
<td>Posttreatment</td>
<td>2.27</td>
<td>0.53</td>
<td>71</td>
</tr>
<tr>
<td>MAWARE ≤ 5</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Pretreatment</td>
<td>2.31</td>
<td>0.54</td>
<td>70</td>
</tr>
<tr>
<td>Posttreatment</td>
<td>2.35</td>
<td>0.60</td>
<td>67</td>
</tr>
<tr>
<td>MAWARE &gt; 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretreatment</td>
<td>2.14</td>
<td>0.69</td>
<td>63</td>
</tr>
<tr>
<td>Posttreatment</td>
<td>2.19</td>
<td>0.59</td>
<td>59</td>
</tr>
</tbody>
</table>

Note. MAWARE = metacognitive awareness (all memories); MAWARE ≤ 5 = metacognitive awareness (memories in past 5 months); MAWARE > 5 = metacognitive awareness (memories before past 5 months).
of depressing experiences rather than artifactual effects of CT on the way depressing experiences were described at recall.

The design of Study 2, in particular the timing of posttreatment MACAM in relation to the treatment effect on relapse, did not permit more detailed mediational analyses of metacognitive awareness, as described by Hollon, Evans, and DeRubeis (1990). For this reason, Study 2 could not formally demonstrate the causal status of change in metacognitive awareness as the mediator of the relapse prevention effects of CT.

**Study 3**

Results of Study 2 are consistent with the hypotheses that (a) lack of metacognitive awareness during depressing experiences increases the risk that such experiences will progress to states of major depression characteristic of relapse and (b) CT reduces relapse by increasing metacognitive awareness at such times. However, the findings of Study 2 are also consistent with an alternative hypothesis. In the introduction, it was suggested that changes in metacognitive awareness mediate the preventative effects of CT. This view contrasts with the more commonly held assumption that, although CT may lead to changes in metacognitive awareness, such changes are important only to the extent to which they facilitate change in belief in negative thoughts and underlying assumptions. On this latter view, changes in belief in underlying dysfunctional attitudes and assumptions are the route through which CT achieves its preventative effects and any changes in metacognitive awareness are merely the means through which these changes are achieved. Within this alternative perspective, the effects of CT on metacognitive awareness observed in Study 2 are seen as correlates of effective belief change rather than as the primary route through which relapse prevention was achieved.

A way to distinguish between these two alternative hypotheses is to examine the effects of an intervention designed to prevent depressive relapse by training patients in increased metacognitive awareness directly, without any explicit attempt to change belief in negative thoughts or underlying dysfunctional assumptions. Mindfulness-based cognitive therapy (MBCT; Segal, Williams, & Teasdale, 2002) was designed as such a treatment and has been shown to reduce relapse in recurrently depressed patients (Teasdale et al., 2000). In Study 3 we examined the effects of MBCT on MACAM measures of metacognitive awareness, using data from the trial on which Teasdale et al. (2000) reported clinical outcomes. If MBCT both reduces relapse and increases metacognitive awareness without targeting change in belief, it is parsimonious to assume that reduction in relapse by CT in Study 2 was also caused by changes in metacognitive awareness produced by CT.

Effects of MBCT on metacognitive awareness and relapse are also directly relevant to evaluating the hypotheses guiding development of MBCT (Teasdale et al., 1995). Teasdale et al. (1995) proposed that CT for depression reduces relapse by implicitly fostering a centered relationship to negative thoughts and feelings (i.e., increasing metacognitive awareness). This hypothesis suggests that a program designed to teach a centered relationship directly, without trying to change beliefs, could have comparable effects with CT in preventing relapse and increasing metacognitive awareness. Teasdale et al. described such a program, Attentional Control (Mindfulness) Training, subsequently renamed Mindfulness-Based Cognitive Therapy. If MBCT, like CT, prevents relapse and increases metacognitive awareness, it will support the hypothesis that they both operate through a common mechanism: increased metacognitive awareness.

**Method**

**Design**

At two treatment sites (Cambridge, England and Toronto, Canada) 100 patients, currently in remission or in recovery from major depression, were randomized to continue with treatment-as-usual (TAU) or, additionally, to receive MBCT. (MACAM data were not collected at the third treatment site in this trial.) Following an initial 7-week treatment phase, patients entered a 1-year follow-up phase.

**Participants**

Patients were recruited from community health care facilities and by media announcements. Inclusion criteria were (a) being between the ages of 18 and 65 years; (b) meeting enhanced DSM–III–R criteria for a history of recurrent major depression (these normally require a history of two or more previous episodes of DSM–III–R major depression in the absence of a history of mania or hypomania). We also required at least two episodes of major depression within the past 5 years and that at least one of those episodes have occurred within the past 2 years; (c) having a history of treatment by antidepressant medication but being off of it and in recovery or remission at the baseline assessment and for a minimum of the preceding 12 weeks; (d) having a score of less than 10 on the 17-item HRSD at the baseline assessment. Exclusion criteria were history of schizophrenia or schizoaffective disorder; current substance abuse, eating disorder, or obsessive–compulsive disorder; organic mental disorder, pervasive developmental delay, or borderline personality disorder; dysthymia before age 20; four or more lifetime sessions of CT; current psychotherapy or counseling more than once per month; current practice of meditation more than once per week or of yoga more than twice per week.

**Measures**

**HRSD and BDI**. These were described in Study 1.

**Relapse or recurrence**. The primary clinical outcome variable was relapse or recurrence to DSM–III–R criteria for major depressive episode, assessed by the Structured Clinical Interview for DSM–III–R (Spitzer, Williams, Gibbon, & First, 1992) at bimonthly assessments, covering the period from previous assessment. Assessors were doctoral-level psychologists. To maintain masked review by the assessors, we asked patients not to reveal their treatment condition or details that might prejudice awareness. To overcome occasional unmasking, interviews were audiotaped and, whenever patients met screening criteria for major depression, tapes were evaluated by an independent, masked, research psychiatrist (any information revealing patients’ treatment allocation was excluded from these tapes). In cases of disagreement, the masked ratings of the independent rater were used for analysis.

Following a baseline assessment, we interviewed patients at points corresponding to completion of the main MBCT-training phase and then bimonthly for a year.

**MACAM**. This was described in Study 1. Patients received, randomly, either Version 1 or Version 2 of the MACAM (see description in Study 1) 22 weeks after the start of the MBCT-treatment program. MACAM raters were unaware of treatment condition.
Treatment

TAU. Patients were instructed to seek help from their family doctor or from other sources as they normally would.

MBCT. MBCT is a manualized group skills-training program (Segal et al., 2002; Teasdale et al., 2000, p. 618). MBCT integrates aspects of CT for depression (Beck et al., 1979) with components of the mindfulness-based stress reduction program of Kabat-Zinn (1990). It is designed to teach patients in remission from recurrent major depression to become more aware of and to relate differently to their thoughts, feelings, and bodily sensations (e.g., relating to thoughts and feelings as passing events in the mind rather than identifying with them or treating them as necessarily accurate read-outs on reality). The program teaches skills that allow individuals to disengage from habitual (automatic) dysfunctional cognitive routines, in particular from depression-related ruminative thought patterns, as a way to reduce risk of relapse and recurrence of depression.

After an initial individual orientation session, the MBCT program is delivered by an instructor in eight weekly 2-hr group-training sessions involving up to 12 recovered recurrently depressed patients. During this period, the program includes daily homework exercises. Homework invariably includes guided (taped) or unguided awareness exercises directed at increasing moment-by-moment nonjudgmental awareness of bodily sensations, thoughts, and feelings, together with exercises to integrate awareness skills into daily life. Key themes of the program include empowerment of participants and a focus on awareness of experience in the moment. Participants are helped to cultivate an open and accepting mode of response, intentionally facing and moving into difficulties and discomfort, and to develop a decentered perspective on thoughts and feelings, viewing them as passing events in the mind. After the eight weekly meetings, four follow-up meetings occur at intervals of 1, 2, 3, and 4 months.

Instructors

The two instructors were experienced cognitive therapists who had participated in development of the MBCT program. Each had previously led at least one cohort of recovered depressed patients through the MBCT program.

Results

Because we were interested in the effects of MBCT in patients exposed to at least a predetermined “minimally adequate dose” of MBCT, results are presented for the 39 (75%) of the 52 patients allocated to MBCT who received 4 or more of the 8 weekly sessions and the 48 patients allocated to the TAU condition.

Patient Characteristics

TAU and MBCT groups were similar on baseline variables, with the exception of age (Table 7). When ages in the two groups were compared with conventional statistics, a result corresponding to a significant difference was obtained, t(85) = 2.55, p = .02. Given the size of the difference in means in relation to standard deviations, age was included as a covariate in comparisons of the effects of treatment.

Comparison of the 13 insufficient treatment MBCT patients, who either attended no MBCT sessions or dropped out before completing four sessions, with the 39 patients who completed four or more sessions revealed no statistically significant differences between these groups on baseline characteristics (smallest p = .46).

Table 7

<table>
<thead>
<tr>
<th>Study 3: Baseline Characteristics of Samples</th>
<th>TAU group (n = 48)</th>
<th>MBCT group (n = 39)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristic</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>46.4</td>
<td>10.6</td>
</tr>
<tr>
<td>Female (%)</td>
<td>81</td>
<td>74</td>
</tr>
<tr>
<td>HRSD</td>
<td>3.4</td>
<td>3.2</td>
</tr>
<tr>
<td>BDI</td>
<td>10.1</td>
<td>7.3</td>
</tr>
<tr>
<td>Length of last episode, weeks</td>
<td>19.3</td>
<td>14.0</td>
</tr>
<tr>
<td>No. of previous episodes (Mdn)</td>
<td>4.00</td>
<td></td>
</tr>
</tbody>
</table>

Note. TAU = treatment-as-usual; MBCT = mindfulness-based cognitive therapy; HRSD = Hamilton Rating Scale for Depression; BDI = Beck Depression Inventory.

Outcome Analysis: Relapse or Recurrence to Major Depression

Outcome data on the total three-center sample in the trial were previously reported by Teasdale et al. (2000). Here we report, for the first time, outcome for patients in two of the centers, Cambridge, England and Toronto, Canada, the only ones where patients were tested with the MACAM. Data on relapse or recurrence were available for 39 (100%) MBCT and 45 (94%) TAU patients. Times to onset of relapse or recurrence (in weeks) for MBCT and TAU were compared by Cox proportional hazards regression (SPSS Inc., 1994, pp. 291–328), with treatment condition as a categorical (indicator) variable and TAU as the reference condition. Hazard of relapse or recurrence was significantly less in MBCT, Wald χ²(1, N = 84) = 7.59, p < .01, hazard ratio = .397, 95% confidence interval = .205–.766. Overall 36% (14/39) experienced relapse or recurrence in MBCT compared with 62% (28/45) in TAU. χ²(1, N = 84) = 5.79, p < .02, a 42% reduction in risk in MBCT. This difference yields an h value of 0.53, a medium effect size of treatment (Cohen, 1988, p. 185).

Effects of MBCT on Posttreatment Metacognitive Awareness

Posttreatment MACAM assessment occurred 22 weeks after treatment began (i.e., 15 weeks after completion of the 8 weekly sessions by the MBCT group). Mean MAWAVE scores, based on all memories recalled, were 2.38 (SD = 0.34, n = 37) for controls and 2.59 (SD = 0.36, n = 37) for MBCT, a medium effect size (.60) of treatment (Cohen, 1988, p. 26). Analysis of covariance, with age as covariate, revealed a significant effect of treatment condition, F(1, 73) = 6.05, p < .02, metacognitive awareness being greater following MBCT than TAU. This difference remained significant when a dummy variable indicating whether patients had experienced relapse or recurrence before the MACAM assessment was added as an additional covariate, F(1, 72) = 5.33, p < .05, and also when the BDI score obtained at the time of the MACAM was added as a further covariate, F(1, 68) = 4.06, p < .05. These data indicated that, in response to depression-related cues, the accessibility of metacognitive sets with respect to negative thoughts and feelings was greater following MBCT than TAU.
Further, this difference could not be attributed to differences in age, relapse, or depression at the time of the MACAM assessment.

Discussion

Addition of MBCT to the treatment recovered, recurrently depressed patients normally receive led to a reduction in relapse or recurrence to major depression and an increase in metacognitive awareness in patients’ most accessible memories of depressing events. These findings show that, (a) as intended, MBCT increased patients’ metacognitive awareness with respect to negative thoughts and feelings and (b) an intervention primarily designed to increase metacognitive awareness by changing patients’ relationships to negative thoughts and feelings without any attempt to change belief in the content of negative thoughts or underlying assumptions can significantly reduce relapse or recurrence in depression. In light of these findings, it is parsimonious to assume that effects of CT in reducing relapse in Study 2 were also directly related to changes in metacognitive awareness produced by CT rather than to assume that such changes in metacognitive awareness were only important to the extent that they facilitated change in belief in thoughts and assumptions.

However, as with Study 2, the design of Study 3 did not permit a full mediational analysis. For this reason, Study 3 could not formally demonstrate the causal status of change in metacognitive awareness as the mediator of the relapse prevention effects of MBCT.

General Discussion

Metacognitive awareness describes a cognitive set in which negative thoughts and feelings are seen as passing events in the mind rather than as inherent aspects of self or as necessarily valid reflections of reality. We examined predictions from four hypotheses relating metacognitive awareness to vulnerability to recurrence of major depression and to mediation of relapse prevention effects of CT (Moore, 1996; Segal et al., 2002; Teasdale, 1997b; Teasdale et al., 1995): (a) increased vulnerability to further depression in individuals with a history of depression involves reduced metacognitive awareness to negative thoughts and feelings activated by dysphoric mood; (b) CT for depression, although explicitly focused on changing belief in the content of negative thoughts, leads, implicitly, to changes in relationships to negative thoughts and feelings, in particular, to increased metacognitive awareness; (c) increased metacognitive awareness mediates at least some of the relapse prevention effects of CT; and (d) interventions that increase metacognitive awareness will reduce risk of depressive relapse.

We used the MACAM measure of metacognitive awareness to assess the accessibility, in the context of depressing experiences, of metacognitive sets to negative thoughts and feelings. The MACAM measures metacognitive awareness in the memories most readily accessed by depression-related scenarios.

Study 1 found lower metacognitive awareness in memories recalled to depression-related cues by depressed patients, known to be at high risk of relapse, than in memories recalled by age- and gender-matched nondepressed controls. In patients, metacognitive awareness was generally independent of the level of current symptoms of depression, suggesting that the lower metacognitive awareness in patients than in controls could not, in general, be accounted for merely by greater depression in patients. These findings confirm our prediction that accessibility of metacognitive sets to cues present in relapse-related situations would be less in a group vulnerable to further onsets of major depression than it would be in a group low in vulnerability.

Study 2 investigated the relationship between accessibility of metacognitive sets and risk of relapse in a group of depressed patients at high risk of relapse. Patients who, at baseline, showed lower accessibility of metacognitive sets to depression-related cues experienced more onsets of depression soon thereafter. The relationship of metacognitive awareness to vulnerability was over and above that of depression to relapse. These results further support a role for reduced metacognitive awareness in vulnerability.

The hypothesis that changes in metacognitive awareness mediate the effects of CT in preventing relapse predicts that, when CT reduces relapse relative to a comparison treatment, it will increase the accessibility of metacognitive sets to depression-related material more than the comparison treatment. Study 2 confirmed this prediction. The finding that effects of CT on metacognitive awareness were shown only for memories encoded after CT had begun suggests that CT changed the way depressing experiences were encoded rather than merely the way they were described at recall. Effects of CT on metacognitive awareness were not secondary to effects of CT on depression levels or relapse. The design of Study 2, in particular the timing of posttreatment MACAM in relation to the treatment effect on relapse, did not permit more detailed mediational analyses of metacognitive awareness, as described by Hollon, Evans, and DeRubeis (1990).

Study 3 examined effects of MBCT in recovered, recurrently depressed patients. MBCT was designed to foster a metacognitive relationship to negative thoughts and feelings without any explicit attempt to change belief in thought content. Compared with TAU, MBCT both reduced relapse to major depression and increased accessibility of metacognitive sets to negative thoughts and feelings. Effects of MBCT on metacognitive awareness were not secondary to effects on depression or relapse. Thus, Study 3 showed that an intervention, other than CT, that increased metacognitive awareness also reduced further onsets of depression. These findings are consistent with the hypothesized role of changes in metacognitive awareness in mediating relapse prevention. However, as with Study 2, the design of Study 3 did not permit a full mediational analysis. The findings of Study 3, together with the general failure in the wider literature to show specific effects of CT on measures of belief in negative thoughts, suggest that effects of CT on metacognitive awareness in Study 2 were the likely route through which relapse prevention was mediated rather than merely the means to the end of change in belief in thoughts.

We should note limitations of the present findings. The most important of these concern the modest internal reliability and lack of prior evidence of validity of the MACAM measures. The MACAM was developed at the time that the clinical trials in which it was used were just about to begin. This situation did not allow time for extensive instrument development or for demonstration of validity and reliability prior to the present study. The resulting instrument was clearly useful, as evidenced by the present results,
but should be considered as at a preliminary stage of development. Further improvements in internal reliability and evidence of validity are obviously desirable.

Considering our results more widely, we note that development of meta-awareness of internal experience has been seen as a core task in many forms of psychotherapy (Rice & Greenberg, 1984). The importance of metacognition in vulnerability to emotional disorders and in the way CT alleviates those disorders has also been recognized previously. For example, Ingram and Hollon (1986) suggested

Cognitive therapy relies heavily on helping individuals switch to a controlled mode of processing that is metacognitive in nature and focuses on depression-related cognition...the long-term effectiveness of cognitive therapy may lie in teaching patients to initiate this process in the face of future stress. . . . (p. 272)

These authors used the term metacognition to refer to an awareness of how cognition contributes to depression.

More recently, a Special issue of Clinical Psychology and Psychotherapy (Wells & Purdon, 1999) was devoted to metacognition and cognitive--behavior therapy. Wells (1999) described a metacognitive model and therapy for generalized anxiety disorder on the basis of an assumption that metacognitive beliefs concerning advantages and dangers of worrying maintain pathological worry. He used metacognition to refer to “beliefs concerning worry” (Wells, 1999, p. 86). More recently, Shappard and Teasdale (2000) reported a deficit in metacognitive monitoring of dysfunctional products in major depression. Here, metacognitive monitoring referred to a process in which the initial products of cognitive processing were appraised. The suggestion that CT might have its effects by helping individuals compensate for such a deficit is reminiscent of Barber and DeRubeis’s (1989) suggestion that CT works by teaching patients to reappraise, or have “second thoughts,” in relation to depressive cognition.

These examples show that metacognition has been used to describe a range of phenomena. It is important, therefore, to be clear about what we mean by metacognitive awareness. The distinction between metacognitive knowledge and metacognitive insight (Teasdale, 1999) is relevant here. Metacognitive knowledge refers to beliefs about cognitive phenomena stored in memory as propositional facts in much the same way as other facts. An example would be the belief “worrying helps me avoid disastrous situations” (cf. Wells, 1999). Such propositional beliefs are potentially open to the evaluation procedures traditionally used in CT. Metacognitive insight, on the other hand, refers to the way mental phenomena are experienced as they arise. Metacognitive awareness, as we define it, is a form of metacognitive insight. We can illustrate the difference between metacognitive insight and metacognitive knowledge by using the example “thoughts aren’t facts.” Metacognitive insight (awareness) refers to actually experiencing thoughts as thoughts (that is, as events in the mind rather than as direct readouts on reality) in the moment they occur. By contrast, metacognitive knowledge involves thinking about thoughts as “other than facts” or knowing, intellectually, that the content of thoughts does not always correspond to the state of the world (Teasdale, 1999, p. 147).

Increasing metacognitive awareness involves a change in relationship to thoughts and feelings. Our findings suggest that CT may prevent depressive relapse by increasing metacognitive awareness and that a program targeting such a change in relationship, rather than a change in belief in thought content, can have similar effects to CT. In providing a new perspective on the mechanisms of therapeutic change in CT, these findings open up new avenues for exploration in understanding the process of change in CT and for the development of novel treatment approaches.

References


