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Depressive Rumination: Investigating Mechanisms to Improve Cognitive Behavioural Treatments

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Abstract. Rumination has been identified as a core process in the development and maintenance of depression. Treatments targeting ruminative processes may, therefore, be particularly helpful for treating chronic and recurrent depression. The development of such treatments requires translational research that marries clinical trials, process–outcome research, and basic experimental research that investigates the mechanisms underpinning pathological rumination. For example, a program of experimental research has demonstrated that there are distinct processing modes during rumination that have distinct functional effects for the consequences of rumination on a range of clinically relevant cognitive and emotional processes: an adaptive style characterized by more concrete, specific processing and a maladaptive style characterized by abstract, overgeneral processing. Based on this experimental work, two new treatments for depression have been developed and evaluated: (a) rumination-focused cognitive therapy, an individual-based face-to-face therapy, which has encouraging results in the treatment of residual depression in an extended case series and a pilot randomized controlled trial; and (b) concreteness training, a facilitated self-help intervention intended to increase specificity of processing in patients with depression, which has beneficial findings in a proof-of-principle study in a dysphoric population. These findings indicate the potential value of process–outcome research (a) explicitly targeting identified vulnerability processes and (b) developing interventions informed by research into basic mechanisms. *Key words:* rumination; translational; concrete; specificity; mediator.

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Understanding the mechanisms that determine how cognitive behaviour therapy (CBT) works is a key objective for process–outcome research. First, such knowledge can confirm or refute our theoretical accounts of therapy. Second, identifying the active ingredients of effective CBT could lead to substantial increases in the efficacy and efficiency of CBT through deliberately adapting and refining the therapy to enhance these active components. Third, understanding these mechanisms could enhance therapy training and supervision by focusing on those therapy-specific behaviours actively involved in treatment gains. Fourth, determining moderators of treatment would make the allocation of psychotherapy more systematic by suggesting

which patients under which conditions are most likely to benefit from CBT.

There are several complementary research approaches to investigating how therapy works: (a) the use of process–outcome measures during clinical trials to identify potential variables that predict symptom change and that meet statistical criteria for mediators (Baron & Kenny, 1986; Kraemer, Wilson, Fairburn, & Agras, 2002); (b) dismantling studies, which separate and compare distinct components of effective therapies (e.g. behavioural activation vs. thought challenging; Jacobson et al., 1996); and (c) experimental studies, which manipulate variables relevant to therapy elements in order to test their causal relationship with symptom change.

The subject of the current article is a variant on the dismantling and experimental approaches: the development and evaluation of novel therapies that explicitly target a specific hypothesized mechanism of change. The logic of this approach is that if a specific process causes the maintenance of symptoms, then a treatment that specifically and explicitly alters that process should have therapeutic benefit. Moreover, such a finding would provide proof of principle that this process is potentially involved as a mechanism in treatment response. I describe two examples of this approach for CBT for depression: (a) targeting rumination, which is a key process implicated in the maintenance of depression, and (b) enhancing increased specificity of thinking, which is a hypothesized mechanism of action for effective CBT for depression.

Rumination-focused CBT

Depressive rumination is implicated in the onset and maintenance of depression, with longitudinal studies demonstrating that rumination prospectively predicts the likelihood, severity, and duration of syndromal depression (e.g. Nolen-Hoeksema, 2000; for a review, see Watkins, 2008a). Moreover, experimental studies have demonstrated that inducing rumination in dysphoric participants exacerbates negative mood and negative thinking, relative to inducing distraction, suggesting a causal effect of rumination in maintaining psychopathology (Nolen-Hoeksema, 1991; Watkins, 2008a).

Therefore, one potential mechanism of action for CBT and other effective treatments for depression is the reduction of rumination. However, few studies have assessed rumination as a potential mediator of change. If, as hypothesized, rumination is a mediator of treatment for depression, then adapting CBT to better reduce rumination should improve the efficacy of CBT for depression. To this end, a variant of CBT specifically targeting rumination was developed (rumination-focused CBT [RFCBT]; Watkins et al., 2007).

Although still grounded within the core principles and techniques of CBT for depression, RFCBT involves several additional, novel elements. First, it incorporates the functional-analytic and contextual approach developed in the behavioural activation (BA)

treatment that resulted from a component analysis of CBT (Jacobson et al., 1996; Martell, Addis, & Jacobson, 2001). BA approaches were integrated into RFCBT because BA includes an explicit focus on reducing rumination from a functional-analytical perspective. Within BA and RFCBT, rumination is conceptualized as a form of avoidance, and functional analysis is used to facilitate the reduction of this avoidance and to replace it with more helpful approach behaviours.

Second, the approaches used within RFCBT are derived from recent experimental research suggesting that there are distinct styles of rumination, with distinct functional properties and consequences: a helpful style characterised by concrete, process-focused, and specific thinking versus an unhelpful, maladaptive style characterised by abstract, evaluative thinking (see review in Watkins, 2008a). This research has shown that the abstract style of rumination (characterized by asking “why?” and focusing on evaluating the meanings and implications of feelings and difficulties) increases overgeneral memories, impairs problem-solving, and increases global negative self-judgments relative to a concrete style of rumination (characterized by asking “how” and focusing on the specific contextual details of feelings and difficulties). A key implication of this research is that ruminative self-focus can be constructive or unconstructive, depending on the style of processing, and that there may be therapeutic benefit in coaching patients to shift from a harmful to a beneficial form of self-focused thinking about negative material.

Thus, a key assumption of RFCBT is that rumination is a normal and understandable process, which can be useful if done appropriately. In practice, RFCBT uses functional analysis to help patients realise that their rumination about negative self-experience can be helpful or unhelpful and then to coach them how to shift into the most effective style of thinking. Functional analysis focuses on the variability of (a) rumination (e.g. differences between helpful and unhelpful thinking about problems; differences between short and long bouts of rumination); (b) associated behaviours (e.g. procrastination), and (c) counter-ruminative behaviours such as effective engagement in tasks. This detailed analysis

of context and function is then used to help patients recognise warning signs for rumination, develop alternative strategies and contingency plans (e.g. relaxation, assertiveness), and alter environmental and behavioural contingencies maintaining rumination (e.g. shifting the balance from routine chores and obligations toward self-fulfilling activities). Further, RFCBT uses experiential/imagery exercises and behavioural experiments designed to facilitate a shift into the more helpful concrete thinking style. Patients use directed imagery to vividly recreate previous 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) or experiences of being compassionate to themselves or others. Such exercises provide a direct counter to rumination and can be used within contingency plans. These adaptations mean that RFCBT differs from standard CBT for depression, which focuses on modifying the content of thoughts, by having a greater emphasis on directly modifying the process of thinking.

RFCBT was first investigated in a multiple baseline case series of 14 patients with residual depression, with each patient receiving individual therapy for up to 12 sessions (Watkins et al., 2007). Residual depression was defined as meeting diagnostic criteria for depression within the last 18 months but not in the last 2 months, still experiencing some level of depressive symptoms, and taking antidepressant medication at a therapeutically recommended dose for at least 8 weeks (Paykel et al., 1999). Residual depression was selected as a conservative test of RFCBT because CBT added to antidepressant medication showed no advantage over antidepressant medication alone in reducing acute residual symptoms (Paykel et al., 1999).

The result of this preliminary case series was encouraging, with an average pretreatment to posttreatment reduction of 20 points on the Beck Depression Inventory-II (BDI-II). Seventy-one per cent of patients met criteria for treatment response ($\geq 50\%$ decrease in baseline Hamilton Rating Scale for Depression [HRSD] scores), and 50% met full remission criteria (< 8 on HRSD and < 9 on BDI-II for 4 consecutive weeks). Importantly, there was also a significant mean reduction in self-reported rumination as assessed by the Response Styles Questionnaire.

However, this case series is limited by a small group size, the lack of a comparison/control group, and the lack of rater blindness. Without a control group, the observed improvements cannot be unequivocally attributed to RFCBT. To rectify these limitations, a pilot randomized controlled trial compared treatment as usual (ongoing antidepressant medication) versus treatment as usual plus up to 12 sessions of individual RFCBT for the acute treatment of residual depression (for full details, see Watkins, 2008b). The key preliminary finding from this trial was that, whereas both treatment arms reduced symptoms of depression, there was a significantly greater reduction in symptoms for the treatment-as-usual plus RFCBT arm. Importantly, these results compare favorably with those of Paykel et al. (1999): 62% of patients receiving treatment as usual plus RCBT met full remission criteria versus 21% in the treatment-as-usual condition and 25% in the treatment-as-usual plus CBT arm in Paykel et al. (1999). Thus, there is preliminary evidence that specifically targeting rumination may improve CBT treatment outcome for residual depression, consistent with the hypothesis that rumination may be a mediator of treatment outcome. However, this interpretation needs to be tentative because there has not been a direct comparison of RFCBT versus standard CBT in a single randomized controlled trial.

Moreover, consistent with the hypothesis that change in rumination may be a mediator of symptom improvement, rumination met all the Baron and Kenny (1986) criteria for a mediator (treatment changed symptoms; treatment changed rumination; change in symptoms was associated with change in rumination; treatment was a poorer predictor of change in symptoms once change in rumination was entered into the regression). However, both symptoms of depression and rumination were measured concurrently so there was no temporal precedence for change in rumination relative to symptom change. Without temporal precedence, the relationship between change in the putative mediator and symptom change could reflect reverse causation such that change in depressive symptoms results in change in rumination (Kraemer et al., 2002). Thus, the current findings cannot establish that rumination is a causal mediator

of symptom improvement in RFCBT. Rather, these results are a necessary, but not sufficient, step in determining whether rumination is a mediator of treatment outcome. If rumination was not found to meet Baron and Kenny's (1986) criteria, it would be ruled out as a potential mediator of the effects of RFCBT. Having passed this test, further studies need to determine whether change in rumination precedes change in symptoms. Moreover, it is important to note that in the absence of measurement of rumination as a process–outcome variable in other trials of CBT, it is an open question whether change in rumination is a mechanism of action unique to RFCBT or whether, as hypothesized, it is also a potential mechanism of action for standard CBT (albeit weaker).

Despite these reservations, these findings indicate the potential value of developing a treatment targeted on a core identified process, such as rumination, and developing interventions that are informed by basic research into the mechanisms of that process. Moreover, this research suggests that there is further value in investigating the reduction of depressive rumination as a potential mechanism of action for CBT.

Increasing specificity

Another example of this research approach involves the development of a treatment explicitly targeting a hypothesized mechanism of action for effective CBT: increased specificity of thinking. There is a range of convergent evidence leading to the hypothesis that increasing specificity of thinking is a potential mechanism of action by which CBT reduces depressive symptoms. First, depression is characterized by an increased tendency away from specificity and toward overgeneral thinking, whether overgeneralization, in which a general rule or conclusion is drawn on the basis of isolated incidents and applied across the board to related and unrelated situations (Beck, 1976; Carver & Ganellen, 1983) or increased retrieval of categoric and overgeneral autobiographical memories (Williams et al., 2007). Both overgeneralization and overgeneral memory are specific to depression and prospectively predict subsequent levels of depression (e.g., Carver, 1998; Williams et al., 2007).

Second, there is experimental evidence that manipulating the degree of specificity influences emotional reactivity to a subsequent stressful task, with repeated practice at being specific and concrete, whether through recalling personal memories or imagining emotional scenarios, resulting in less subsequent emotional reactivity than practice at being abstract and general (Raes, Hermans, Williams, & Eelen, 2006; Watkins, Moulds, & Moberly, 2008). For example, participants who practiced focusing on imagined emotional scenarios in a specific and concrete way (“Focus on how it happened, and imagine in your mind as vividly and concretely as possible a ‘movie’ of how this event unfolded”) demonstrated smaller decreases in self-reported positive affect and smaller increases in negative affect following a subsequent failure on an insoluble anagram task compared with participants who practiced more abstract processing (“Think about why it happened, and analyze the causes, meanings, and implications of this event”) when focusing on the same emotional scenarios (Watkins et al., 2008).

Third, there is evidence from clinical trials that increases in specificity are associated with treatment improvements. Concrete treatment techniques within CBT, such as asking for specific examples of difficult events, predict subsequent symptom reduction when assessed early in CBT, whereas more abstract techniques do not (DeRubeis & Feeley, 1990; Feeley, DeRubeis, & Gelfand, 1999). Likewise, patient improvement by midpoint of therapy in the use of situational analysis, which involves generating a specific description of the context relevant to a particular problem and generating specific goal-oriented behaviours, predicts reduced depression at the end of a cognitive behavioural intervention (Manber et al., 2003). Finally, as described previously, pathological rumination is characterised by an abstract, overgeneral style of processing. Experimentally inducing a more specific, concrete style of processing during repetitive self-focus reduces the detrimental effects on mood and cognition observed during more abstract rumination (Watkins, 2008a). Thus, increased rumination and reduced specificity appear to be interlinked and to share abstract processing in common.

If this specificity-as-mechanism hypothesis is correct, then a treatment intervention that specifically and exclusively focuses on increasing specific and concrete thinking should be effective at reducing depressed symptoms. A recent study provided a proof-of-principle test of this hypothesis by randomizing dysphoric participants to an active intervention designed to increase specificity (concreteness training), a bogus training condition that lacked elements to increase specificity but was matched for treatment rationale, therapist contact, and other nonspecific factors, or a wait-list control (Watkins et al., 2009; see also Watkins & Moberly, 2008). The concreteness training consisted of explicit instructions to actively engage in being specific (e.g., focusing on the specific sensory details of an event, on what makes each event specific, unique and distinctive, and on the process of how the event and behaviors unfolded) when imagining emotional events, both standard vignettes and personal autobiographical memories. These instructions were derived from the experimental materials used in Watkins et al. (2008), described previously. Participants in the concreteness training condition practiced this 30-min exercise everyday for a week, using an audio recording of the exercise. The bogus training condition consisted of repeated daily practice on a computerized task that presented short descriptions of social situations that remain ambiguous in overall meaning, until the final word, presented as a fragment to be completed, which resolved the overall meaning for each scenario. Across all the scenarios, each word fragment was chosen to direct the participant into generating a specific interpretation (e.g. "You have been seeing each other for 3 weeks, and it seems that you have found a true soulmate. After dinner one evening, your partner explains that you can't be together anymore. At that moment, you stare at the table and contemplate your empty gl_ss," with "glass" forcing a specific interpretation). To reinforce the required specific interpretation, participants had to correctly type in the missing letter of the fragment and then respond to a comprehension question about the description. Thus, while involving materials that had face validity for influencing specificity and sharing the same explanation as concreteness training concerning the value of becoming more specific, the

bogus training did not involve participants actively generating more specific descriptions of personal events and, therefore, was not expected to directly alter the degree of concrete processing.

Consistent with the hypothesis that increased specificity of thinking may be a mechanism of action responsible for symptom reduction, Watkins et al. (2009) found that the concreteness training condition produced greater symptom reduction on the Hamilton Rating Scale for Depression than both the bogus training and wait-list controls. Moreover, the concreteness training condition resulted in more specific descriptions of problems than the other two conditions and significantly greater reductions in rumination than the wait-list control condition. Thus, these findings provide proof of principle that increased specificity of processing can reduce depressive symptoms and, as such, are consistent with the hypothesis that CBT may work, at least in part, by increasing specificity of processing.

There are, however, several reasons to be cautious about this interpretation. First, this study only examined the effects of the training over 1 week, so there are no data on whether the benefits of training are maintained in the medium or long term. Second, the study did not assess whether the intervention changed diagnostic status. Third, the sample consisted of dysphoric individuals rather than exclusively patients with a diagnosis of major depression, limiting the generalizability of the findings. Fourth, the concreteness training differs somewhat from a full CBT treatment, such that it is premature to be confident that this mechanism is active in CBT or that other mechanisms are not more important in CBT. Nonetheless, concreteness training can be viewed as a more explicit elaboration of an element within CBT, namely encouraging patients to describe situations in specific and concrete detail. As such, it is not implausible that the benefits observed for concreteness training may also apply within full CBT. One avenue for future research is a dismantling study of CBT in which the specificity element is compared with other elements of CBT such as thought challenging. If the specificity-as-mechanism hypothesis is further supported, it suggests the value of CBT becoming even more explicitly focused on

making both therapist and patient more specific.

Discussion

This article described two state-of-the-art examples of how the mechanisms of CBT can begin to be investigated by developing interventions that are focused and targeted on processes of interest as identified by theoretical models and experimental research. The work summarized provides some tentative evidence about potential mechanisms underpinning CBT for depression: the results are consistent with a reduction in rumination and an increase in specificity as potential mechanisms of action for CBT in treating depression. Moreover, these processes are probably not independent because pathological rumination is characterized by more abstract and general processing (Watkins, 2008a). Indeed, this work suggests that training individuals to think more specifically and concretely reduces depressive rumination (Watkins et al., 2009; Watkins & Moberly, 2008). It remains unresolved whether this causal relationship is bidirectional, such that reducing rumination would also cause individuals to become more specific in their thinking, although this seems plausible given that rumination ("being stuck in your head") may reduce attention to the external world and thereby reduce awareness of contextual details. It is also probably most accurate to consider concreteness training as one of several potential means to teach people to ruminate less. As the RFCBT approach illustrates, there are a number of ways to engender a more helpful form of ruminative self-focus in patients, each derived from the particular functional analysis of the patient, including increasing specificity of thinking as well as replacing avoidance behaviours with approach behaviours.

Nonetheless, it is clear that this avenue of research is still preliminary, with the findings to date providing necessary but insufficient evidence to support these hypotheses. Moreover, these examples illustrate the complexity and difficulty of researching the mechanisms of therapy. Nonetheless, it is hoped that these examples indicate the value of translational research to identified vulnerability processes and of developing interventions that are focused on specific putative mechanisms as a

means to further clarify our understanding of how therapy works and thus to improve the efficacy of treatments.

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References

- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.
- Beck, A. T. (1976). *Cognitive therapy and emotional disorders*. New York: Meridian.
- Carver, C. S. (1998). Generalization, adverse events, and development of depressive symptoms. *Journal of Personality*, *66*, 607–619.
- Carver, C. S., & Ganellen, R. J. (1983). Depression and components of self-punitiveness: High standards, self-criticism, and overgeneralization. *Journal of Abnormal Psychology*, *92*, 330–337.
- DeRubeis, R. J., & Feeley, M. (1990). Determinants of change in cognitive therapy for depression. *Cognitive Therapy and Research*, *14*, 469–482.
- Feeley, M., DeRubeis, R. J., & Gelfand, L. A. (1999). The temporal relation of adherence and alliance to symptom change in cognitive therapy for depression. *Journal of Consulting and Clinical Psychology*, *67*, 578–582.
- Jacobson, N. S., Dobson, K. S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., et al. (1996). A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology*, *64*, 295–304.
- Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*, 877–883.
- Manber, R., Arnou, B., Blasey, C., Vivian, D., McCullough, J. P., Blalock, J. A., et al. (2003). Patient's therapeutic skill acquisition and response to psychotherapy, alone or in combination with medication. *Psychological Medicine*, *33*, 693–702.
- Martell, C. R., Addis, M. E., & Jacobson, N. S. (2001). *Depression in context: Strategies for guided action*. New York: Norton.

- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569–582.
- Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*, 504–511.
- Paykel, E. S., Scott, J., Teasdale, J. D., Johnson, A. L., Garland, A., Moore, R., et al. (1999). Prevention of relapse in residual depression by cognitive therapy: A controlled trial. *Archives of General Psychiatry, 56*, 829–835.
- Raes, F., Hermans, D., Williams, J. M. G., & Eelen, P. (2006). Reduced autobiographical memory specificity and affect regulation. *Cognition and Emotion, 20*, 402–429.
- Watkins, E. (2008a). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*, 163–206.
- Watkins, E. R. (2008b). *Rumination-focused cognitive behaviour therapy for residual depression: A pilot randomized controlled trial*. Manuscript submitted for publication.
- Watkins, E. R., Baeyens, C. B., & Read, R. (2009). Concreteness training reduces dysphoria: Proof-of-principle for repeated cognitive bias modification in depression. *Journal of Abnormal Psychology, 118*, 55–65.
- Watkins, E. R., & Moberly, N. J. (2009). Concreteness training reduces dysphoria: A pilot proof-of-principle study. *Behaviour Research and Therapy, 47*, 48–53.
- Watkins, E. R., Moulds, M., & Moberly, N. J. (2008). Processing mode causally influences emotional reactivity: Distinct effects of abstract versus concrete construal on emotional response. *Emotion, 8*, 364–378.
- Watkins, E. R., Scott, J., Wingrove, J., Rimes, K. A., Bathurst, N., Steiner, H., et al. (2007). Rumination-focused cognitive behaviour therapy for residual depression: A case series. *Behaviour Research and Therapy, 45*, 2144–2154.
- Williams, J. M. G., Barnhofer, T., Crane, C., Hermans, D., Raes, F., Watkins, E., et al. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin, 133*, 122–148.