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# Introduction to the Special Issue: Cognitive Mechanisms of Change in the Treatment of Depression

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### Running head: CHANGES IN SELF-SCHEMA STRUCTURE

# Introduction to Special Issue: Cognitive Mechanisms of Change in the Treatment of Depression

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#### **Abstract**

Depression is a highly debilitating and recurrent mental health condition. Efforts to understand the mechanisms of cognitive change in the treatment of depression are important to optimize psychotherapy outcome and to prevent relapse and recurrence. The articles in this special issue examine cognitive change in cognitive behavioral therapy by incorporating clinical samples and clinical settings, utilizing empirically supported assessment instruments, and protocolized psychotherapy techniques, and employing methodologies and statistical strategies designed to address questions related to cognitive mechanisms in treatment outcome. These articles examine the role of cognitive processing, structure, and content over the course of cognitive therapy for depression and evaluate the impact of positive and negative events on treatment outcomes.

# Introduction to Special Issue: Cognitive Mechanisms of Change in the Treatment of Depression

Depression is among the most common and costly of mental health problems, with 1-year and lifetime prevalence rates as high as 7% (Kessler, Chiu, et al., 2005) and 21% (Kessler, Berglund, et al., 2005), respectively. According to the World Health Organization (2013), depression affects more than 350 million individuals worldwide and represents the leading global contributor to disability. Major depressive disorder (MDD) is characterized by a cluster of symptoms that include sadness or loss of interest, impaired concentration, disturbed sleep, alterations in appetite or weight, psychomotor retardation or agitation, loss of energy or fatigue, worthlessness, self-blame or excessive guilt, and suicidality (APA, 2013). MDD is a debilitating (e.g., Hopkins, David, & Kilik, 2014) and highly recurrent (e.g., Boland & Keller, 2009) problem. The risk of subsequent occurrences increases exponentially with each episode (Boland & Keller, 2009; Kessing, 1998; Solomon et al., 2000). As such, prevention of relapse and recurrence represents an important treatment goal in addition to the amelioration of current symptomatology.

Cognitive-behavioral therapy (CBT) is true success story in contemporary psychology (Hofmann, Asmundson, & Beck, 2013) and represents one of the most important developments in the treatment of depression (Young, Rygh, Weinberger, & Beck, 2014). Considerable evidence supports the efficacy of this approach both for the treatment of a current depressive episode and for the prevention of relapse. Numerous randomized controlled trials have found that cognitive behavioral therapy is highly effective for the treatment of depression (see Beck & Dozois, 2011; Butler, Chapman, Forman, & Beck, 2006). CBT is equivalent to other evidence-based psychotherapies (e.g., interpersonal psychotherapy, behavior therapy; e.g., Dobson et al.,

2008; Quilty, McBride, & Bagby, 2008) and to antidepressant medications for the acute treatment of a depressive episode. CBT also hold its own against pharmacotherapy for more severe depression (DeRubeis, Gelfand, Tang, & Simons, 1999; DeRubeis et al., 2005; Hollon et al., 2005).

In addition to its overall treatment efficacy, CBT also exhibits an added prophylactic effect relative to antidepressants. Gloaguen, Cottraux, Cucherat, & Blackburn (1988), for instance, reported that the average risk of relapse (based on follow-up periods of one to two years) was 60% following antidepressant treatment and 25% following CBT. In terms of the prevention of relapse, CBT appears to be at least as efficacious as continuance medication (Dobson et al., 2008; Hollon et al., 2005).

Although CBT is a highly effective and sustainable treatment for depression, the specific variables that account for its short- and long-term success are not presently known. A number of investigations have examined changes in cognitive indices over the course of CBT. CBT for depression is, for example, associated with significant reductions in dysfunctional attitudes, negative attributions, and hopelessness (see Dozois & Bieling, 2010). Garratt, Ingram, Rand, and Sawalani (2007) concluded that the empirical literature is generally consistent with the notion that cognitive therapy yields cognitive change which, in turn, predicts reductions in depressive symptomatology. Tang and DeRubeis (1999) also demonstrated that substantial reductions in depressive symptoms (termed "sudden gains") were preceded by significant cognitive shifts, such as when patients modified a maladaptive core belief. These findings have been replicated in subsequent studies (see DeRubeis, Webb, Tang, & Beck, 2010, for review). Although some findings have supported cognition as an important element of therapeutic change, researchers are

far from understanding the specific mechanisms that account for improvement and the prevention of recurrence (Webb, Auerbach, & DeRubeis, 2012).

Numerous studies have shown that certain psychological interventions are effective for a variety of mental health conditions. What we don't understand well is *why*. Increased research on mechanisms of change is important and could better help clinicians to determine which ingredients to emphasize in therapy (Kazdin, 2008). Demonstration of a connection, for instance, does not necessarily inform us about why such an association exists. The knowledge that gender is a risk factor in depression, for example, does not elucidate the reasons *why* females are at greater risk than males (Ingram & Price, 2010). Similarly, knowing that a treatment works does not ensure that we understand why it is efficacious. By understanding mechanisms of change, researchers and clinicians will be better positioned to emphasize the key ingredients of change, capitalize on and match patients to variables that are critical to outcome (Kazdin, 2008) and administer treatment "doses" in a manner that will maximize resources. Research on mechanisms of change can inform CBT practice in terms of which treatment components will maximize benefit and reduce risk of relapse and recurrence.

The manuscripts in this special issue on "Cognitive Mechanisms of Change in the Treatment of Depression" are well positioned to advance this discussion. The contributors to this special issue are leading researchers in cognitive theory and therapy. In addition, the studies reported examine cognitive change in CBT by incorporating clinical samples and clinical settings, utilizing empirically supported assessment instruments, and protocolized psychotherapy techniques, and employing research designs and statistical strategies that are precisely suited to respond to the research objectives. Three articles (Dozois et al.; Quilty, Dozois, Lobo, Ravindran & Bagby; Vittengl, Clark, Thase & Jarrett) examine the role of cognitive processing, structure,

and/or content over the course of cognitive therapy for depression. The remaining two contributions examine the impact of both positive (sudden gains; German, Lorenzo-Luances, & DeRubeis) and negative (life stressors) events on treatment outcomes (Hawley, Zuroff, Brozina, Ho, & Dobson).

Dozois et al. examined schema content and organization in 42 outpatients with major depressive disorder who received cognitive therapy plus antidepressant medication (CT+ADM) or antidepressant medication (ADM) alone. Participants completed a measure of core beliefs and an index of cognitive organization that utilized a card sort methodology. Although the treatment groups were comparable on a number of measures, patients who received CT+ADM showed greater cognitive organization of positive self-referent content than did individuals who received ADM alone, and exhibited an increased sense of self-control or efficacy, which is associated with depression relapse (Vittengl et al., 2010).

Quilty et al. examined cognitive processing and structure in 104 outpatients who received CBT or ADM for MDD. Patients completed measures of depressive symptomatology, information processing and cognitive structure before, during and after treatment. Most measures of cognitive processing and structure changed to a similar degree across both treatments.

Moreover, evidence for the mediating role of cognition was limited, and not specific to CBT.

Vittengl et al. investigated changes in cognitive content in 523 outpatients with recurrent MDD who received cognitive therapy. These researchers also examined the extent to which improvement in cognitive content might account for decreases in depressive symptomatology (as well as testing the reverse relation). Measures of cognitive content changed substantially over treatment; however, evidence for cognitive mediation of symptom reduction was limited and equivalent to evidence for symptom mediation of cognitive content improvement.

These studies suggest that evidence for causal role of cognition remains mixed. First, changes in cognition may not be specific to CBT. For example, patients with maladaptive cognitive functioning were assisted by medications administered according to best practices. This seems to be the case even for cognitive organization which has historically demonstrated greater stability and promise as a vulnerability factor for depression. Second, the degree to which changes in cognition mediate improvements in symptomatology is unclear. The jury is still out on this question, however, as there are numerous methods by which to evaluate the causal or mediating role of cognition and the conclusions drawn will be influenced by both the definitions and methodology chosen.

Future research is necessary to examine the impact of cognitive change on the improvement of depression and its relapse/recurrence. Although the articles in this special issue included diverse assessment procedures, measures of depression often incorporate cognitive constructs. Strategies to reduce this confound and potential tautologies are riddled with complexities. Additional research using multitrait, multimethod approaches (including brain imaging techniques, self-report and laboratory indices of relevant cognitive constructs) to assess cognitive change in treatment is warranted. The timeframe of when cognitive and depression change is assessed is also likely to be critical. In this regard, research would benefit from a comprehensive assessment battery that is administered at different times over the course of treatment (including in-session change). Such research would, of course, be incredibly complex to conduct as researchers would need to take into account issues related to, among other things, repeated measurement. Alternate etiological models – including reciprocal causal pathways and common causes – are difficult to test empirically but may be more consistent with accumulated results. Finally, the use of priming in treatment outcome research is recommended as it enhances

the accessibility of cognitive structures, processes, and products. Such cognitive reactivity following a sad mood induction has been shown to predict relapse/recurrence in individuals who were no longer depressed (Segal et al., 2006) and may be an even more important element of change than the modification cognitive content and structure itself.

The articles of German et al. and Hawley et al. are also important contributions to this special issue as they examine the influence sudden gains and life stressors, respectively, on treatment outcome. German et al. examined the attributions of 46 patients to sudden gains experienced during CBT for MDD, and the association between these attributions and treatment outcomes. Using a novel coding system, these researchers reported that patients most commonly attribute sudden gains to cognitive, behavioural/environmental, or problem-solving causes; attributions to problem solving and behavioral or environmental changes were associated with less sustained improvement in the medium and long-term. German et al. suggested that, at least in CBT, "a gain may be more likely to lead to further improvement when a cognitive change is associated with the gain, relative to when the gain is attributed to non-cognitive phenomena."

Finally, Hawley et al. examined the how perfectionism and life stressors combine to influence the maintenance of treatment gains in patients followed for 24 months after CBT for MDD. Only patients with high self-critical perfectionism after treatment exhibited vulnerability to negative life events (i.e., increases in depression following life stressors). These findings highlight the importance of individual differences in understanding long-term treatment outcomes.

Patient individual differences relevant to resiliency versus vulnerability are important to model when testing the maintenance of gains during and after treatment. Lasting change may be

contingent on the capacity of a treatment to address a long-term vulnerability and/or of a patient to recognize the source of his or her improvement.

In summary, the articles in this special issue provide an interesting array of findings related to cognitive mechanisms of change in the treatment of depression. These articles also raise some important methodological and conceptual issues that need to be addressed in future research. We hope that this series of articles will be of interest to the readership of this journal and will stimulate further research and development in this important area of treatment change.

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