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TREATMENT APPLICATIONS

Cognitive Behavioural Therapy for Mood Disorders

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Abstract. This paper provides a selected review of recent studies highlighting key aspects of mood disorders research. Cognitive models and clinical trials of cognitive therapy of depression are evolving and adapting to increase applicability to the spectrum of depressive symptoms and syndromes experienced by clients, as well exploring beyond acute phase treatment to relapse prevention. In contrast, cognitive models of bipolar disorders and the effectiveness of the therapy are unclear and there are more questions than answers for researchers in this area.

Keywords: Cognitive therapy, mood disorders, depression, bipolar disorders.

Introduction

Beck (2005) recently highlighted the breadth of research on cognitive models of psychopathology and noted a substantial body of evidence supporting the theory of depression. In contrast, cognitive behavioural therapy (CBT) for bipolar disorders (BP) is a "work in progress".

There are now a number of CBTs for mood disorders that follow the "theoretical conceptualization-therapy development" framework including those predominantly focused on learning theory or behavioural activation (BA), predominantly cognitive models such as Cognitive Therapy (CT/CBT), and models incorporating additional elements such as Cognitive Behavioural Analysis System Psychotherapy (CBASP) and Mindfulness-Based Cognitive Behavioural Therapy (MCBT). The majority fulfill the criteria for "well-established" empirically supported therapy (EST). Recognition as an EST means the efficacy has been established in two or more carefully designed methodologically reliable randomized controlled trials (RCTs) that evaluate the treatment of a specific disorder. Whilst there are some concerns about employing criteria mostly employed in pharmacotherapy research, it is partly because the CBT community has been prepared to be judged by such standards that CBT, more than any

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other therapy, has a perceived evidence-base that ensures its inclusion in treatment guidelines for mood disorders (e.g. National Institute of Clinical Excellence).

Space limitations dictate that we discuss selected recently published theory and therapy research. Whilst we focus on RCT research, there is a great deal of empirical support for CBT in the treatment of depression from open trials, case series and studies using other methodologies (Scott and Beck, 2008). For unipolar disorders recent outcome and process research is reviewed. In BP, outcome research and ongoing attempts to clarify the cognitive model are explored.

Depression

Outcome research

A number of meta-analytic studies (e.g. Butler, Chapman, Foreman and Beck, 2006) show that, compared to waiting list or placebo treatments, CBT is highly effective for depressive disorders, but is equivalent or only marginal superior to other active treatments. Whether the treatment effects maintain post-therapy is more difficult to determine as few CBT RCTs have adequate statistical power to assess reliably durability of any gains at follow-up. Despite this, Butler et al. (2006) concluded CBT has persistent benefits and reduces relapses in unipolar depression. However, there are still limitations in our understanding and several issues need to be addressed.

Ameliorating symptoms. Recent RCTs of CBT for depressive symptoms (cf. depression meeting diagnostic criteria) are limited to comparisons with treatment as usual/waiting list. A meta-analysis of seven studies (Cuijpers, Smit and van Straten, 2007), with most using CBT approaches, found that significant post-therapy benefits were lost at 6 and 12-month follow-up. Many individuals treated in primary care or non-specialist settings have undifferentiated, mixed depressive symptoms not meeting diagnostic criteria. Thus, we need to explore further which CBT approaches best meet their needs, identify the factors resulting in reduced benefit post-therapy, and investigate the impact of therapists' level of expertise.

Acute depressive syndromes. There is a good deal of consistent evidence to support the efficacy of CBT, BT, and BA over waiting list, drug placebo, treatment as usual (TAU), or other treatment control conditions (Wampold, Minami, Baskin and Cullen Tierney, 2002). However, Wampold et al. reported more modest benefits for CBT over "attention control" therapies. Who should receive CBT in preference to other EST or medication and/or how to predict differential benefit still needs to be established (Scott and Beck, 2008).

Severe depression. Debate continues regarding the efficacy of CBT for severe depression (Scott, 2000). Guidelines generally suggest pharmacotherapy plus CBT is preferable to CBT alone. A recent two-centre placebo-controlled RCT suggested that although CBT showed equal efficacy to antidepressants, this was mainly because therapy at one centre with a strong clinical and research track record in CBT compensated for its reduced efficacy at the second centre (DeRubeis et al., 2005).

CBT has been found to be more effective than inter-personal therapy (IPT) for some severe depressions (Luty et al., 2007), but less effective than BA in others (Dimidjian et al., 2006). Therapist expertise has been inadequately studied in RCTs for depression, but the available evidence suggests that competency is positively associated with client outcomes in moderate

to severe depression and/or more complex presentations (DeRubeis et al., 2005; Scott and Beck, 2008). These studies also indicate that behavioural approaches are more beneficial than predominantly cognitive or inter-personal approaches for some clients. Clearly these findings need to be replicated, but they indicate that CBT for severe depression may require modification to, for example, compensate for attentional impairments.

Chronic depression. Combined CBT and medication is recommended for chronic depression in most treatment guidelines. For clients whose depression persists despite adequate pharmacotherapy, adjunctive CBT results in significantly fewer relapses over 18 months, with some benefits maintaining for up to 3 years post-CBT (Paykel et al., 1999, 2005). CBT modified to focus more on rumination has produced encouraging results and the possibility of briefer 8-session CBT (Watkins et al., 2007).

A large-scale RCT of CBASP in clients with $\geqslant$2 years of depression found equivalent remission and recovery rates for therapy and antidepressants, but significantly better outcomes for the combination (Keller et al., 2000). Further analysis shows that CBASP is efficacious for medication non-responders (and vice versa). Thus, the strategy of "switching" is supported empirically in chronic disorders (unlike in acute depression STAR*D study). Interestingly, CBASP alone is as effective as the combined treatment and more effective than medication in a particular subgroup of individuals with a history of abuse and neglect.

Prevention of relapse and recurrence. A review of eight studies reported a 30% relapse rate for CBT over 1–2 years compared with 60% for maintenance antidepressants (Gloaguen, Cortraux, Cucherat and Blackburn, 1998). Critics have argued that the analysis did not distinguish clients who continued with prophylactic medication from those who did not. However, an RCT that addressed this issue still found that acute CBT-responders were less likely to relapse over the following year (24%), compared with medication-responders who either had their antidepressant withdrawn (69%) or continued on antidepressants (58%) (Hollon et al., 2005).

Risk of recurrence is higher in individuals with many previous episodes, so Jarrett et al.'s (2001) findings of greater benefit from acute plus maintenance CBT compared with acute CBT are noteworthy. Bockting et al. (2005) found that CBT significantly reduced relapses over 24 months in clients in remission with $\geqslant$5 previous episodes (relapse 46% v 72%), mirroring findings on MCBT in clients in remission with $\geqslant$3 previous episodes (e.g. Ma and Teasdale, 2004). Whilst these studies provide grounds for optimism, future studies of relapse must recruit samples that give adequate statistical power to detect real differences during the post-CBT rather than the acute phase.

Process research

Beyond establishing CBT efficacy and effectiveness, it is important to determine the mechanisms, moderators and predictors of response. The alliance between therapist and client, and the client's pre-treatment expectations of effectiveness predict active engagement in, and speed of improvement with CBT. However, finding specific answers to questions such as "How does therapy work?", "What determines/predicts the efficacy of therapy?", and "How do we target therapy more effectively?" are essential to the process of matching CBT to clients' needs. Some clinically observable and measurable phenomena that may relate to CBT outcome are highlighted below.

Tang and DeRubeis' original work on "sudden gains" found that some depressed clients undergoing CBT showed substantial symptom improvement in a single between-session interval. Research confirms that these sudden gains usually occur early in therapy, are associated with better long-term outcomes, and are not measurement artefacts (Tang, DeRubeis, Hollon, Amsterdam and Shelton, 2007). So understanding this phenomenon may increase our ability to prevent relapse. Hayes et al. (2007) investigated the phenomenon of transient worsening and found that these "depression spikes" also predicted lower post-treatment depression. Weekly diaries also indicated that clients with spikes show more cognitive-emotional processing during this period of arousal than those without. This study confirms the clinical observation that transient worsening is not necessarily indicative of poor outcome.

Several studies have indicated that changes in the processing of depression-related information rather than in thought content may be important in the mechanism of action of CBT. Teasdale et al. (2000) reported that CBT modifies the form of thinking in chronic depression and reductions in all-or-nothing thinking style mediated its relapse prevention effect. This is consistent with the notion that CBT helps clients to acquire compensatory or meta-cognitive skills, hinting that shifting the mode of processing may be the critical factor. Studies have also found that improvements in a specific cognitive skill, "situational analysis", early in CBT predicted level of depression at termination.

Fresco, Segal, Buis and Kennedy (2007) found that CBT-responders exhibited significantly greater gains in "de-centring" compared with antidepressant-responders and that high levels of de-centring post-CBT was associated with the lower relapse rates at 18 months. The findings of an experimental study by Singer and Dobson (2007) involving depressed patients in remission suggest that preventative interventions may operate by reducing the intensity of sad moods and altering one's attitudes towards and/or acceptance of temporary moments of sadness.

The above gives a snap-shot of recent process research, but it is important to acknowledge Hollon, Stewart and Strunk's (2006) comments that we remain uncertain whether the durable benefits of CBT are a consequence of the amelioration of the causal processes that generate risk or the introduction of compensatory strategies that offset them, and it is unclear whether these effects reflect the mobilization of cognitive or of other mechanisms.

Bipolar disorders

RCTs of BP have regarded CBT as an adjunct to medication/TAU rather than an alternative. A brief selective review of the available evidence and a discussion of the limitations of the current cognitive model are provided below.

Outcome research

There have been eight RCTs of CBT for relapse prevention since Cochran's (1984) small but successful brief intervention to enhance lithium adherence. We review a selection, starting with the only unequivocal findings to date (Lam et al., 2000, 2003). Lam reports two RCTs of CBT plus TAU vs TAU alone for stably remitted BP clients. Only 44% of clients treated with CBT had a relapse within a year, compared with 75% in the control (Lam et al., 2003). The benefit of CBT plus TAU was maintained to an extent at 2-year follow-up, but between group differences were only apparent for depression and faded over time (Lam, Hayward, Watkins,

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Wright and Sham, 2005). A problem for CBT, evident in this and other studies, is that 20–25 CBT sessions targeted even at remitted clients have relatively circumscribed, time-limited prophylactic effects. Furthermore, it is unclear if any observed benefits are due to specific formulation-driven interventions or result from generic psychoeducation approaches. Recent relapse prevention studies from Canada (e.g. Zaretsky, Segal and Gemar, 1999) report that seven sessions of psychoeducation are as equally effective at 12 months as seven sessions of psychoeducation plus 13 sessions of CBT. Likewise, Meyer and Hautzinger (2005) compared 20 sessions of CBT with 20 of supportive therapy and found 12-month relapse rates of 50% in both groups, with few specific differences between CBT and supportive therapy.

Scott, Garland and Moorhead (2001) and Scott et al. (2006) used a formulation-based CBT approach to address specific individual problems with BP, focusing on acute symptoms as well as relapse prevention. In a pilot study, CBT showed greater reductions in depression, with a trend towards fewer remissions and more relapses in the control group. However, in the largest CBT RCT so far ($n = 254$), adjunctive therapy was not superior to TAU (Scott et al., 2006). Reasons for the lack of treatment effect might relate to the pragmatic 5-centre design of the trial that specifically targeted general adult clients at very high risk of relapse. The authors concluded that when treating BP in routine clinical settings, 22 sessions of CBT were inadequate. A post-hoc analysis showed that individuals at an earlier stage of their BP career benefited significantly more from CBT compared to TAU; and those with fewer episodes showed significantly lower relapse rates in the CBT condition.

There are no RCTs of CBT for hypomania/mania, but two publications explore CBT for acute bipolar depression. In a non-randomized comparison, Zaretsky et al. (1999) showed significant improvements in depression in clients with BP that were comparable with changes for a unipolar depression group. However, unlike the unipolar group, there was no significant change in dysfunctional attitudes in the bipolar group. A recent large RCT of bipolar depression explored the benefits of 30 sessions of adjunctive CBT, modified IPT, or family focused treatment with a 6-session collaborative care approach (Miklowitz et al., 2007). All three therapies led to faster and more frequent remission than collaborative care, but the initial report only included the 3-month post-therapy follow-up, so we do not yet know if therapy gains are maintained.

Cognitive models for bipolar disorders

Given the inconsistent and sometimes disappointing findings for CBT in BP, it is relevant to review the robustness of cognitive models for onset and relapse. Early models lacked an adequate theoretical framework to explain how cognitive vulnerability to mood disorders (e.g. underlying dysfunctional beliefs) could precipitate depression at certain times but mania at another. Also, most early interventions, even when labelled as CBT, consisted of generic interventions to prevent relapse. Despite the acknowledgement that mania and depression might be associated with different risk factors (e.g. Johnson, 2005; Meyer and Maier, 2006), there is still little empirical research that explains this dual vulnerability. Furthermore, in BP, elevated levels of dysfunctional attitudes and/or negative thoughts are often epiphenomena of sub-syndromal depression (e.g. Jones et al., 2005; Lex, Meyer, Marquat and Thau, 2008) that is frequent and represents the main symptom burden in BP (e.g. Mansell, Colom and Scott, 2005). Also, cognitive models often present mania as the polar opposite of depression, but the elated mania prototype is less common than dysphoric, irritable or labile forms.

More sophisticated models of mania/hypomania have recently been proposed that take these issues into account (e.g. Jones, 2001). Approaches adopting a similar strategy as for bipolar depression, by trying to specify mania-related cognitions and dysfunctional attitudes, have found evidence that global and stable attributions for success and failure can increase symptoms of depression and mania in individuals "at risk" for, but without diagnosed BP (e.g. Alloy, Reilly-Harrington, Fresco, Whitehouse and Zechmeister, 1999). Observed disruptions in circadian rhythms has led to hypotheses that misattributions of associated biological dysrhythmias (e.g. decreased sleep) trigger a spiral of behavioural activation and/or changes in mood and self-esteem that progress to a mania-like state (e.g. Scott, 2007; Meyer, in press). Further, goal-related cognitions and a "hyper-positive sense of self" are proposed as specific triggers/personality styles that increase the risk of manic relapse (e.g. Johnson, 2005: Lam, Wright and Sham, 2005). Analogue studies of ascent into mania (e.g. Johnson, Ruggero and Carver, 2005; Meyer and Maier, 2006) offer opportunities to examine cognitive processes without potential confounds such as medication or hospitalizations; however, such findings only have validity for dimensional models of mania. The latter have utility for the bipolar spectrum, but current thinking on mania increasingly identifies activation level rather than mood state or cognitive shift as the primary determinant of polarity of bipolar relapse. This is likely to mean that cognitive interventions will be targeted at modifying dispositional or situational cognitions that arise at the beginning of a relapse prodrome rather than there being a classic mania-vulnerability triad. In the end, CBT may well be better targeted at the type II variant of BP, where the clinical picture is dominated by acute depression and residual symptoms that are only partially responsive to medications (Mansell et al., 2005).

Concluding comments

Kraemer, Wilson, Fairburn and Agras (2002) suggest that RCTs should now routinely include an evaluation of the mediators and moderators of outcomes. This is an attractive proposition for studies of depressive disorders, but is premature for BP where more detailed theoretical work and coherent modelling are required to produce CBT interventions with greater specificity and durability. Research in mood disorders is advancing towards exploration of the mind-brain interface with fMRI scanning studies of those receiving CBT or medication to look at the locus and process of change in cognitive-emotional networks. With a few exceptions (e.g. Goldapple et al., 2004) this research is biased by allocation of subjects to preferred treatments and a paucity of pre-study hypotheses. Nonetheless, there are the beginnings of consistency in differential observed patterns of changes with CBT and medication that opens up exciting opportunities for future empirical research on the neuro-scientific underpinnings of CBT (Miterschiffthaler, Williams, Scott and Fu, 2008).

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