CHAPTER 1

Why a Treatment Targeting Rumination?

This manual details a novel form of cognitive-behavioral therapy (CBT) designed to treat depression and its common comorbid disorders by targeting ruminative thinking. In Part I, I introduce the rationale behind developing the therapy and the theoretical and clinical principles that underpin it. A guiding principle in my work has been that we need to do better at treating and preventing depression. Understanding and targeting key mechanisms causing depression is an effective means to do this.[1]

Addressing a Major Treatment Gap

Depression Is a Major Global Challenge

Depression is a highly prevalent disorder, affecting 20% of women and 10% of men in their lifetimes. Further, it is a chronic, debilitating, and recurrent disorder (Kessler et al., 1994). The medical, social, economic, and personal costs of depression are enormous, as it erodes quality of life, reduces productivity in the workplace, impairs fulfilment of social and familial roles, increases the risk of suicide and self-harm, and substantially increases global disease burden (World Health Organization, 2008).

[1]A key distinction at the outset is between unipolar depression and bipolar disorder. Unipolar depression, which is the principal focus of the rumination-focused treatment work, includes only depressive conditions occurring in the absence of current or past mania or hypomania. The treatment described in this manual has to date only been developed and evaluated in the context of unipolar depression. The use for patients with mania or hypomania would not be advised.

In this context, depression principally refers to the diagnosis of major depression within agreed guidelines, such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), now in its fifth edition (DSM-5; American Psychiatric Association, 2013), and the *International Classification of Diseases*, now in its 10th edition (ICD-10; World Health Organization, 1992). The essential features of major depression include seriously compromised mood (at least 2 weeks of continuous depressed mood or loss of interest/pleasure/motivation, such as anhedonia) and at least four additional cognitive, behavioral, or physical symptoms. Individuals must show the symptoms all or most of the day, nearly every day for at least 2 weeks. In order to be diagnosed, the episode must be clinically significant, in terms of causing distress or impaired functioning in the person's typical social or occupational roles. Furthermore, alternative causes for the symptoms, such as bereavement (although this has been removed in DSM-5) or the direct physiological effects of medical illness (e.g., hypothyroidism), medications, and substance misuse need to be ruled out before a diagnosis of a major depressive episode can be reached.

Major depression produces the second-largest burden of disease in the world today and is by far the leading cause of disability (World Health Organization, 2001); it is estimated that by 2020 it will have the second-highest disease burden across all disorders (Murray & Lopez, 1996).

Other forms of depressive disorder include dysthymic disorder, which is diagnosed if symptoms persist for at least 2 years, although there might be brief periods of normal mood lasting no more than 2 months. Additionally, in order to be diagnosed, dysthymic disorder must be seen to cause significant distress or disruption in the person's significant areas of functioning. Minor depressive disorder is diagnosed for at least 2 weeks of symptoms but with fewer than the five symptoms required for major depressive disorder. Recurrent brief depression refers to episodes lasting 2 days to 2 weeks, occurring at least once a month for a year. Combined together, these different forms of depression are the most common presentation of mental health difficulties, accounting for 38% of all outpatient diagnoses in the United States. Each is associated with distress and disability.

A Major Treatment Gap

Critically, despite the high frequency and impact across all forms of depression, we still face a major treatment gap. Most people with depression do not receive treatment, about one-third of those who do receive treatment do not respond to current approaches, and over half of those who experience a first onset of a major depressive episode will experience one or more recurrences. Thus, although we have effective treatments such as antidepressant medication and CBT (Hollon et al., 2005; Nathan & Gorman, 2007) there is still considerable scope to improve treatments. Limitations of cur-

rent effective therapies include substantial rates of partial or non-response (greater than 40%) and disappointing rates of remission (less than a third; Hollon et al., 2005; Nathan & Gorman, 2007). Moreover, even effective treatments have high rates of relapse and recurrence (50–80%), such that few patients actually enjoy sustained recovery (Bruce et al., 2005; Judd, 1997). Improved relapse prevention has been identified as a priority for treatment research in depression, because a significant proportion of people with depression experience a chronic or recurrent life course. We need to improve the efficacy and sustained effects of our treatments.

For example, after patients with major depression are treated with recommended doses of antidepressant medication, approximately 30% experience partial remission, that is, no longer meet criteria for major depression but still have elevated depressive symptoms, which cause significant distress and disability (Cornwall & Scott, 1997; Paykel et al., 1995). This subtype of depression is characterized as residual depression, and sometimes called treatment-refractory or medication-resistant depression. It is a chronic and persistent form of depression. Residual depression is important because of its frequency and because residual symptoms increase the likelihood of future relapse and recurrence of depression. In prospective longitudinal studies, elevated residual symptoms provide one of the best predictors of future depressive relapse (Fava, 1999; Judd, 1997; Judd, Paulus, & Zeller, 1999; Paykel et al., 1995). Furthermore, treatments that reduce residual symptoms reduce the risk of relapse (Fava, Zielezny, Savron, & Grandi, 1995). Chronicity of depression is also associated with substantial distress, high rates of comorbidity, marked functional impairments, and increased health care utilization. Randomized controlled trials (RCTs) of CBT suggest that it is effective at reducing subsequent depressive relapses when it is effective at reducing acute symptoms, but that it is less effective at achieving remission in chronic depression. Finding treatments for depression that are better at tackling residual depression and treatment-refractory chronic depression, and at achieving remission and preventing recurrence, is a pressing need.[2]

How Do We Make Our Treatments Effective and Lasting?

It is thus evident that improving the efficacy and sustained effect of psychological interventions requires better reduction of residual symptoms in

[2]Technically, the diagnosis of residual depression in a patient is operationalized as experiencing an episode of a major depressive disorder in the last 18 months while not meeting criteria for major depression in the last 2 months, but nonetheless having depressive symptoms (scores of at least 8 on the 17-item Hamilton Rating Scale for Depression [HRSD] and 9 on the Beck Depression Inventory [BDI]). Participants also need to have been taking antidepressant medication for at least the previous 8 weeks, with 4 or more weeks at a minimum clinically recommended daily dose (e.g., equivalent to at least 125 mg of amitriptyline).

depression. There are many common residual symptoms, including irritability, anxiety, loss of confidence, insomnia, and a tendency to worry and ruminate about difficulties. One potential approach to enhancing treatments is to identify and specifically target these residual symptoms.

In parallel to targeting key residual symptoms, recent recommendations to improve psychological treatments have emphasized the value of targeting an identified psychopathological mechanism to enhance treatment efficacy (Barlow, 2004). While CBT for depression is effective and influences depressogenic information processing (Hollon et al., 2005), it has changed little from the seminal treatment manual (Beck, Rush, Shaw, & Emery, 1979), despite considerable subsequent psychopathology research. In contrast, CBT for anxiety has evolved in response to psychopathology research, resulting in new and more effective interventions (e.g., the work of David Clark and Anke Ehlers in panic disorder, social anxiety, and posttraumatic stress disorder [PTSD]). There is thus scope to improve psychological interventions for depression by focusing on underlying mechanisms, as recommended in the recent Research Domain Criteria initiative from the U.S. National Institute of Mental Health (Sanislow et al., 2010, p. 631).

The treatment presented in this book does just that. In particular, it focuses on the mechanism of rumination. Happily, focusing on ruminative thought in depression has the potential to kill two birds with one stone, as it targets both a residual symptom of depression (Roberts, Gilboa, & Gotlib, 1998) and a key mechanism implicated in its onset and maintenance (Nolen-Hoeksema, 2000; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008). To be precise about our terms, rumination is here defined as recurrent and repetitive thinking on symptoms (e.g., fatigue, low mood), feelings, problems, upsetting events, and negative aspects of the self, typically with a focus on their causes, meanings, and implications. More specifically, Susan Nolen-Hoeksema defined rumination as "passively and repetitively focusing on one's symptoms of distress and the circumstances surrounding these symptoms" (Nolen-Hoeksema, McBride, & Larson, 1997, p. 855).

Rumination as a Primary Therapeutic Target

There are good reasons to choose rumination as a primary therapeutic target. First, it is a common residual symptom, remaining elevated after both partial and full remission from depression (Riso et al., 2003; Roberts et al., 1998). Both currently depressed and formerly depressed patients report elevated levels of rumination compared to those who have never been depressed (Roberts et al., 1998). Moreover, elevated rumination is associated with less responsiveness to both antidepressant medication and cognitive therapy (Ciesla & Roberts, 2007; Schmaling, Dimidjian, Katon, & Sullivan, 2002), suggesting it may contribute to partial remission.

Second, there is extensive and robust evidence implicating rumination in the onset and maintenance of depression (Nolen-Hoeksema et al., 2008; Watkins, 2008). Prospective longitudinal studies have found that self-reported rumination, typically assessed on the Response Styles Questionnaire (RSQ; Nolen-Hoeksema, 1991; Treynor, Gonzalez, & Nolen-Hoeksema, 2003), predicts (1) the future onset of a major depressive episode across a range of follow-up periods in initially nondepressed individuals (Just & Alloy, 1997; Nolen-Hoeksema, 2000), with Spasojević and Alloy (2001) finding that rumination mediated the effect of other risk factors on onset of depression; (2) depressive symptoms across a range of follow-up periods in initially nondepressed individuals, after controlling for baseline symptoms (Abela, Brozina, & Haigh, 2002; Butler & Nolen-Hoeksema, 1994; Hong, 2007; Nolen-Hoeksema, 1991; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Parker, & Larson, 1994; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Sakamoto, Kambara, & Tanno, 2001; Smith, Alloy, & Abramson, 2006); and (3) depressive symptoms in patients with clinical depression, after controlling for baseline depression (Kuehner & Weber, 1999; Nolen-Hoeksema, 2000; Rohan, Sigmon, & Dorhofer, 2003).

In addition, experimental studies provide convergent evidence that rumination plays a causal role in a range of unconstructive outcomes associated with depression, including exacerbating negative mood and increasing negative thinking. These studies used a standardized rumination induction, in which participants are instructed to spend 8 minutes concentrating on a series of sentences that involve rumination about themselves, their current feelings and physical state, and the causes and consequences of their feelings (e.g., "Think about the way you feel inside") (Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema & Morrow, 1993). As a control condition, a distraction induction is typically used, in which participants are instructed to spend 8 minutes concentrating on a series of sentences that involve imagining visual scenes unrelated to the self or to current feelings (e.g., "Think about a fire darting round a log in a fireplace").

Compared to the distraction induction, the rumination induction is reliably found to have negative consequences for mood and cognition. Critically the differential effects of these manipulations are found only when participants are already in a sad mood before the manipulations, indicating a moderating role of existing mood. Under these conditions, compared to distraction, rumination exacerbates negative mood (Lavender & Watkins, 2004; Lyubomirsky & Nolen-Hoeksema, 1995; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1993; Watkins & Teasdale, 2001); increases negative thinking (Lyubomirsky & Nolen-Hoeksema, 1995); increases negative autobiographical memory recall (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998); reduces the specificity of autobiographical memory retrieval (Kao, Dritschel, & Astell, 2006; Park,

Goodyer, & Teasdale, 2004; Watkins & Teasdale, 2001; Williams et al., 2007); increases negative thinking about the future (Lavender & Watkins, 2004); impairs concentration and central executive functioning (Lyubomirsky, Kasri, & Zehm, 2003; Watkins & Brown, 2002); impairs controlled memory retrieval (Hertel, 1998); and impairs social problem solving (Donaldson & Lam, 2004; Lyubomirsky & Nolen-Hoeksema, 1995; Lyubomirsky, Tucker, Caldwell, & Berg, 1999).

Taken together, the prospective and experimental studies strongly implicate rumination in the onset and maintenance of depression. Third, depressive rumination partially accounts for the 2:1 rates of depression in women relative to men: once we statistically adjust for the greater tendency for women to ruminate, there is no longer a difference between men and women in rates of depression (Butler & Nolen-Hoeksema, 1994; Grant et al., 2004; Nolen-Hoeksema, Larson, & Grayson, 1999).

Fourth, clinical experience suggests that rumination is a key and often neglected component of patient's phenomenology in depression. For patients, depressive rumination often involves dwelling on past losses, analyzing past mistakes, and making social-evaluative judgments and comparisons. This thinking often includes "why" questions, such as "Why did this happen to me?"; "Why do I feel like this?"; "What went wrong?"; "Why can't I get things right?" Depressive rumination is often characterized by evaluative thinking, with patients making negative comparisons between themselves and others ("Why do I have problems other people don't have?"), between their current state and desired state ("Why can't I get better?"), and between the current self and past self ("Why can't I work as well as before?"). Patients report rumination as unintended, hard to stop, persistent, and repetitive. It is experienced as distressing and with a sense of being hard to control. There is the sense of being driven to ruminate, with a quality of "having to do it." The common reported consequences of rumination are increased sadness, distress, and anxiety, reduced motivation, insomnia, and increased tiredness, procrastination, self-criticism, pessimism, and hopelessness.

Thus, the logic of this approach is that successfully targeting rumination would both tackle a residual symptom of depression and reduce an important mechanism contributing to its onset and maintenance, thereby improving treatment outcomes.

Rumination as a Transdiagnostic Process

There is one further potential benefit of targeting rumination within psychological treatments. Rumination has been identified as a transdiagnostic or cross-cutting process, which means it is a mechanism that is (1) shared across multiple disorders and (2) causally contributes to the onset, maintenance, recurrence, or recovery from multiple disorders (Harvey, Watkins,

Mansell, & Shafran, 2004). There is evidence that rumination is common to multiple emotional disorders, in particular, depression, generalized anxiety disorder (GAD), social anxiety, PTSD, and eating disorders (for reviews see Aldao, Nolen-Hoeksema, & Schweizer [2010]; Ehring & Watkins [2008]; Nolen-Hoeksema & Watkins [2011]; Watkins [2008]), and causes both depression and anxiety disorders.

For example, Aldao et al. (2010) examined the relationships between emotion regulation strategies, including rumination, and symptoms of psychopathology across anxiety, depression, and eating- and substance-related disorders from 114 studies. There was a large effect size for rumination across all psychopathologies. Moreover, two large-scale longitudinal studies found that rumination explained the concurrent and prospective associations between symptoms of anxiety and depression (McLaughlin & Nolen-Hoeksema, 2011). In other studies, rumination prospectively predicted substance abuse (Nolen-Hoeksema et al., 2007; Skitch & Abela, 2008), alcohol abuse (Caselli et al., 2010), and eating disorders (Holm-Denoma & Hankin, 2010; Nolen-Hoeksema et al., 2007), after controlling for initial symptoms. Nolen-Hoeksema et al. (2007) examined the relationship between rumination and symptoms of depression, bulimia, and substance abuse in 496 female adolescents followed prospectively over time. Rumination predicted future increases in bulimic and substance abuse symptoms, as well as onset of major depression, binge eating, and substance abuse. This evidence suggests that rumination is a strong candidate to be considered a transdiagnostic process that contributes to psychopathology.

Consistent with the tenets of the transdiagnostic approach (Harvey et al., 2004), targeting rumination may thus have the further advantage of addressing comorbid presentations. After all, it is more common than not that a patient seeking help for depression will actually be suffering from multiple disorders—that is, presenting with two or more comorbid conditions. Most typically, such patients are dealing with both anxiety and depression. There are high rates of such comorbidity. Twelve-month rates of comorbid anxiety and/or depression are estimated as high as 40–80% (Kessler, Chiu, Demler, & Walters, 2005).

In practice, this means that a key decision for clinicians is how to treat comorbidity. As a therapist, you no doubt have frequently had patients present with a mixture of symptoms and difficulties. Further, one of your first and often hardest decisions is determining which difficulty or disorder to target first. If a patient has both social anxiety and depression, should you focus first on treating the social anxiety or on the depression? To date, we don't really have good empirical guidance for this all-too-common situation. The majority of our therapy models, for example, cognitive-behavioral treatments, have focused on individual distinct diagnoses, with evidence showing worse outcomes for patients with comorbidity.

Using a transdiagnostically focused treatment, which acts to reduce multiple emotional disorders simultaneously, may be one way to address comorbidity. One approach is to build a treatment package that has a range of therapeutic components integrated together, with a view to addressing multiple disorders. The best example of this approach is the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders, developed by David Barlow and his colleagues (Barlow, Allen, & Choate, 2004; Wilamowska et al., 2010). This treatment essentially takes all the common elements across different CBT treatments for anxiety and depression and combines them into one package. For example, this treatment includes reducing avoidance, with exposure to external feared stimuli and to interoception; increasing behavioral activation (BA); and thought challenging. Preliminary evidence indicates this approach may have benefit (Wilamowska et al., 2010). Similarly, transdiagnostic treatment packages for eating disorders have been developed with some success by Christopher Fairburn and his colleagues (Fairburn, Cooper, & Shafran, 2003).

An alternative transdiagnostic treatment approach, as proposed by Harvey et al. (2004) and Mansell, Harvey, Watkins, and Shafran (2008), is to identify transdiagnostic mechanisms that cut across multiple disorders and then to target those mechanisms explicitly. It has been argued that a transdiagnostic approach to treatment may provide an efficient means to address comorbidity. Mansell et al. (2008) proposed several potential advantages of a treatment focused on identified transdiagnostic processes. First, it potentially enables us to match interventions to the specific vulnerabilities and processes that are relevant to the individual. For example, if an individual was assessed and found to be highly prone to rumination, it would be sensible to select treatment elements that reduce rumination. Second, it directly targets fundamental active mechanisms, rather than symptom clusters, and, as such, is hypothesized to improve treatment efficacy (Barlow, 2004; Sanislow et al., 2010). Third, it enables a flexible treatment approach that can be applied across a range of presentations, including comorbidity. Indeed, such a transdiagnostic treatment has the potential to produce more potent interventions that better address comorbidity than treatments based on diagnoses or disorders (Mansell et al., 2008).

Because rumination is found to causally contribute to both anxiety and depression, there is a strong case for selecting rumination as the focus of a transdiagnostic intervention (Topper, Emmelkamp, & Ehring, 2010). Successfully reducing rumination should reduce both anxiety and depression. The treatment described in this book is one of the first attempts to develop and evaluate such a transdiagnostic process intervention. It is important to note that to date this therapy has only been evaluated in RCTs for acute treatment or prevention of depression. However, the theoretical and transdiagnostic rationale behind an intervention for rumination suggests that

it should provide a useful therapeutic module to include in psychological interventions for other disorders that include repetitive negative thought, including GAD, PTSD, and social anxiety.

Summary of Rumination-Focused Cognitive-Behavioral Therapy

Before going into more detail about the principles, rationales, and techniques of rumination-focused cognitive-behavior therapy (RFCBT), I briefly overview what it involves and how it compares to existing psychological therapies for depression. RFCBT is a manualized CBT treatment, typically consisting of up to 12 individual sessions scheduled weekly or biweekly.

The therapy is theoretically informed by experimental research indicating that there are distinct constructive and unconstructive forms of rumination (Watkins, 2008). This research suggests that there are distinct styles of rumination, with distinct functional properties and consequences: a helpful style characterized by concrete, process-focused, and specific thinking versus an unhelpful, maladaptive style characterized by abstract, evaluative thinking (Treynor et al., 2003; Watkins, 2004a; Watkins & Baracaia, 2002; Watkins & Moulds, 2005; Watkins & Teasdale, 2001, 2004). Building on these findings, the therapy is designed to coach individuals to shift from unconstructive rumination to constructive rumination, through the use of functional analysis (FA), experiential and imagery exercises, and behavioral experiments. FA is an approach aimed at determining the functions and contexts under which desired and undesired behaviors occur and thereby finding ways to systematically increase or reduce target behaviors. It is focused on studying the variability and context of behavior within an individual's personal experience and using this to guide interventions.

These adaptations distinguish RFCBT from standard CBT for depression (Beck et al., 1979), which focuses on modifying the content of individual thoughts, by placing a greater emphasis on directly modifying the process of thinking. Although still grounded within the core principles and techniques of CBT for depression (Beck et al., 1979; e.g., collaborative empiricism, Socratic questioning, behavioral experiments), RFCBT involves several additional, novel elements.

First, it incorporates the functional-analytic and contextual approach developed in the BA treatment that resulted from a component analysis of CBT (Addis & Martell, 2004; Jacobson et al., 1996; Jacobson, Martell, & Dimidjian, 2001; Martell, Addis, & Jacobson, 2001). This approach is based on the view that rumination is a learned habitual behavior that has developed through negative reinforcement. RFCBT incorporates the functional-analytic and contextual principles and techniques of BA (Martell, 2003), but explicitly and exclusively focused on rumination. Within BA and

RFCBT, rumination is conceptualized as a form of avoidance, and FA is used to facilitate more helpful approach behaviors.

Second, RFCBT makes much less use of thought challenging than standard CBT does. Socratic questioning in RFCBT tends not to focus on the evidence and accuracy of thinking or on generating alternative interpretations, but rather on the function, purpose, and usefulness of thoughts and behaviors. There is a focus on the pattern and sequence of thoughts rather than the meaning of individual thoughts. This shift in focus has the advantage of avoiding the risk of getting into disputes and arguments with patients over the meaning and interpretation of thoughts, events, and situations.

Third, a key innovative element within RFCBT is the focus on shifting a patient's processing style from unconstructive forms of thinking to more constructive forms of thinking, using FA, imagery, and experiential approaches. RFCBT uses FA to help individuals realize that their rumination about negative self-experience can be helpful or unhelpful and to coach them in how to shift to a more helpful style of thinking. In addition, patients use directed imagery to re-create previous mental states when a more helpful thinking style was active, such as memories of being completely absorbed in an activity (e.g., "flow" or "peak" experiences). Shifting to these states acts directly counter to rumination.

Empirical Evidence

RFCBT has been investigated in three clinical studies: a case series of individual RFCBT for patients with residual depression (Watkins et al., 2007), an RCT of individual RFCBT for patients with residual depression (Watkins et al., 2011, funded by the National Alliance for Research on Schizophrenia and Depression [NARSAD]), and an RCT of group RFCBT and Internet-based RFCBT to reduce and prevent depression in a high-risk group of young adults selected for having elevated levels of worry and rumination (Topper, Emmelkamp, Watkins, & Ehring, 2014). There have also been trials of concreteness training, which is a specific element within the RFCBT treatment package (Watkins et al., 2012; Watkins, Baeyens, & Read, 2009, funded by the UK Medical Research Council). All of these treatment evaluations have had positive findings, indicating that RFCBT and its components are efficacious at reducing rumination and depression. This section briefly summarizes each of the relevant studies.

Case Series of Individual Face-to-Face RFCBT for Residual Depression

A case series investigated 12 weekly 60-minute sessions of RFCBT for 14 consecutively recruited patients meeting criteria for medication-refractory residual depression (Watkins et al., 2007). Treatment produced signifi-

cant improvements in depressive symptoms and comorbid disorders: mean reduction in Beck Depression Inventory of 20 points, pre- to posttreatment within-subject effect size (Cohen's *d*) of 2.5, 50% of patients achieving full remission from depression, and a 71% reduction in comorbid Axis I diagnoses. Importantly, RFCBT significantly reduced self-reported rumination, with rumination at pretreatment equivalent to that found in currently depressed patients but the range of scores at posttreatment equivalent to levels of rumination observed in never-depressed participants. This study provides initial evidence that RFCBT may be an efficacious treatment for depressive rumination and that it can tackle both depression and comorbid disorders.

Phase II RCT of Individual Face-to-Face RFCBT for Residual Depression

The study (Watkins et al., 2011) was approved by the U.K. National Health Service South London and Maudsley Research Ethics Committee and was conducted in community mental health teams and psychological treatment services in South East London and Devon, United Kingdom. Patients who were referred to outpatient services for depression and/or on the waiting list for psychological therapies were approached, and those who met inclusion criteria and gave written informed consent to participate were randomly allocated to treatment as usual (TAU) alone or to TAU plus RFCBT. TAU consisted of ongoing antidepressant medication and outpatient clinical management. Randomization was performed by an off-site researcher using computer-generated random numbers and stratified according to gender and the duration of the index episode of major depression. All participants were assessed by research staff masked to treatment allocation at intake baseline assessment and again 6 months later. Patients were included in the trial if they were over 18 years old and met criteria for medication-refractory residual depression, defined as meeting diagnostic criteria for major depression within the past 18 months but not in the past 2 months and with elevated residual symptoms of depression, and taking antidepressant medication at a recommended therapeutic dose. Patients were excluded from the trial if they had a history of bipolar disorder, psychosis, current drug or alcohol dependence, intellectual disability, or organic brain damage or were receiving concurrent psychotherapy at point of entry to the study. There were no exclusion criteria with respect to comorbid anxiety disorders or Axis II personality disorder diagnoses. Forty-two patients were randomized in the trial and followed up.

Adding RFCBT to TAU significantly reduced residual symptoms and improved remission rates relative to TAU alone, with a mean difference in change in symptoms from pre to post-treatment of 7.57 between the treat-

ments on the Beck Depression Inventory scores (95% confidence interval = 1.86–19.08). The between-treatment effect size (standardized mean difference) was $d = 1.11$, which is good for a psychological treatment. Furthermore, there was a significant effect of treatment condition on rates of treatment response (TAU 26% vs. RFCBT 81%), rates of remission (TAU 21% vs RFCBT 62%), and rates of relapse between baseline and postintervention assessments (TAU 53% vs. RFCBT 9.5%). RFCBT therefore significantly outperformed continuing with maintenance antidepressants alone.

The outcomes found for 12 sessions of RFCBT (remission rates of 62%; between-treatment effect sizes of 0.94–1.1) for patients with residual depressive symptoms compare favorably with 20 sessions of standard CBT for depression (Paykel et al., 1999; remission rates of 25%; between-treatment effect size of 0.3) in an identically defined sample of participants with residual depression. Moreover, we found that the addition of a psychological intervention beneficially augmented pharmacotherapy, in contrast to other recent trials (e.g., Kocsis et al., 2009). Although we have to be cautious when comparing between differently powered studies, the outcomes for our TAU condition closely match the outcomes for the TAU arm in the Paykel et al. (1999) trial. In the absence of a definitive large-scale RCT of RFCBT with a larger sample and a longer follow-up, these results raise the possibility that the modifications made to CBT in RFCBT may engender better treatment outcomes in residual depression.

The number of comorbid Axis II diagnoses at study end, covarying for initial rates, was significantly less in the RFCBT group than the TAU group (TAU: $M = 0.67$, $SD = 0.97$; RFCBT: $M = 0.24$, $SD = 0.44$). There was also a similar, but nonsignificant trend for fewer comorbid Axis I disorders in the RFCBT group than the TAU group at follow-up (TAU: $M = 1.05$, $SD = 0.97$; RFCBT: $M = 0.62$, $SD = 0.86$, $p = .068$). Thus, consistent with the transdiagnostic hypothesis, there is some evidence that targeting rumination reduces both depression and other comorbid disorders.

Moreover, RFCBT significantly reduced self-reported rumination more than TAU, and the treatment effects on depression were mediated by change in rumination, although this was only measured concurrently. This provides evidence that the treatment reduced rumination as intended. It was also found to significantly reduce worry, as assessed using the Penn State Worry Questionnaire (PSWQ).

Concreteness Training

Consistent with a causal relationship between processing mode and individual differences in rumination, a proof-of-principle randomized controlled treatment intervention trial found that training depressed individuals to be more concrete when faced with difficulties reduced depression, anxiety, and rumination relative to a no-treatment control (Watkins et al., 2009).

The concreteness training involved repeated practice at asking "How?" and focusing on specific details when thinking about recent difficulties.

In a Phase II RCT, concreteness training was found to be superior to TAU in reducing rumination, worry, and depression in patients with major depression recruited in primary care (Watkins et al., 2012). Thus, shifting depressed patients into a more concrete processing mode reduced rumination and associated symptoms.

RCT of Group RFCBT and Internet-Delivered RFCBT to Target Rumination and Prevent Depression and Anxiety

This recently completed treatment trial (Topper, Emmelkamp, Watkins, & Ehring, 2016) examines two adaptations of RFCBT (a group format and an Internet-delivered format; see Chapter 13 for further details) as an intervention to prevent depression and anxiety. Because of the extensive evidence that rumination predicts the onset and maintenance of depression, individuals with elevated tendency to rumination are at greater risk to develop depression. This makes the targeting of high ruminators a plausible strategy for preventing the initial onset of depression, as rumination increases the likelihood of someone developing depression, is easily identifiable, and is a tractable psychological process (Topper et al., 2010). Topper et al. (2010) recently made a strong case for treatments that explicitly target rumination as a potential approach to preventing depression. Moreover, because of the evidence that rumination is a transdiagnostic process, targeting rumination may also help to prevent anxiety disorders, eating disorders, and substance and alcohol misuse.

A completed randomized trial comparing the group and Internet versions of RFCBT found that both RFCBT adaptations were effective relative to waiting-list control groups for reducing depression, anxiety, worry, and rumination in young adults selected for their vulnerability to worry and rumination, in a high-risk prevention intervention design conducted in Amsterdam (n = 251, project team: Prof. Thomas Ehring, Prof. Paul Emmelkamp, Dr. Maurice Topper, Prof. Ed Watkins, supported by ZonMw funding to Principal Investigator Prof. Thomas Ehring; see Topper et al., 2016). This study selected both males and females ages 15–21 with elevated worry and rumination scores but no current major depression or anxiety disorder, and randomized them to Internet RFCBT, group RFCBT, or waiting-list control and then followed up for 12 months. Intent-to-treat analyses showed that both versions of RFCBT intervention significantly reduced worry and rumination (controlled effect size Cohen's d = 0.53 to 0.89), as well as symptom levels of anxiety and depression (Cohen's d = 0.36 to 0.72) at postintervention, relative to the waiting-list control, with these effects maintained at 1-year follow-up. There were no differences between the group and Internet online versions of RFCBT on any of the outcome measures. The interven-

tions also resulted in a significantly lower 1-year incidence rate of major depression (group intervention 15.3%, Internet intervention 14.7%) and GAD (group intervention 18%; Internet intervention 16%), compared to the waiting list (32.4% and 42.2%, respectively). However, these findings are based on caseness cutoffs on well-established self-report measures rather than structured diagnostic interviews, and further replication using diagnostic interviews is necessary to confirm the findings on preventing depression. Nonetheless, these results provide proof of principle that rumination increases the risk for the onset of major depression and GAD, given the elevated incidence rates in the untreated group of people with elevated levels of worry/rumination relative to the general population. The findings also provide further evidence that RFCBT can be an effective intervention to reduce worry and rumination, and that it can be effectively delivered in both group and Internet-based formats. Moreover, the findings are consistent with the transdiagnostic hypothesis of rumination, as targeting rumination reduced both depression and anxiety.

Case Series of Individual Face-to-Face RFCBT for PTSD

A version of RFCBT has also been used to tackle persistent PTSD in a population of young survivors of the 1994 genocide in Rwanda (Sezibera, Van Broeck, & Philippot, 2009). The rationale for using RFCBT in this population is that rumination has been identified as a major maintaining factor in PTSD (Michael, Halligan, Clark, & Ehlers, 2007), consistent with its hypothesized transdiagnostic role. In this study, all the participants were orphans of the Rwandan genocide in 1994 who met criteria for PTSD assessed on self-rating scales 11 years after the genocide and again 13 years after the genocide, indicating that the PTSD was persistent. Twenty-two individuals ages 15–18 years were treated (54.5% female). The treatment incorporated elements from RFCBT, including psychoeducation and FA of rumination, as well as narrative exposure to trauma reminders, and lasted for 10 weekly sessions of maximum duration 2 hours. The intervention was associated with a reduction in PTSD symptoms, with gains maintained at 2-month follow-up. Although this is an uncontrolled study and therefore needs to be interpreted with caution, it provides further evidence that targeting rumination may have transdiagnostic benefit.

Group RFCBT for Residual Depression

A further independent trial (Teismann et al., 2014) has confirmed that group-delivered RFCBT improved depressed mood and reduced rumination relative to a waiting-list condition in patients with residual depression, with treatment gains maintained over 1-year follow-up. This RCT assigned 60

patients with residual depression to a group-delivered RFCBT treatment incorporating elements of both RFCBT and metacognitive therapy versus a waiting-list control. Group RFCBT outperformed waiting list (remission rates 42% vs. 10.3%), with effects maintained for 1 year. This study provides an important confirmation of the potential benefits of RFCBT from an independent research group.

Comparisons between RFCBT and Other Treatments

CBT for Depression

Despite the growing evidence that rumination is an important mechanism in depression, the original CBT for depression (Beck et al., 1979) did not explicitly focus on treating rumination in detail. There is a brief mention of rumination in the seminal 1979 book *Cognitive Therapy of Depression*, but no specific elaborations on how to address it, presumably under the assumption that repeated challenging of negative thoughts would suffice.

The clinical experience my colleagues and I have accumulated in our clinic through treating highly ruminative chronically depressed patients over the last 20 years has shown us that the classic CBT approach for depression (Beck et al., 1979) can sometimes be effective, but that it has a number of limitations and difficulties. First, focusing on challenging individual thoughts is not effective when dealing with a strong and habitual stream of negative thoughts, as is characteristic of depressive rumination. Trying to stop one thought does not prevent the full flow of rumination, because the first negative thought is simply followed by another thought in the chain, often in the form of a "Yes, but" thought.

The clinical experience of using a classic CBT approach with patients who ruminate can be like trying to stop a waterfall by catching one drop of water at a time. The difficulty of changing rumination is entirely consistent with conceptualizations of rumination as a habitual response (Nolen-Hoeksema, 1991) because habits are argued to be difficult to change by challenging beliefs. Our experience was that thought challenging can be helpful for rumination but only under two specific circumstances. It can prevent rumination when it catches the start of the chain of ruminative thoughts and nips it in the bud. Alternatively, when patients conscientiously practised thought challenging so that it became a habit in its own right, it could displace the habit of ruminative thinking.

Second, thought challenging can itself act as a further trigger to rumination. For example, once you have successfully challenged the evidence for a negative automatic thought with a depressed patient, the patient may then dwell on the thought "Why couldn't I do that before?" or "Why am I so stupid?," and the cycle of recurrent thinking is off and running again.

To effectively treat rumination, we hypothesized that it may be better for patients to step back from the thought process itself, rather than from any individual thought.

Third, with patients highly prone to rumination, any form of discussion and disputation can become focused on *talking about* what has happened and what it might mean to the patient. When that happens it is easy to become trapped in ruminating aloud with the patient, where sequences of negative thinking are repeatedly discussed in detail without any therapeutic change. For patients who ruminate, there is also a strong press toward thinking about and talking about the causes, meanings, and implications of their symptoms and difficulties. This can easily become the focus of the treatment session, with the patient bringing up difficulties to reflect on each week. One indicator of such "corumination" is the realization that large amounts of a treatment session have passed without any sense of progress. This was my experience with the first few patients I treated where rumination was a prominent difficulty. We would have often interesting and engaging discussions about the big issues in the patient's life and what they might mean, but little therapeutic progress was made, and symptoms did not improve.

Outcome evidence finds that standard CBT interventions are less effective at treating depression in high ruminators compared to low ruminators (Ciesla & Roberts, 2002; Schmaling et al., 2002). Furthermore, to date, there is no reported evidence from RCTs that standard CBT can reduce rumination.[3]

RFCBT uses standard CBT approaches, organization, and components such as a structured format, here-and-now focus, collaborative empiricism, agenda setting, use of feedback and summaries, homework, guided discovery, and behavioral experiments. However, as mentioned earlier, there are adjustments and alterations from the standard CBT protocol.

Behavioral Activation

There are emerging interventions that more directly target rumination than classic CBT, although direct evidence for their efficacy at reducing rumination is still lacking. BA was originally one component of the full CBT intervention, consisting of activity monitoring and activity scheduling. A trial comparing the different components of CBT found that BA alone was as effective at reducing symptoms as BA plus thought challenging and as the full CBT protocol (Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996). As a consequence, BA was elaborated into a stand-alone treatment, focusing on understanding the function and context in which depression occurs and targeting avoidance behaviors in depression (Martell et al.,

[3]Of course, we need to be careful about interpreting the current trials because until recently rumination was not an outcome measure in the majority of treatment trials.

2001). In a large-scale RCT, BA has been found to be an effective intervention for depression, producing outcomes as good as pharmacotherapy and better than CBT for severe depression (Dimidjian et al., 2006). However, to date, its effect on rumination has not been formally assessed.

As noted earlier, the RFCBT described in this book shares a number of similarities with BA, as well as several key differences. Both approaches incorporate a functional-analytic and contextual approach to behaviors. The development of RFCBT has been informed by the work within BA and by dialogue with leading proponents of BA, such as Christopher Martell. Within both BA and RFCBT, rumination is conceptualized as a form of avoidance, and FA is used to facilitate the reduction of this avoidance and to replace it with more helpful approach behaviors. Like BA, RFCBT treats rumination as behavior, even if covert, which can be contextually and functionally understood. However, RFCBT has greater elaboration in its approaches to rumination than BA. Moreover, an additional novel element not shared with either BA or standard CBT is the explicit focus on shifting thinking style during rumination, derived from my experimental research. The experiential exercises in RFCBT designed to shift thinking style (e.g., concreteness training, absorption work, compassion work) are not found in BA, although they are consistent with its functional-contextual principles.

Mindfulness-Based Cognitive Therapy

Another recent treatment that is explicitly designed to reduce rumination is mindfulness-based cognitive therapy (MBCT). MBCT incorporates elements of a mindfulness-based stress reduction program (Kabat-Zinn, 1990) into CBT to create a relapse prevention treatment for recurrent depression (Teasdale, Segal, & Williams, 1995). MBCT is delivered in weekly group training sessions, in which participants practice and develop a moment-by-moment awareness of sensations, thoughts, and feelings through the use of formal and informal meditation exercises, such as watching the breath and the body scan. The theoretical rationale behind MBCT is that training patients to step back and observe their thoughts and feelings as mental events and to become connected with direct experience in the present moment would reduce evaluative ruminative thinking.

In two RCTs, for patients with a history of three or more episodes of major depression but who were currently symptom-free, MBCT significantly reduced risk of relapse and recurrence over 1 year compared to TAU (Ma & Teasdale, 2004; Teasdale et al., 2000). Kuyken et al. (2008) demonstrated that MBCT has similar rates of relapse over 1 year follow-up to continuation antidepressant medication for patients with recurrent depression.

Consistent with the proposed theoretical underpinnings, mindfulness approaches have been found to reduce rumination, although not all studies randomized to treatment condition or used clinical populations. In

an experimental analogue study, Feldman, Greeson, and Senville (2010) compared mindful breathing, progressive muscle relaxation, and loving-kindness meditation on negative reactions to repetitive thoughts in undergraduates and found that the association between frequency of repetitive thoughts and degree of negative reaction to thoughts was significantly less in the mindful breathing condition relative to the other two, suggesting that mindfulness reduced the impact of rumination. Two studies comparing pre-to-post change in rumination in mindfulness versus a matched waiting-list control (mindfulness meditation, Chambers, Lo, & Allen, 2008; mindfulness-based stress reduction [MBSR], Ramel, Goldin, Carmona, & McQuaid, 2004) demonstrated a reduction in rumination in the treatment group, although neither study randomized to condition. A randomized trial of mindfulness meditation versus relaxation in a nonclinical sample demonstrated a reduction in rumination (Jain et al., 2007). In an RCT, Geschwind, Peeters, Drukker, van Os, and Wichers (2011) found that MBCT reduced self-reported rumination pre- to postintervention relative to waiting-list control in patients with a history of major depression and current residual symptoms. However, Kuyken et al. (2008, 2010) failed to find that MBCT reduced rumination more than continuation antidepressant medication in patients with a history of recurrent depression.

RFCBT differs from MBCT in its suggested target population and treatment content and style. To date, MBCT has been shown to be effective in preventing relapse in people who are not currently depressed but have a history of recurrent depression (three or more episodes). It is not known whether MBCT is of value for patients in an acute episode of depression when rumination is at its most fierce. It may be that it is difficult or counterproductive to try to meditate when experiencing acute depressed mood and strong rumination. Future research will need to determine whether MBCT is effective for patients with acute symptoms of depression.

In contrast, RFCBT is designed to be used with patients experiencing acute symptoms of depression and rumination, whether in a major depressive episode or with residual symptoms. Indeed, one further advantage of targeting rumination is that it is observed to be elevated as a risk factor prior to the onset of depression, during episodes of major depression, in partial remission, and in full remission from depression. Elevated rumination is thus found at all points in the course of depression. Targeting rumination thus has potential for primary prevention, acute treatment, and prevention of relapse and recurrence, gaining further efficiency from the treatment (see Chapter 13).

RFCBT is a much more direct intervention than MBCT. It is explicit about what it is trying to achieve and how it is trying to coach patients into more helpful ways of coping through active practice of exercises. In contrast, MBCT is more indirect and involves patients learning more gradually through their own experience during meditation.