Cognitive Theory and Therapy of Bipolar Disorders

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Introduction

This paper will explore the evolution of cognitive theory and therapy for individuals with bipolar disorder. Unlike most of the other papers in this issue, there is a relatively smaller body of research data available on these topics. Until recently, bipolar disorders were widely regarded as a biological disorder best treated with medications (Prien & Potter, 1990; Scott, 1995a; Scott & Colom, 2005). This view is gradually changing for two reasons. First, in the past three decades, there has been a greater emphasis on stress-diathesis models. This has led to the development of new etiological theories of severe mental disorders that emphasise psychological and social aspects of vulnerability and risk. It has also increased the acceptance of brief psychological therapies, such as cognitive therapy, as an adjunct to medication for individuals with treatment-resistant schizophrenia, and severe and chronic depressive disorders (Scott & Wright, 1997). Second, there is a significant efficacy-effectiveness gap for pharmacological treatments for bipolar disorder (Guscott & Taylor, 1994; Tacchi & Scott, 2005). Mood stabilizer prophylaxis protects about 60 % of individuals against relapse in research settings, but protects only 25–40 % of individuals against further episodes in clinical settings (Dickson & Kendall, 1986). The introduction of newer medications has not improved prognosis (Scott, 1995a; Tacchi & Scott, 2005). This has also increased interest in other treatment approaches in bipolar disorder.

This paper explores key aspects of cognitive models of bipolar disorder, especially research on dysfunctional attitudes and beliefs; it then comments on the clinical applicability of cognitive therapy for bipolar disorder and reviews the outcome studies available. The final section offers an overview of key areas for further research.

Cognitive Models of Bipolar Disorder

Early Descriptions

Beck's original cognitive model (1967) suggests that depressed mood states are accentuated by patterns of thinking that amplify mood shifts. For example, as people become depressed they become more negative in how they see themselves, their world and their future (called the negative cognitive triad). Hence they tend to jump to negative conclusions, overgeneralize, see things in all-or-nothing terms, and personalize and self-blame to an excessive degree (cognitive distortions). Changes in behaviour, such as avoidance of social interaction, may be a cause or a consequence of mood shifts and negative thinking. Cognitive vulnerability to depression is thought to arise as a consequence of dysfunctional underlying beliefs (e.g. "I'm unlovable") that develop from early learning experiences, and drive thinking and behaviour. It is hypothesised that these beliefs may be activated by life events that have specific meaning for that individual. For example, an individual who holds a belief that "I'm unlovable" may experience depression in the face of rejection by a significant other.

Beck's original description suggested that mania was a mirror image of depression and was characterised by a positive cognitive triad of self, world, and future, and positive cognitive distortions. The self was seen as extremely loveable and powerful with unlimited potential and attractiveness. The world was filled with wonderful possibilities, and experiences were viewed as overly positive. The future was one of unlimited opportunity and promise. Hyper-positive thinking (stream of consciousness) was typified by cognitive distortions, as in depression, but in the opposite direction. For example, jumping to positive conclusions, such as "I'm a winner" and "I can do anything"; under-estimating risks, such as "There's no danger"; minimising problems, such as "Nothing can go wrong"; and overvaluing immediate gratification, such as "I will do this now". Thus, cognitive distortions provided biased confirmation of the positive cognitive triad of self, world and future. Positive experiences were selectively attended to, and it was hypothesised that in this way underlying beliefs and self-schema that guide behaviours, thoughts and feelings were maintained and strengthened. Examples of such underlying beliefs and self-schema include: "I'm special" and "Being manic helps me to overcome my shyness".

In contrast to Beck's model of depression, there have been relatively few attempts to confirm or refute his ideas about mania through research. Beck's original model of mania was largely derivative, based on the careful observation of individuals in a manic state. It took a traditional stance, viewing mania as the polar opposite of depression. Neither mixed states nor dysphoric mania were considered. There are gaps in this early model. For example, there is no discussion about any similarities or differences in the specific dysfunctional beliefs held by individuals with bipolar disorders as compared to unipolar disorders and the role of personality styles (sociotropy and autonomy) was not incorporated. Also, the nature of life events that may "match" certain beliefs and uniquely precipitate mania as opposed to depression remained unexplored. However, it is important to see the model in context. It was a useful step forward from psychoanalytic models, and has recently provided an important starting point for the research that is now reviewed below.

Empirical Evidence

This section reviews studies of cognitive models of bipolar disorder. It is organized into two sub-groups: those exploring cognitive style and those exploring dysfunctional beliefs and the role of "matching" life events in symptom exacerbation in clinical and non-clinical samples.

Descriptive Studies

Most studies of cognitive models in bipolar disorder have used the model of unipolar disorder as a template. As such, the early research comprised cross-sectional studies comparing subjects with bipolar disorder with other client populations on measures such as dysfunctional beliefs, self-esteem and cognitive processing. Apart from one early study, data on dysfunctional attitudes in individuals with bipolar disorder who were not currently manic demonstrate a similar pattern to that seen in individuals in the euthymic and depressive phases of unipolar disorder. Unfortunately, there is limited data on changes during the manic phase. Silverman, Silverman and Eardley (1984) explored dysfunctional attitudes using the Dysfunctional Attitudes Scale (DAS; Weisman & Beck, 1978) in a heterogeneous clinical sample and suggested that individuals with bipolar disorder who were currently euthymic showed lower levels of dysfunctional attitudes than all other diagnostic groups. These findings were not supported by Scott, Stanton, Garland and Ferrier (2000). Silverman and colleagues also demonstrated that in a depressive phase, unipolar and bipolar subjects showed a similar increase in lev-els of dysfunctional attitudes. A finding confirmed by Hollon, Kendall and Lumry (1986)

who reported that, compared to healthy control subjects, individuals with either unipolar or bipolar depression showed higher levels of dysfunctional attitudes and negative automatic thoughts. However, there were no significant differences between the unipolar and bipolar groups when individuals were depressed or in remission. Alloy, Reilly-Harrington, Fresco, Whitehouse and Zeichmeister (1999) found that attributional style and dysfunctional attitudes were similar in individuals with cyclothymia and dysthymia and were more negative than normal controls. It is not known how these findings relate to major depressive disorders or bipolar disorder, but individuals with cyclothymia and dysthymia have a greater risk of major affective disorder than individuals in the general population. In a study by Hammen, Ellicott and Gitlin (1992), unipolar and bipolar subjects who were asymptomatic did not differ on measures of sociotropy or autonomy.

Only two studies have explored mood congruent or mood dependent memory in individuals with bipolar disorder. Weingartner, Miller and Murphy (1977) showed that eight subjects who experienced several cycles of mania and depression were more able to retrieve memories when their mood at testing matched their mood at encoding. Recall when mood at testing matched mood at production was twice as high (35 % versus 18 %) as when there was a mismatch. More recently, Eich, Macauley and Lam (1997) assessed mood dependant memory in ten individuals with rapid cy-cling bipolar disorder. They noted that, like healthy controls, their subjects showed mood dependent changes in autobiographical memory recall. However, their subjects also demonstrated mood-dependant recognition, an effect that healthy controls rarely show. Eich and colleagues propose that this effect may arise because subjects with BP experience stronger more intense moods.

Scott and colleagues (2000) explored several aspects of the cognitive model simultaneously, including dysfunctional attitudes, positive and negative self-esteem, autobiographical memory and problem-solving skills. The sample comprised of individuals with bipolar disorder (n = 40) who were rated by an observer as euthymic and a matched control group of healthy subjects (n = 20). Interestingly, although reported levels of observer rated manic and depressive symptoms in subjects with bipolar disorder were minimal, self-rated depression scores suggested significant levels of residual dysphoria and/or ongoing sub-syndromal depression. A finding confirmed by the rigorous 12-year follow-up of Judd et al. (2002) who reported that individuals with bipolar disorder have sub-syndromal depressive symptoms for almost half of the time.

In comparison to healthy controls, clients with bipolar disorder had more fragile, unstable levels of self-esteem, and higher levels of dysfunctional attitudes (particularly related to need for social approval and perfectionism). These statistically significant differences persisted when current depression ratings were taken into account. Within the bipolar disorder group, those individuals who had multiple previous affective episodes and/or earlier age of onset of bipolar disorder showed the greatest level of cognitive dysfunction. Scott et al. (2000) argue that, although it was not possible to determine whether these abnormalities of cognitive style were a cause or a consequence of relapse in bipolar disorder, it was noteworthy that these differences from healthy controls persisted in clients who were currently adherent with prophylactic medication. This suggests that long-term medication alone may not extinguish cognitive and affective symptoms nor fully protect against relapse. Furthermore, the researchers noted that clients with bipolar disorder showed a similar cognitive style to individuals with unipolar depression.

To explore further whether dysfunctional beliefs in individuals with bipolar disorder is similar to that observed in unipolar disorder, Scott and Pope (2003) identified 77 individuals with bipolar disorder who were currently being treated in secondary care and were receiving regular mood stabilizer medication, and compared their cognitive style to 17 individuals with unipolar disorder

who were receiving similar treatment (to try to ensure differences could not simply be explained by differences in severity of disorder or treatment approach). When current symptoms and other demographic and clinical characteristics were controlled for, unipolar and bipolar subjects showed more similarities than differences in cognitive style. Overall scores on the self-esteem rating and the dysfunctional attitudes scale did not show any significant differences. On sub-scale scores, subjects differed on only two measures. Individuals with unipolar disorder had higher mean levels of negative but not positive self-esteem (Negative Self-Esteem mean bipolar = 12.7 < mean unipolar = 15.5; F 3.99, $p = 0.03$) and subjects with bipolar disorder showed higher levels of Preference for Affiliation (mean bipolar = 29.7 > mean unipolar = 25.3; F = 5.2, $p = 0.02$). When subjects with bipolar disorder were classified as depressed (n = 38), hypomanic (n = 13) or remitted (n = 26), some interesting sub-group differences emerged. There was a fairly consistent pattern of remitted subjects with bipolar disorder having the highest mean score for self-esteem, and the lowest for dysfunctional attitudes. This pattern was reversed for depressed subjects with bipolar disorder. The mean scores for hypomanic subjects with bipolar disorder lay between those of the other sub-groups. It was noteworthy that whilst total SEQ scores followed the same pattern (scores for hypomania falling between remission and depression), hypomanic subjects with bipolar disorder recorded the highest mean scores on both the Negative and Positive self-esteem sub-scales.

Other studies have explored self-esteem, and social desirability. Using a repertory grid, Ashworth, Blackburn and McPherson (1982) demonstrated abnormally high levels of self-esteem during mania, low levels in depression and a return to normal levels in remission. Winters and Neale (1985) wrote a highly influential article in which they hypothesized that although subjects with remitted bipolar disorder do not usually report impaired self-esteem, these subjects possess a cognitive schema of low self-esteem. This idea evolved from data suggesting that, although subjects with bipolar disorder and healthy controls showed higher levels of self-esteem than individuals prone to unipolar depression, the bipolar disorder group scored higher than either the unipolar or the healthy control group on measures of social desirability and self-deception. Neale (1988) later proposed that unstable self-esteem coupled with unrealistic standards for success may be predisposing factors for bipolar disorder. Pardoen, Bauwens, Tracy, Martin and Mendlewicz's (1993) study of self-esteem confirmed the presence of social conformism in subjects with bipolar disorder. A key methodological lesson from these studies is the need to consider the use of implicit as well as explicit measures of cognitive style.

Lyon, Startup and Bentall (2000) used explicit and implicit measures of attributional style and a recall measure of self-schema in subjects with bipolar disorder who were currently manic or currently depressed and compared their results with a group of healthy controls. Manic subjects showed a normal self-serving bias on the explicit attributional style questionnaire, attributing positive events more than negative events to self. As predicted, depressed subjects attributed negative events rather than positive events to self. However, on implicit measures, manic and depressed subjects with bipolar disorder both attributed negative events more than positive events to self. On the self-referent encoding memory task, manic subjects were more likely than depressed subjects to endorse positive words as true of themselves. However, in a surprise recall test, both manic and depressed subjects recalled more negative than positive trait words.

Bentall, Kinderman and Manson (2000) measured self-discrepancies in subjects with bipolar disorder who were currently manic, currently depressed or currently in remission and compared them with healthy control subjects. Manic subjects showed higher self-actual:self-ideal consistency than healthy controls, whilst depressed subjects with bipolar disorder showed abnormally high self-

actual:self-ideal discrepancies. Participants in all four groups showed little evidence of discrepancies between how they viewed themselves and how they thought others viewed them.

One of the main difficulties with all the above studies was the problem of sample selectivity. Therefore Scott in collaboration with genetics researchers (Jones et al., 2005), explored dysfunctional beliefs and self-esteem in carefully defined samples of individuals with bipolar disorder (n = 116) and unipolar disorder (n = 265), and compared their ratings with matched controls (n = 264) who did not have a history of mood disorders. There were statistically significant differences between groups on all measures. The patient groups differed significantly from controls but there were few differences in the pattern of results in the unipolar and bipolar probands. The unipolar group showed the lowest levels of self-esteem (low positive, and high negative, self-esteem), and the bipolar group scored significantly lower on the self-esteem ratings than the controls but higher than the unipolar group. The unipolar patients also showed the highest level of dysfunctional attitudes, followed by the bipolar group, followed by controls. This pattern was true for each of the sub-scales of the dysfunctional attitudes scale. When current levels of depression were taken into account no differences emerged between the two patient groups on any of the measures. However, both patient groups still exhibited lower levels of self-esteem, and more dysfunctional attitudes than the control subjects. As such they did not show a pattern of dysfunctional beliefs that is unique to individuals with bipolar disorder.

The above studies identify that unipolar disorder and bipolar disorder are indistinguishable in the depressed phase, sharing a similar cognitive profile that includes abnormalities in information processing, dysfunctional attitudes and attributional style. Remitted subjects with bipolar disorder show similar cognitive style to remitted unipolar subjects on explicit measures, but this is not always as clear on implicit measures. Data on levels of self-esteem is equivocal as variations may be a function of lability or of differences between implicit and explicit ratings. Scott and colleagues have identified that labile self-esteem as compared with the fixed low level of self-esteem may potentially distinguish bipolar disorder and subjects with severe unipolar disorders in the depressed or euthymic phases of disorder. However, unstable self-esteem and low level of self-esteem are both known to confer similar levels of risk for depressive relapse (Kernis, Cornell, Sun, Berry, & Harlow, 1993). Alternatively, the discrepancy between explicit and implicit measures of self-esteem or self-representations in remitted unipolar and subjects with bipolar disorder might be explained if we assume that social desirability schemata are activated even in subjects with bipolar disorder even in the euthymic state. There is minimal evidence that the abnormalities in individuals with bipolar disorder are trait vulnerabilities that specifically predispose to onset of bipolar disorder. It is possible that trait aspects of cognitive style may increase the likelihood of early age of first episode or influence the frequency of recurrence. However, without more detailed longitudinal research little can be said about the stability of cognitive style over time in subjects with bipolar disorder. We will discuss this later in our chapter.

Interactions between Life Events and Dysfunctional Beliefs

According to Beck's model, certain maladaptive core beliefs interact with stressors that carry a specific meaning for the individual, increasing the probability of an affective episode occurring. In their excellent review, Johnston and Miller (1997) confirm the association between adverse life events and either an exacerbation of affective symptoms or relapse into an episode of bipolar disorder. However, only five studies have explored the interaction between aspects of cognitive style and life events. A small early study (Hammen, Ellicott, Gitlin & Jamison, 1989) failed to find support for a cognitive stress-vulnerability model for individuals with bipolar disorder. However, a later study by Hammen, Ellicott, and Gitlin (1992) reported that individuals with bipolar disorder who had high

levels of sociotropy experienced an exacerbation of affective symptoms in response to interpersonal life events. Hammen and colleagues did not identify whether manic or depressive exacerbations were more frequent. In a similar study, Swendsen, Hammen, Heller and Gitlin (1995) then explored the relationship between life stress and personality traits known to be associated with negative cognitive style – namely introversion and obsessionality. They demonstrated that these negative styles interacted with non-specific stressful life events to predict relapse of bipolar disorder.

Three other studies have prospectively explored symptom exacerbation in sub-syndromal bipolar disorder or non-clinical populations. In a sample of 60 bipolar I patients, Johnson and Fingerhut (2004) found that cognitive style, as assessed by the Dysfunctional Attitudes Scale, the Negative Automatic Thoughts Questionnaire and the Positive Automatic Thoughts Questionnaire, predicted increases in depression with time but were not related to mania. However, this study did not assess life events. Alloy, Reilly-Harrington, Fresco, Whitehouse, and Zeichmeister (1999) reported that an internal, stable, global, attributional style interacted with life stress to predict increases in affective symptoms in individuals with sub-syndromal bipolar disorder and unipolar disorder (see this book for further details). In a further study, Reilly-Harrington, Alloy, Fresco, and Whitehouse (1999) screened a non-clinical sample on the General Behavioural Index (Depue, Krauss, Spoont, & Aribisi,1989) for bipolar disorder or the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) for unipolar disorder. Subjects scoring above the established cut-offs were also administered the Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978). Reilly-Harrington and colleagues initially assessed each individual's attributional style, dysfunctional attitudes and negative self-referent information processing, then re-assessed these factors in subjects one month later. In individuals with bipolar disorder, negative cognitive style at initial assessment interacted significantly with high number of negative life events to predict an increase in manic or depressive symptoms. The interaction between dysfunctional attitudes and negative life events accounted for a greater proportion of the variance in symptoms (16 %) than the interaction between attributional style and negative life events (10 %).

The study of Johnston and colleagues (2000) is of particular interest as it fits with much of the current thinking on cognitive and neurotransmitter theories of bipolar disorder and reward systems. Johnston et al. showed that goal attainment life events might specifically precede manic as compared to depressive relapse. This finding appears to concur with information provided by clients in clinical settings and also supports the hypothesis that individuals with bipolar disorder may have abnormalities in the Behavioural Activation System (BAS; e.g. see Depue & Zald, 1993). This system is thought to control psychomotor activation, incentive motivation and positive mood. The BAS may show both higher baseline level of activity and greater day-to-day variability in individuals at risk of bipolar disorder. However, even healthy controls show increases in positive affect and energy following goal attainment life events; suggesting increased BAS activity. Individuals with bipolar disorder show a greater increase in BAS activity, slower return to normal baseline levels and consequently an increase in manic symptoms in the two months after a goal attainment life event. It is hypothesized that the symptoms occur in vulnerable individuals because of a failure to regulate motivation and affect after the trigger.

Summary: It is increasingly recognized that the cognitive-behavioural processes that may maintain psychological disorders are common to several disorders, and can be regarded as "transdiagnostic" (Harvey, Watkins, Mansell, & Shafran, 2004). However, the cognitive content appears to vary substantially across different disorders and is important in distinguishing between disorders (see Harvey et al., 2004, pages 14, 16, 272). Given the very different clinical presentations

seen in manic as compared to depressive episodes, it is perhaps surprising that there are more similarities than differences in the dysfunctional beliefs of individuals with unipolar disorder and bipolar disorder. Most of the studies reviewed earlier have either used a non-clinical control group or a unipolar disorder control group. Fewer studies have used both. Only studies with both control groups can identify factors that are both associated with psychopathology (i.e. elevated with respect to non-clinical controls) and specific to bipolar disorder (i.e. elevated with respect to patients with unipolar disorder). However, the fact that certain studies have used both clinical and non-clinical control groups and yet still found that individuals with bipolar disorder are similar to those with unipolar disorder (e.g. Jones et al., 2005) suggest there may be other explanations for the research findings. For example, it has been noted that patients with bipolar disorder may engage in a form of social acquiescence that may distort their self-ratings (Winters & Neale, 1985) and so it is possible that people with bipolar disorder provide socially desirable answers. Another possibility is that bipolar disorder and unipolar disorder are really not different in any significant way. Certainly both groups of patients experience bouts of depression and a similar range of comorbid psychological disorders. The manic defence hypothesis (e.g. Abraham, 1911) suggests that mania serves as a way to mask or deny the existence of depression, which continues throughout a manic episode. Alternative accounts and more modern reformulations of the manic defence hypothesis (e.g. Bentall, 2003; Blalock, 1936; Neale, 1988) do not propose that mania necessarily masks depression but that the drive into mania is an active process that prevents or suppresses feelings of failure or negative mood states. In some ways this explanation bypasses the question of why certain individuals, i.e. those with bipolar disorder, find themselves becoming manic as a way of avoiding negative experience at certain times in their lives but not at others, whereas those with unipolar disorder never become manic as a method to avoid negative experiences. The lack of current evidence should not be taken as proof of a lack of specificity in the nature of the beliefs held by individuals with bipolar disorder as compared to unipolar disorder. At risk of saying the obvious, researchers can only find what they look for. As patients with bipolar disorder are also at high risk for depression, they are likely to share similar cognitive styles. However, there is preliminary evidence that certain specific items on the DAS may be elevated in patients with bipolar disorders relative to patients with unipolar disorders. Initially, Lam, Wright and Smith (2004) found no differences between patients with bipolar disorders and patients with unipolar disorders on their ratings on short form of the Dysfunctional Attitudes Scale (Power et al., 1994), but a factor analysis led to the identification of three factors: “Goal-Attainment”, “Dependency” and “Achievement”. In another post-hoc analysis, they also found that patients with remitted bipolar disorder reported higher levels of “anti-dependency” beliefs as assessed by two particular items: “I don’t need the approval of others to be happy” and “A person doesn’t need to be liked to be happy”. These items appear to represent the autonomous beliefs that may characterise bipolar patients, especially during an upswing in their mood.

The study of dysfunctional beliefs in bipolar disorder is beginning to provide important insights into the evolution of episodes. It appears that bipolar disorder is similar to other psychological disorders in being associated with elevated levels of dysfunctional beliefs, which is encouraging with respect to the potential of psychological interventions. In addition, there are some indications that certain dysfunctional beliefs may be more specific to bipolar disorder, or activated during elevated mood states, e.g. higher than expected levels of perfectionism characterize individuals with bipolar disorder. Although this is also true of many individuals with chronic depression, eating disorders and chronic fatigue syndrome (Scott et al., 1996; Shafran & Mansell, 2001), its importance cannot be discounted. For example, high levels of perfectionism may predict poor outcome from psychological

or pharmacological treatments and as such are an important target for change in psychotherapy (Scott, 2002). However, to develop specific therapeutic interventions for bipolar disorder we require a considerable amount of further research to fully establish the shared and unique assumptions and beliefs, and most importantly we need a comprehensive account of their implications for the development of mania. This work is ongoing and will hopefully improve further the model of cognitive therapy being practised in bipolar disorder in the future.

Cognitive Therapies of Bipolar Disorders

Brief Overview of Cognitive Therapy

An optimal course of cognitive therapy begins with a cognitive formulation of the individual's unique problems related to bipolar disorder, particularly emphasizing the role of core maladaptive beliefs (such as excessive perfectionism, unrealistic expectations for social approval) that underpin and dictate the content of dysfunctional automatic thoughts and drive patterns of behaviour. This formulation dictates which interventions are employed with a particular individual used and at what stage of therapy that approach is used. Although each individual will define a specific set of problems, Scott (2000), Basco and Rush (1996), Lam et al. (1999) and Newman et al. (2001) have identified several common themes that arise in cognitive therapy for patients with bipolar disorder. These are summarised in Table 1.

Table 1. Common themes arising in cognitive therapy of bipolar disorders

Cognitive therapy may be used to:

1	facilitate adjustment to the disorder and its treatment
2	enhance medication adherence
3	improve self-esteem and self-image
4	reduce maladaptive or high risk behaviours
5	recognise and modify psycho-bio-social factors that destabilize the individuals day-to-day functioning and mood state
6	help the individual recognise and manage psychosocial stressors and interpersonal problems
7	teach strategies to cope with the symptoms of depression, hypomania, and any cognitive and behavioural problems
8	teach early recognition of relapse symptoms and develop effective coping techniques
9	identify and modify dysfunctional automatic thoughts (negative or positive), and underlying maladaptive beliefs
10	improve self-management through homework assignments

At the first cognitive therapy session, the individual is encouraged to tell their story and to identify problem areas through the use of a life chart. Current difficulties are then classified under three broad headings into intra-personal (e.g. low self-esteem, cognitive processing biases), interpersonal (e.g. lack of social network), and basic problems (e.g. symptom severity, difficulties coping with work). These issues are explored in about 20–25 sessions of cognitive therapy that are held weekly until about week 15 and then with gradually reducing frequency. The last two sessions are offered at about week 32 and week 40. These "booster sessions" are used to review the skills and techniques learned. The overall cognitive therapy programme comprises four stages:

(A) *Socialisation into the cognitive therapy model and development of an individualised formulation and treatment goals*: Therapy begins with an exploration of the patients understanding of bipolar disorder and a detailed discussion of previous episodes focusing on identification of prodromal signs, events or stressors associated with onset of previous episodes, typical cognitive and behavioural concomitants of both manic and depressive episodes and an exploration of interpersonal functioning

(e.g. family interactions). A diagram illustrating the cycle of change in bipolar disorder is used to allow the individual to explore how changes in all aspects of functioning may arise. Early sessions include development of an understanding of key issues identified in the life chart, education about bipolar disorder, facilitation of adjustment to the disorder by identifying and challenging negative automatic thoughts, and developing behavioural experiments particularly focused on ideas about stigmatisation and fragile self-esteem. Other sessions include collating accurate information and enhancing understanding about the epidemiology, treatment approaches and prognosis of bipolar disorder, and beginning to develop an individualised formulation of the patient's problems, which takes into account underlying maladaptive beliefs.

(B) *Cognitive and behavioural approaches to symptom management and dysfunctional automatic thoughts*: Using information gathered previously, sessions are used to help people learn self-monitoring and self-regulation techniques, which enhance self-management of depressive and hypomanic symptoms, and to explore skills for coping with depression and mania. For example, this involves establishing regular activity patterns, daily routines, regular sleep patterns, developing coping skills, time management, use of support, and recognising and tackling dysfunctional automatic thoughts about self, world and future using automatic thought diaries.

(C) *Dealing with cognitive and beha-vioural barriers to treatment adherence and modifying maladaptive beliefs*: Problems with adherence to medication and other aspects of treatment are tackled e.g. through exploration of barriers (challenging automatic thoughts about drugs; beliefs about bipolar disorder, excessive self-reliance; or exploring attitudes to authority and control) and using behavioural and cognitive techniques to enhance treatment adherence (Scott, 1999). This and data from previous sessions are used to help the patient identify their maladaptive assumptions and underlying core beliefs, and to commence work on modifying these beliefs.

(D) *Anti-relapse techniques and belief modification*: Further work is undertaken on recognition of early signs of relapse and coping techniques (fortnightly sessions). For example, developing self-monitoring of symptoms, identifying possible prodromal features (the "relapse signature"), developing a list of "at risk situations" (e.g exposure to situations that activate specific personal beliefs), high risk behaviours (e.g. increased alcohol intake), combined with a hierarchy of coping strategies for each; identifying strategies for managing medication intake and obtaining advice regarding it; and planning how to cope and self-manage problems after discharge from cognitive therapy. Sessions also include typical cognitive therapy approaches to the modification of maladaptive beliefs, which may otherwise increase vulnerability to relapse.

Does Cognitive Therapy Improve Outcome?

Despite the lack of a coherent cognitive stress-vulnerability theory of bipolar disorder, encouraging anecdotal and single case reports on the use of cognitive therapy in clients with bipolar disorder have been published over the last twenty years (Chor, Mercier, & Halper, 1980; Wright & Schrodt, 1992; Scott, 1995b). These have been followed by a range of small-scale open studies and case series through to large-scale randomised controlled trials (RCTs). This section will give an overview of these studies and then consider the role of cognitive therapy in individuals with bipolar disorder.

Studies Using Cognitive and Behavioural Techniques

The aim of Cochran's study was to add brief cognitive therapy to standard clinical care in order to enhance adherence with prophylactic lithium treatment. It compared 28 clients who were randomly assigned to either six sessions of group cognitive therapy plus standard clinic care or standard clinic

care alone (Cochran, 1984). Following treatment, enhanced lithium adherence was reported in the intervention group with only three patients (21 %) discontinuing medication as compared with eight patients (57 %) in the standard clinic care group. There were also fewer hospitalisations in the group receiving cognitive therapy. Unfortunately no information was available on the nature of any affective relapses.

Scott and Tacchi (2002) applied the ideas proposed by Cochran (1984) to undertake a pilot study of a brief cognitive therapy intervention (seven sessions of 30 minutes each) for ten individuals with bipolar disorder who were poorly adherent with lithium. They reported significant improvements in how the individual viewed the disorder, improvements in medication adherence and increases in serum lithium levels.

Perry and colleagues (1999) have undertaken the largest study of technique-driven approaches (n = 69), using cognitive and behavioural techniques to help people identify and manage early warning signs of relapse in a group of clients at high risk of further relapse of bipolar disorder who were in regular contact with mental health services. The results demonstrated that, in comparison to the control group, the intervention group had significantly fewer manic relapses (27 % versus 57 %), significantly fewer days in hospital, significantly longer time to first manic relapse, higher levels of social functioning and better work performance. The lack of impact on depression may be a function of the brevity of the intervention and the lack of techniques targeting subsyndromal depressive symptoms (see Scott & Colom, 2005; for more details).

Studies of Group Cognitive Therapy

In Palmer and colleagues (1995) initial exploratory study, six clients with bipolar disorder were offered cognitive therapy in a group format. The focus of the programme was psycho-educational, recognising the process of change, enhancing coping strategies, and dealing with interpersonal problems. Overall findings indicated that group therapy combined with mood-stabilising medications was effective for some but not all of the participants. All participants improved on one or more measure of symptoms or social adjustment, but the pattern of change varied greatly across individuals. A more recent naturalistic study by these investigators (A. Palmer, presented at the annual meeting of the British Association of Behavioural and Cognitive Psychotherapists, Durham, UK, 1998) included a larger number of participants receiving group cognitive therapy (n = 25) and a comparison group (n = 12) receiving only treatment as usual. The results showed that, in comparison to the control group, cognitive therapy reduced non-specific psychological symptoms and increased social adjustment. There were no significant differences in actual relapse rates in those receiving group cognitive therapy or those receiving treatment as usual alone.

Weiss and colleagues (2000) used a group format to deliver therapy to individuals with comorbid bipolar disorder and substance dependence. The therapy was described as “Integrated Group Therapy”, but incorporated a number of cognitive and behavioural elements. Twenty-one individuals receiving group therapy were compared with 24 clients receiving usual treatment and regular assessments. The main outcome measures were severity of addiction and number of months abstinent. The group therapy subjects showed statistically significantly greater improvement on both of these measures at six-month follow-up.

The STEP BD study is soon to be published. The findings will important to the field of group cognitive therapy for bipolar disorder as it is the largest RCT undertaken and compares cognitive therapy to other psychotherapies.

Studies of Individual Cognitive Therapy

Zaretsky, Segal, and Gemar (1999) used a matched case-control design to compare the benefits of 20 sessions of cognitive therapy plus mood stabilizer medication for individuals with bipolar depression (n = 11) with an equivalent course of cognitive therapy for individuals unipolar depression (n = 11). Both groups achieved similar reductions in level of depressive symptoms, but Zaretsky and colleagues reported that only subjects with unipolar depression showed a significant post-therapy reduction in levels of dysfunctional attitudes.

Following a successful small scale (n = 25) pilot study (Lam et al., 2000), Lam and colleagues (2003) undertook a larger scale single centre RCT of over 100 participants with euthymic bipolar disorder. Participants were randomly allocated to cognitive therapy as an adjunct to mood stabilizing medication or to usual treatment alone (mood stabilizers plus outpatient support). The cognitive therapy model particularly utilizes cognitive therapy techniques to cope with the prodromal symptoms of an affective episode and subjects were offered up to 20 sessions of therapy over about six months. Although the approach has some similarities to Perry et al.'s (1999) model, Lam and colleagues also targeted longer-term vulnerabilities and difficulties arising as a consequence of the disorder. Independent assessments demonstrated that, after controlling for gender and illness history, the intervention group had significantly fewer bipolar disorder relapses (cognitive therapy group = 43%; control group = 75 %), psychiatric admissions (15 % versus 33 %) or total days in episode (about 27 days versus 88 days) over 12 months than the control group. The reduction in total number of episodes comprised significant reductions in major depressive (21 % versus 52 %) and manic episodes (17 % versus 31 %) but not mixed episodes. The intervention group also showed significantly greater improvements in social adjustment and better coping strategies for managing prodromal symptoms of mania. Furthermore, the benefits of cognitive therapy extended for about two years after entering the RCT, although most the benefit was in reduction in risk of depressive relapse Lam et al., 2005).

The pilot study by Scott, Garland, and Moorhead (2001) examined the effect of 20 sessions of cognitive therapy in 42 clients with bipolar disorder. To maximise participation in the study and to increase the data available from a pilot study, Scott and colleagues chose a trial design that offered all subjects the opportunity to receive cognitive therapy at some point. Individuals initially allocated to the control group (n = 21) were therefore offered the opportunity to receive cognitive therapy after six months. Subjects could also enter the study during any phase of bipolar disorder. Clients were initially randomly allocated to the intervention group or to a "waiting list" control group. This randomized phase (six months) allowed assessment of the effects of cognitive therapy plus usual treatment as compared with usual treatment alone. At initial assessment, 30 % of participants met criteria for an affective episode, 35 % for substance misuse, and 45 % had a comorbid disorder. The results of demonstrated that, compared with subjects receiving treatment as usual, those who received additional cognitive therapy experienced statistically significant reductions in symptoms, and improvements in global functioning and work and social adjustment. Data from all subjects who received cognitive therapy and were followed up for 12 months post-cognitive therapy (n = 29) demonstrated a 60 % reduction in relapse rates in the 18 months after commencing cognitive therapy as compared with the 18 months prior to receiving cognitive therapy. This study was the fore-runner of the Medical Research Council pragmatic effectiveness trial which recruited over 250 subjects with bipolar disorder from five centres across Britain (Scott et al., 2006a) and randomly assigned subjects to usual treatment or usual treatment plus 22 sessions of cognitive therapy. Patients were assessed every eight weeks for 18 months. Over 50 % of the sample had a recurrence by 18 months with no evidence of significant differences between groups. However, there was a highly significant interaction between randomized

treatment and number of episodes recorded at baseline assessment (Hazard Ratio 0.74; 95 % ci 0.56 to 0.98, $p = 0.04$) such that adjunctive cognitive therapy was significantly more effective than treatment as usual in subjects with fewer than 12 previous episodes, but less effective in those with more episodes. The authors suggest that cognitive therapy may benefit individuals with bipolar disorder who have comparatively fewer previous mood episodes. However, this study recruited a sample that was more heterogeneous than previous RCTs in bipolar disorder patients, including individuals with (a) current substance abuse or dependence, (b) very recurrent bipolar disorder (often reporting 20–30+ previous episodes) who had relapsed in the previous six months, c) other comorbid axis 1 disorders or (d) were in a major bipolar disorder episode at randomization (30 % had significant depressive symptoms). As such, it may be that the most important finding is that cognitive therapy is highly efficacious in preventing relapses in those with fewer comorbidities or additional consequences of bipolar disorder who are euthymic when therapy commences (as in Lam's studies), but less effective in those who have complicated or extensive histories of illness. A secondary analysis of other studies of 22 sessions of evidence-based psychological therapies in bipolar disorder has supported the notion that those with a prior history of multiple episodes do not do any better than those offered usual treatment (Scott et al., 2006b)

Conclusions

The empirical data reviewed indicate that research into cognitive theory and therapy for bipolar disorder is still evolving. There is no robust evidence that underlying schemata play a unique causal role in first onset of mania, nor are there consistent differences in the underlying beliefs of those at risk of bipolar disorder as compared with unipolar disorder (Power, 2005). However, there is evidence that cognitive factors may influence vulnerability to bipolar disorder relapse. The potential interaction between life events and cognitive style is easier to understand for depressive than for manic relapse as the events associated with the onset of bipolar disorder depression have many similarities to those linked with unipolar depression (Mansell et al., 2005). Mania may arise in association with negative life events such as bereavement, but also following events that disrupt an individual's day-to-day social rhythms (Malkoff-Schartz et al., 1999), following long-haul flights, the sudden cessation of mood stabilizing medication, or the onset of significant physical disorder, etc. A number of researchers suggest that a common link between these events is that they all can significantly disrupt circadian rhythms (e.g. Ehlers, Frank, & Kupfer, 1988; Goodwin & Jamison, 1990). In turn, circadian dysrhythmia may lead to sleep disturbance and affective shifts. The work of researchers such as Johnson and Lam on goal attainment life events and hyper-positive sense of self (see Mansell & Scott, 2005; for a review) is an exciting area of development that makes intuitive sense in thinking about the specific factors that may lead to the development of manic episodes or prevent recovery from bipolar disorder episodes.

Even if we have not yet established a robust specific cognitive therapy model of bipolar disorder, it is clear that psychosocial problems may be causes or consequences of bipolar disorder relapses and adding cognitive therapy to usual treatment approaches may improve the prognosis of those at risk of persistent symptoms or frequent episodes. The different types of approaches are technique-driven interventions, group cognitive therapy and individual cognitive therapy. The fundamental difference between the technique-driven interventions and formal cognitive therapy is that the former are briefer than the specific therapies (about 6–9 sessions compared to about 20–22 sessions) and usually do not use individualized formulations, but offer a generic, fixed treatment package targeted at a

circumscribed issue such as medication adherence or managing early symptoms of relapse. These brief interventions can be delivered by a less skilled or experienced professional than the specific models and appear to be potentially very useful in day-to-day clinical practice in general adult psychiatry settings. However, the main benefit is in preventing the cascade of early symptom of mania into a hypomanic or manic episode, with little effect on preventing depression. The reasons for this are not entirely clear, but it is possibly related to the evolution of symptoms into episodes and the nature and speed of action of available treatments (Scott & Colom, 2005). To clarify this, larger scale randomised trials should be encouraged. The results of the multi-centre RCT in the USA with group cognitive therapy as a treatment option is awaited with interest as the group format has the additional advantage of allowing individuals to share their views of bipolar disorder with others and to learn adaptive coping strategies from observing other group members as well as having regular contact with an expert therapist. There is also a potential cost saving from 1–2 therapists simultaneously leading a group that may include 8–12 individuals with bipolar disorder.

Is there a problem about individual cognitive therapy? The results of Lam et al.'s study (2003) and Scott et al.'s study (2006a) are not really that difficult to integrate – together these RCTs demonstrate which populations are likely to do well with adjunctive cognitive therapy and which are not likely to benefit or will struggle to make gains unless offered an extended course of cognitive therapy of more than 22 sessions. Given that in both the large RCTs there were reductions in relapse rates and hospitalisations associated with the use of cognitive therapy as an adjunct to medication in euthymic subjects with bipolar disorder who did not currently have multiple complications, these studies indicate that the *targeted* use of cognitive therapy is likely to prove to be both clinically and cost effective as well as contributing to a significant improvement in the quality of life of a significant proportion of individuals with bipolar disorder (and indirectly to that of their significant others). As such, cognitive therapy and other evidence-based brief therapies represent an important component of good clinical practice in the management of bipolar disorder, the only real argument is about who the individuals are with bipolar disorder who will benefit most from cognitive therapy and when is the best moment in time to introduce therapy (Scott et al., 2006b).

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Key publications

Jones, L., Jones, I., Sham, P., McGuffin, P., Farmer, A., Scott, J., & Craddock, N. (2005). Cognitive and personality style in bipolar disorders. *British Journal of Psychiatry, 187,* 431–437.

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Until recently, bipolar disorders were widely regarded as a biological disorder best treated only with medications. However, there is a significant efficacy-effectiveness gap for pharmacological treatments for bipolar disorder and this, plus the greater acceptance of stress-diathesis models for all severe disorders has led to the development of (i) new etiological theories of bipolar disorders that emphasise psychological and social aspects of vulnerability and risk and (ii) an increased interest in non-pharmacological treatment approaches in bipolar disorder. This paper explores key aspects of cognitive models of bipolar disorder, especially research on dysfunctional attitudes and beliefs; it then comments on the clinical applicability of cognitive therapy for bipolar disorder and reviews the outcome studies available. The final section offers an overview of key areas for further research- it is clear we have problems with current models on the evolution of mania, but there are benefits from the adjunctive use of cognitive therapy in clients with bipolar disorders when they are euthymic and in the earlier stages of their disorder.

Keywords: dysfunctional attitudes, beliefs, bipolar disorders, mood disorders, cognitive theory, cognitive therapy