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Psychological therapy for anxiety in bipolar spectrum disorders: A systematic review

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HIGHLIGHTS

- Anxiety is common in bipolar spectrum disorders and associated with poor outcomes.
- Combined CBT for anxiety and mood may reduce anxiety in bipolar spectrum disorders.
- CBT for comorbid anxiety disorders appears promising in pilot studies.
- Psychoeducation alone does not appear to reduce anxiety.
- Developing specific treatment protocols for bipolar anxiety may improve outcomes.

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ABSTRACT

Comorbid anxiety is common in bipolar spectrum disorders [BPSD], and is associated with poor outcomes. Its clinical relevance is highlighted by the "anxious distress specifier" in the revised criteria for Bipolar Disorders in the Diagnostic and Statistical Manual 5th Edition [DSM-5]. This article reviews evidence for the effectiveness of psychological therapy for anxiety in adults with BPSD (bipolar I, II, not otherwise specified, cyclothymia, and rapid cycling disorders). A systematic search yielded 22 treatment studies that included an anxiety-related outcome measure. Cognitive behavioural therapy [CBT] for BPSD incorporating an anxiety component reduces anxiety symptoms in cyclothymia, "refractory" and rapid cycling BPSD, whereas standard bipolar treatments have only a modest effect on anxiety. Preliminary evidence is promising for CBT for post-traumatic stress disorder and generalised anxiety disorder in BPSD. Psychoeducation alone does not appear to reduce anxiety, and data for mindfulness-based cognitive therapy [MBCT] appear equivocal. CBT during euthymic phases has the greatest weight of evidence. Where reported, psychological therapy appears acceptable and safe, but more systematic collection and reporting of safety and acceptability information is needed. Development of psychological models and treatment protocols for anxiety in BPSD may help improve outcomes.

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1. Bipolar spectrum disorders prevalence and characteristics

Bipolar disorder [BP] is defined as episodes of extreme moods; mania (or hypomania in BP-II) and depression (American Psychiatric Association [APA], 2013a). It has a lifetime prevalence of around 1% (Merikangas et al., 2011). Bipolar spectrum disorders [BPSD] additionally include BP Not Otherwise Specified [NOS], which does not clearly fall within the BP-I or II criteria, and cyclothymia (recurrent episodes of hypomania and minor depressive episodes, thought to be a symptomatically mild, yet chronic, type of BPSD), and all forms can present as rapid cycling BP (four or more acute episodes per year), a further challenge to treatment and prognosis. Medical management is complex and around 60% of patients relapse within 2 years of remission from a major depressive or manic episode (Geddes & Miklowitz, 2013). Poor outcomes are associated with early age of onset, delay in diagnosis and treatment, presence of residual symptoms, and comorbidities including subsyndromal anxiety (Perlis et al., 2006; Treuer & Tohen, 2010). A more detailed consideration of anxiety and its treatment within BPSD is clearly warranted.

Psychosocial stressors are implicated in the onset of BPSD episodes (Ghaemi, Boiman, & Goodwin, 1999) and psychosocial interventions are increasingly emphasised in clinical guidance (Goodwin, 2003; Hirschfeld et al., 2004; National Institute for Health & Clinical Excellence [NICE], 2014). The need for close integration between psychological/psychosocial and pharmacological approaches, with psychological intervention adjunctive to long-term maintenance pharmacotherapy, is now well recognised (Goodwin & Consensus Group of the British Association for Psychopharmacology, 2009). Although psychoeducation is beneficial and possibly more cost-effective than structured psychological treatment in terms of relapse prevention (Geddes & Miklowitz, 2013), there is also evidence that all psychological therapies outperform psychoeducation when global functioning is measured alongside clinical outcome (Miklowitz et al., 2007).

1.1. Treatment of BPSD

Cognitive behaviour therapy [CBT] has been developed for BPSD based on protocols for major depression, but results from major clinical trials remain inconclusive (Lam, Hayward, Watkins, Wright, & Sham, 2005; Parikh et al., 2012; Scott et al., 2006). Overall, the weight of evidence finds little or no benefit of existing CBT protocols for relapse prevention (Lynch, Laws, & McKenna, 2010; Szentagotai & David, 2010). Consequently there has been increasing interest in innovating and improving CBT for BPSD, driven by development of novel cognitive theories (e.g. Holmes, Geddes, Colom, & Goodwin, 2008; Johnson, 2005; Jones, 2001; Mansell, Morrison, Reid, Lowens, & Tai, 2007), qualitative research (Mansell, Powell, Pedley, Thomas, & Jones, 2010), and single case series (Mansell, 2007; Searson, Mansell, Lowens, & Tai, 2012).

There are several possibilities for improving therapeutic strategies, for example, achieving a better characterisation of the BPSD phenotypes and maintaining mechanisms, and measuring treatment effectiveness not solely by acute relapse rate, but by including maintenance of inter-episodic daily mood stability, patients' global psychosocial functioning, coping strategies and life satisfaction (Geddes & Miklowitz, 2013). Novel approaches have recently recommended "staging" the different phases of BPSD to better address the specific presenting needs and dysfunction. The prodromal stage is characterised by mild unspecific mood symptoms, high rates of nonspecific anxiety, and additional bipolar risk factors. The first acute mood episode stage may also include significant anxiety symptoms, and later stages are characterised by subsyndromal symptoms and syndromal relapses despite treatment, e.g. with more prominent cognitive impairment (Kapczinski et al., 2014; Scott et al., 2013). Psychological interventions such as CBT would be indicated for early stages (Berk et al., 2013) or for addressing later residual symptoms (Cosci & Fava, 2012; Fava, Bartolucci, Rafanelli, & Mangelli, 2001).

1.2. Anxiety in BPSD

Comorbid anxiety can be debilitating in itself and prevalence within BPSD is high. Lifetime comorbidity is estimated between 24% (Henry et al., 2003) and 74.9% (Merikangas et al., 2007). Around one third of BP-I and II patients meet criteria for an anxiety disorder, most commonly social anxiety (22.0% lifetime; 12.7% current), panic disorder with or without agoraphobia (17.3%; 8.0%), obsessive-compulsive disorder [OCD] (9.9%; 5.7%), post-traumatic stress disorder [PTSD] (17.2%; 5.1%), agoraphobia without panic (8.5%; 4.4%), and generalised anxiety disorder [GAD] (18.4%; 2.3%) (Otto et al., 2006; Simon et al., 2004), see McIntyre, Soczynska, et al. (2006) for a detailed review.

Comorbid anxiety has been linked to a range of poor outcomes such as illness severity (Lee & Dunner, 2008), suicide attempts (Goldberg & Fawcett, 2012), lower quality of life (Kauer-Sant Anna et al., 2007), and physical ill-health (Albert, Rosso, Maina, & Bogetto, 2008), for reviews see El-Mallakh and Hollifield (2008) and McIntyre, Konarski, et al. (2006). Anxiety has been described as a "clinically meaningful correlate of poor outcome in the acute treatment of bipolar I disorder" (Feske et al., 2000, p.961). This evidence has led to the introduction of a new diagnostic specifier in the Diagnostic and Statistical Manual 5th Edition [DSM-5] (APA, 2013a), described as an "anxious distress specifier". The aim is to identify patients with BPSD with anxiety symptoms that are additional to the BPSD criteria (APA, 2013b) and that may be effectively targeted with specific interventions. For example, recent data suggests that the presence of anxiety could indicate good response to adjunctive psychological treatment for acute BPSD depression (Deckersbach et al., 2013). Moreover, prodromes and early stages of BPSD appear to be particularly characterised by high levels of anxiety, which may represent an important target for successful early intervention strategies (Duffy et al., 2013).

Unlike BPSD, there is clear evidence that (non-comorbid) anxiety disorders can be treated effectively with CBT. For example, the UK National Institute of Health and Clinical Excellence [NICE], a government-funded, independent body that considers the cost-effectiveness of treatments and makes recommendations for health services, recommends CBT as a first-line intervention for GAD and panic with/without agoraphobia (NICE, 2011), PTSD (NICE, 2005b), OCD (NICE, 2005a), and social anxiety disorder (NICE, 2013). A review of meta-analyses of RCTs found medium to large effect sizes for CBT for several anxiety disorders (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012), and meta-analyses of clinically-representative effectiveness studies suggest the results generalise to clinical settings (Hans & Hiller, 2013; Stewart & Chambless, 2009).

For people with BPSD and comorbid anxiety, current guidance recommends "treatment in line with the relevant NICE clinical guideline, in addition to treatment for bipolar disorder" (NICE, 2014, p.15). However, there are no established protocols for treating anxiety in BPSD, and people with BPSD are routinely excluded from anxiety treatment studies (Hoertel, Le Strat, Blanco, Lavaud, & Dubertret, 2012). The need for a well-established evidence-based psychological treatment for anxiety in BPSD is therefore urgent, particularly given the current heated debate around the use of anti-anxiety pharmacological interventions in BPSD (Goodwin & Consensus Group of the British Association for Psychopharmacology, 2009; Vázquez, Baldessarini, & Tondo, 2014). Pharmacological options for anxiety disorders include benzodiazepines, whose efficacy has recently been revisited (Balon, 2013; Offidani, Guidi, Tomba, & Fava, 2013; Rickels, 2013) although their potential for abuse and dependence remains highly controversial. The other most common option is selective serotonin reuptake inhibitor [SSRI] antidepressants, which can be difficult to manage and may worsen the course of BPSD (Pacchiarotti et al., 2013). For example, studies have reported the risk of a "treatment emergent affective switch" in BPSD patients treated with SSRIs (Post et al., 2006; Tondo, Vázquez, & Baldessarini, 2010). Development of excessive arousal or worsening of impulsivity and irritability symptoms by SSRI treatment could be particularly problematic in youth BPSD populations with anxiety (Offidani, Fava, Tomba, & Baldessarini, 2013; Strawn et al., 2013). Mood stabilisers include lithium, anticonvulsants and antipsychotics and are the recommended medication for acute and long term treatment of BPSD (NICE, 2014). They can reduce mood oscillations of both polarities and initial evidence suggests some beneficial effects on anxiety, for example the antipsychotic quetiapine used for BPSD depression. However, further systematic assessment is needed to develop clear clinical guidelines (Vázquez et al., 2014).

Thus, it is unclear how clinicians should best proceed in tackling the debilitating, and commonly untreated, comorbidity of BPSD and anxiety.

1.3. Objectives and importance of the current review

This review aims to identify, synthesise, and critically evaluate research on the psychological treatment of anxiety within BPSD. The recent inclusion of the anxious distress specifier in DSM-5 highlights increased recognition of the problem posed by anxiety in BPSD. Similarly, a study protocol has recently been published testing a specific intervention developed for anxiety in BPSD (Jones et al., 2013) suggesting that such a review is timely. While there are previous reviews in this area (Provencher, Hawke, & Thienot, 2011; Rakofsky & Dunlop, 2011), they have included only a small number of studies (11 in total) and given predominantly a narrative account. The current review provides an advance by identifying and including a further 11 studies, and using an established systematic review methodology with a critical evaluation of research methods and outcomes. The review is guided by the Cochrane Collaboration's (2011) handbook and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses [PRISMA] checklist (Moher, Liberati, Tetzlaff, Altman, & The PRISMA Group, 2009). The current review aims to ascertain, in BPSD populations, how effectively comorbid anxiety disorders and/or anxiety symptoms are reduced by psychological therapy:

- designed specifically to target BPSD anxious distress,
- targeting both anxiety and BPSD mood symptoms using a combined approach,
- targeting comorbid anxiety disorders only in BPSD populations, or
- targeting BPSD mood symptoms only.

2. Method

Titles and abstracts in AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, and HEALTH BUSINESS ELITE databases were systematically searched on 16/05/2013, using terms related to BPSD, anxiety, and psychological therapy (see Appendix A). This was augmented by a manual search using reference lists of related papers, contacting researchers, and Google Scholar internet searches. Due to the heterogeneity of the studies a formal synthesis (i.e. meta-analysis) was not possible. Instead, a systematic review was conducted, using the principles cited above.

2.1. Inclusion criteria

Studies were included if (1) participants included people with BPSD, (2) pre–post outcome data related to anxiety (e.g. symptom measures or diagnostic interviews) were reported, (3) treatment targeted anxiety and/or BPSD, and (4) the paper was published in a peer-reviewed journal.

2.2. Exclusion criteria

No limitations were placed on year or language of publication (although searches were in English). Theoretical papers, those including children and/or adolescents, or with a distinctly different treatment focus (e.g. addiction, eating disorders), were excluded.

3. Results

Fig. 1 provides a flow-chart for the selection of eligible studies. Ten eligible studies were identified from previous reviews (Baer, Minichiello, & Jenike, 1985; Dusser, Romo, & Leboyer, 2009; Hamblen, Jankowski, Rosenberg, & Mueser, 2004; Miklowitz et al., 2009; Mueser et al., 2007, 2008; Rosenberg, Mueser, Jankowski, Salyers, & Acker, 2004; Van Gent, 2000; Van Gent & Zwart, 1993b; Williams et al., 2008). One of these papers (Van Gent, 2000) provides follow-up data only, leaving nine studies.

Seven further studies were identified through a systematic literature search using the criteria specified above (Da Costa et al., 2011; Docteur, Mirabel-Sarron, Guelfi, Rouillon, & Gorwood, 2013; González-Isasi et al., 2010; Perich, Manicavasagar, Mitchell, Ball, & Hadzi-Pavlovic, 2013; Proudfoot et al., 2012; Satterfield, 1999; Totterdell, Kellett, & Mansell, 2012).

Manual searching identified eight further eligible papers (Docteur et al., 2007; Fava, Rafanelli, Tomba, Guidi, & Grandi, 2011; González Isasi, Echeburúa, Limiñana, & González-Pinto, 2012; Lu et al., 2009; Reilly-Harrington et al., 2007; Thienot, Provencher, & St-Amand, 2013; Van Gent, Vida, & Zwart, 1988; Van Gent & Zwart, 1993a). Two of these papers (González Isasi, Echeburúa, Limiñana & González-Pinto, 2012; Van Gent & Zwart, 1993a) provide follow-up data only, leaving six studies.

The 22 included studies are summarised in Table 1.

Table 1 includes details of sample age, location; anxiety type and BPSD type; participants' mood at the time of intake and any exclusion criteria; the psychological treatment; and the study design. Three types of treatment approach are delineated: treatments that target both anxiety and BPSD symptoms; treatments that target specific anxiety disorders in a BPSD population; and treatments that primarily target BPSD symptoms and additionally report anxiety outcomes. No treatment studies were found specifically designed to target BPSD anxious distress (or a comparable construct).

3.1. Summary of study characteristics

3.1.1. Participants

All studies recruited participants between episodes except Proudfoot et al. (2012), which recruited newly diagnosed patients of whom 48% were euthymic at intake according to self-report. The studies varied in how symptom remission was defined, and most included people experiencing some mood symptoms. This reflects the growing acknowledgement that the course of BPSD is rarely one of discrete episodes with full symptom remission between, but that patients frequently experience significant mood symptoms or mood instability inter-episode. Few studies report ethnicity, though where it is reported the large majority of participants are Caucasian, except Lu et al. (2009),

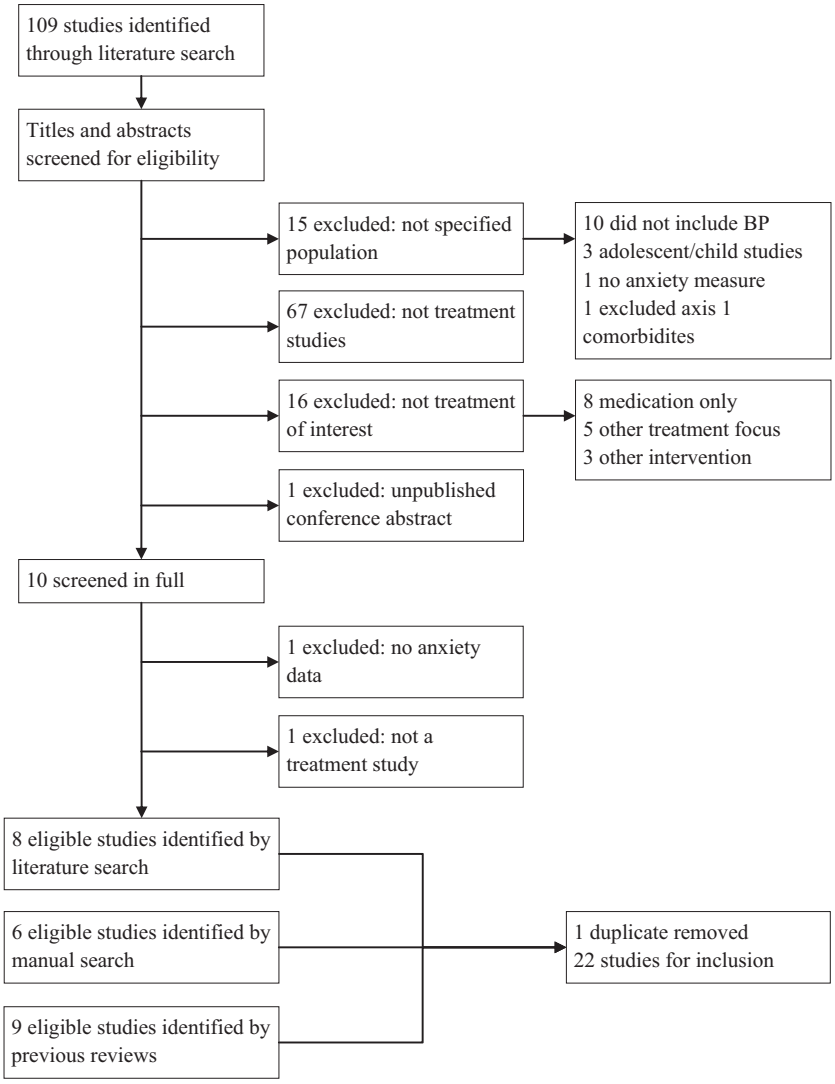

Fig. 1. Flow-chart for the selection of eligible studies.

where 42% were European-American, 37% African-American, 11% Hispanic, and 11% Other. Results may not be generalizable to younger (under 18 years) and older (over 65 years) adults given that child and adolescent studies were excluded and no older adult studies were found. Notably, all the studies suggest complexity in the participants' presentations; for example, comorbidity, multiple episodes, persistent symptoms, risk/suicide history, and social/functional impairment. This suggests good ecological validity in relation to clinical populations.

3.1.2. Interventions

The majority of studies (14/22) used CBT with between six and 21 sessions, of which eight were individual- and six group-based interventions (Table 1). Three studies employed Mindfulness Based Cognitive Therapy [MBCT] in groups, two psychoeducation in groups, one psychoeducation via an online programme, and one Exposure and Response Prevention [ERP] on an individual basis. Fava et al. (2011) sequentially applied CBT and wellbeing therapy [WBT]. All used an established (if adapted) manual except Dusser et al. (2009) and Satterfield (1999). Two studies included family members (Baer et al., 1985; Da Costa et al., 2011). All studies but one expected patients to continue with mood-stabilising medication, and the majority of participants were taking medication including lithium, neuroleptics, anticonvulsants, antidepressants, and/or anxiolytics. Some studies did not systematically collect/report information on medication use (Lu et al., 2009; Mueser et al., 2008; Rosenberg et al., 2004; Thienot et al., 2013). Changes to medication were reported during the study for 18/95 participants in Perich et al. (2013), and 2/6 in Reilly-Harrington et al. (2007). In the study by Fava et al. (2011), patients using mood stabilisers or antidepressants were excluded, and participants were titrated off benzodiazepines, constituting a distinct population compared to the other studies.

3.1.3. Outcomes

Most studies assessed self- or clinician-reported anxiety symptoms using continuous measures, e.g. Beck Anxiety Inventory [BAI] (Beck & Steer, 1993), State-Trait Anxiety Inventory [STAI] (Spielberger, Gorsuch, & Lushene, 1970). Seven focussed on a single anxiety diagnosis; e.g. GAD (Thienot et al., 2013), and a further two on mixed anxiety disorders (Fava et al., 2011; Reilly-Harrington et al., 2007).

3.1.4. Study design

Seven of the 22 studies comprise randomised controlled trials [RCTs]. Three RCTs report using intent-to-treat analysis [ITT] (Fava et al., 2011; Mueser et al., 2008; Perich et al., 2013). Two of the RCTs have longer term outcomes; two years (Fava et al., 2011), and five years (González Isasi et al., 2012). Van Gent and Zwart (1993a) and Van Gent (2000) have five year follow-ups, but do not collect anxiety data.

Ten are pilot studies, and may be precursors to larger feasibility studies and RCTs. Three include a comparison group but no randomization or blinding, and seven have no comparison group. Pilot studies are thought to have a risk of selection bias, information bias, and systematic confounds due to non-randomisation, but may also confer better external validity than RCTs due to the type of population included (Grimes & Schulz, 2002). Those without blinding and with only clinician-rated measures are especially at risk of bias (e.g. Docteur et al., 2013). Most of the pilot studies report only data from those participants who completed treatment, (or failed to specify) except Mueser et al. (2007), which used ITT, and Miklowitz et al. (2009), which imputed missing data with the sample's mean score.

Five studies used single-case designs. Three benefit from experimental AB designs, either establishing a stable baseline (Thienot et al., 2013) or – given that instability is the key feature of BP – a multiple baseline design that characterises the typical instability (Satterfield, 1999; Totterdell et al., 2012). Two are narrative case reports (Baer et al., 1985; Hamblen et al., 2004), which increases the risk of bias. The small sample sizes in all these studies limit generalisability.

Table 2 provides an overview of the key findings, summarises the results, and details drop-out rates for each study. Studies are now reviewed in turn, according to the three categories of treatment outlined above.

3.2. Treatments that include components targeting both anxiety and BPSD ($N = 7$)

3.2.1. Randomised controlled trials

Three RCTs were identified, all of which included blind assessment and follow-ups, of five years (González-Isasi, et al., 2010; González Isasi, et al., 2012), two years (Fava et al., 2011), and one year (Perich et al., 2013). Two (Fava et al., 2011; Perich et al., 2013) used concealed randomisation, ITT, and an active control (clinical management with equal contact time; TAU plus written psychoeducation resources, respectively).

González-Isasi, et al. (2010) and González Isasi, et al. (2012) $N = 42$ RCT evaluated CBT for BPSD based on the protocol by Lam et al. (2010) with the addition of "relaxation and breathing, self-instruction and cognitive distraction" to target anxiety symptoms. Participants had "refractory" BP-I and II; persistent, treatment-resistant affective symptoms, although "patients with poor medication adherence, according to the doctor or relatives' report, were excluded" (González Isasi et al., 2012, p.135). Retention was 100%, and the control group remained free from therapy for the entire five years, while the "psychiatrist provided support when necessary" (p.82). Within-groups, there was significant improvement in the treatment group, and deterioration in the TAU group, in state anxiety. Between-groups, there were significant differences in state anxiety at follow-up (6 months, 12 months, and 5 years). This study suggests that CBT for BPSD with an additional anxiety management component can reduce anxiety and improve other outcomes in the long term.

Fava et al.'s (2011) $N = 62$ RCT sequentially applied CBT to target phobic anxiety (Marks, 1987) and depression (Basco & Rush, 1996), and then WBT for hypomania. WBT aims to increase autonomy, personal growth, environmental mastery, purpose in life, positive relationships with others and self-acceptance (based on Ryff, 1989), thereby substituting hypomania with sustained well-being. It does not include psychoeducation, medication adherence or social rhythm therapy (Fava, 1999; Fava & Tomba, 2009). Participants had cyclothymia and, notably, were not taking mood stabilising/antidepressant medication and were titrated off benzodiazepines during treatment. The treatment group had significantly higher levels of affective symptoms than the control group at intake but were otherwise matched. Comorbidities were various anxiety disorders, hypochondriasis (health anxiety) and body dysmorphic disorder. There was a significant reduction in comorbid diagnosis rates, maintained at two years, and a significant group-by-time interaction, suggesting that CBT/WBT effectively reduced comorbid anxiety disorders compared to the control condition.

Perich et al.'s (2013) $N = 95$ RCT adapted the eight week MBCT course (Segal, Williams, & Teasdale, 2002) for BP-I and II to include awareness of mania and anxiety prodromes in addition to depression and suicidal thoughts. Attrition was high, with only 34 participants (36%) completing the final follow-up (22 MBCT, 12 TAU). A significant condition-by-time interaction suggested that MBCT reduced state anxiety compared to the control condition. Similar trend-level improvements were found for trait anxiety and stress. Comorbid anxiety diagnosis rates fell in both groups, but with no significant between-group difference at one year. MBCT modestly reduced state anxiety and stress, but did not appear to reduce diagnosis rates (nor affective symptoms) compared to TAU. The high rate of attrition means that these results must be interpreted with caution.

Table 1
Study characteristics.

	N, Mean age Location	Anxiety type BPSD type	Mood at intake/exclusions	Intervention	Study design
Treatments that include components targeting both anxiety and BPSD (N = 7)					
Dusser et al. (2009)	*N* = 10 18+ mean age 47 France.	Continuous anxiety symptoms ("stress"). BP-I & II between episodes.	Patients with current melancholic depression or (hypo)mania were excluded (not operationalised). Mean baseline scores: BDI-13 5.4 (mild), MADRS 5, MRS 1.87	Group CBT for BPSD (novel approach). 6× 2 h weekly sessions.	Pilot. No comparison group. 6 month f'up.
Fava et al. (2011)	*N* = 62 18–65 Italy.	Mixed anxiety diagnoses (In order of prevalence in CBT/WBT vs. CM: GAD 3 vs. 5; panic & agoraphobia 5 vs. 2; OCD 4 vs. 2; social anxiety 4 vs. 2; agoraphobia 1 vs. 3; panic disorder 4 vs. 0; BDD 2 vs. 1; health anxiety 2 vs. 1; and simple phobias 2 vs. 1.) Cyclothymia. Excluded medication within last 2 years..	Cyclothymia diagnosis, i.e. no history of major depression or mania (SADS). No min/max mood scores, mean modified CID score at intake CBT/WBT 42.32, CM 36.87; MAS 9.26, 9.19.	Group/Indiv (not known) CBT then WBT (Fava, 1999; Fava & Tomba, 2009), included anxiety module (novel approach). 10× 45 min fortnightly sessions.	RCT vs. "Clinical management" (active control). Concealed randomisation. 2 year f'up. Blind raters.
González-Isasi, et al. (2010) and González Isasi, et al. (2012)	*N* = 42 18–65 Spain.	Continuous anxiety symptoms. "Refractory" BP-I & II (≥2 relapses within 1 yr, suicide attempts, social-occupational difficulties, or persistent affective symptoms defined by more than 3 months BDI>7 and/or YMRS>6) between episodes.	Patients euthymic or with subsyndromal symptoms at intake (BDI > 7; YMRS > 6), max score not specified. Mean baseline scores BDI, 11.06 (mild) TAU, 11.05 CBT, YMRS 2.06 TAU, 2.50 CBT.	Group CBT for BP (Lam et al., 1999) plus additional anxiety module. 20× 1.5 h weekly groups of 10.	RCT vs. TAU. Blind assessment. 5 year f'up.
Miklowitz et al. (2009)	*N* = 22 Mean age 41 US & UK.	Continuous anxiety symptoms. BP-I & II between episodes.	BP-I or II "currently in remission" using MINI. A full-blown episode within last 2 years but onset not within 3 months of trial start. Subsyndromal symptoms not excluded. Min/max scores not specified.	Group MBCT (Segal et al., 2002) included anxiety/mania prodromes. 8× 2 h weekly groups of 7–15.	Pilot. No comparison group, non-blinded. No follow-up
Perich et al. (2013)	*N* = 95 18+ (mean age not reported) Australia.	Continuous anxiety symptoms. BP-I & II between episodes.	Current DSM-IV major depressive, hypomanic or manic episode (using SCID) were excluded. Mean intake scores: MADRS 11.71 MBCT, 14.97 TAU; YMRS 4.98 MBCT, 5.47 TAU; DASS depression 14.79 MBCT, 19.5 TAU.	Group MBCT (Segal et al., 2002) included anxiety/mania prodromes. 8× 2–2.5 h weekly groups of 4–8.	RCT vs. TAU (+ written psychoed'n). Concealed, computerised, randomisation. Blind raters. Fidelity checks. 12 month f'up.
Reilly-Harrington et al. (2007)	*N* = 10 Mean age 38 US.	Mixed anxiety diagnoses, continuous anxiety symptoms. Rapid-cycling BP-I, between episodes.	Current manic or mixed episode were excluded (using SCID). Mean intake scores: MADRS 22.67, YMRS 13.5.	CBT for BPSD (Newman, Leahy, Beck, Reilly-Harrington, & Gyulal, 2001) with anxiety module (e.g. Barlow & Craske, 2006). 20x 50 min weekly sessions, booster at 4 weeks.	Pilot. No comparison group. 8 week f'up.
Satterfield (1999)	*N* = 1 33 US.	Continuous anxiety symptoms. Rapid cycling BP-I.	No raw data for BDI or YMRS presented. Scores at intake indicated moderate depression (using BDI cutoffs).	CBT for rapid cycling BPSD (novel approach). Weekly ➔ monthly sessions for 12 months.	AB single case design. No multiple baseline assessment.
Treatments that target specific anxiety disorders in a BPSD population (N = 7)					
Baer et al. (1985)	*N* = 2 37 & 45 US.	OCD. Mania only and BP-I Between episodes.	Not specified	ERP for OCD. 30–51 sessions biweekly, on-going at publication.	Case study No comparison group or baseline. 15 month f'up.
Hamblen et al. (2004)	*N* = 3 (*n* = 1 BP). Mean age 39 US.	PTSD. BPSD type not specified.	"Severe depression" using BPRS, raw score not reported. Manic symptoms not assessed.	CBT for PTSD. (Mueser et al., 2004). 12 sessions over 10 weeks.	Case study. No comparison, no stable baseline. 3 month f'up
Lu et al. (2009)	*N* = 14 (*n* BPSD not reported). Mean age 42 US "ethnically diverse".	PTSD. BPSD type not specified.	Mean BDI-II score at intake: 31.14. Manic symptoms not assessed. Data not presented separately for BPSD participants.	CBT for PTSD. (Mueser et al., 2004). 12–16× 1 h weekly sessions	Pilot replication. No comparison group. Fidelity checks. 6 month f'up.
Mueser et al. (2007)	*N* = 80 (*n* = 7 BPSD). Mean age 43 US.	PTSD. BPSD type not specified.	Mean BDI score at intake: 31.07 (total sample); 29.97 (treatment completers); 34.74 (dropouts) ns. Manic symptoms not assessed. Data not presented separately for BPSD participants.	Group CBT for PTSD. (Mueser et al., 2004). 21 sessions, groups of 6–8.	Pilot. No comparison group. Fidelity checks. 3 month f'up.
Mueser et al. (2008)	*N* = 108 (*n* = 25 BPSD). Mean age 44 US.	PTSD. BPSD type not specified.	Mean BDI–II score at intake CBT 31.48; TAU 31.76. Manic symptoms not assessed. Data not presented separately for BPSD participants.	CBT for PTSD. (Mueser et al., 2004). 12–16× 1 h weekly sessions.	RCT, vs. TAU. Concealed, computerised randomisation. Blind raters. Fidelity checks. 6 month f'up.
Rosenberg et al. (2004)	*N* = 13 (*n* = 2 BPSD) Mean age 48	PTSD. BPSD type not specified.	Phase of BPSD not specified. Mean score at intake BPRS affect subscale 21.33, depression subscale not reported.	CBT for PTSD. (Mueser et al., 2004).	Pilot. No comparison group.

	US.		Mania symptoms not assessed. Data not provided separately for BPSD participants.	12–16× 1 h weekly sessions.	Non-blinded, uncontrolled. 3 month f'up.
Thienot et al. (2013)	$N = 4$ ($n = 2$ BP-I, 1 BP-II, 1 BPSD NOS) Mean age 49 Canada.	GAD/ high worry. Mixed BPSD type.	Participants in euthymic phase or subsyndromal symptoms at intake (SCID). Mean scores at intake: BDI-II 24 +/− 9.2, YMRS 2.3 +/− 3.2.	CBT for GAD (Dugas, 2004). 12× 1 h weekly sessions.	AB multiple baseline single case series experimental design.
Treatments that target BPSD symptoms only and report anxiety outcomes ($N = 8$)					
Da Costa et al. (2011)	$N = 41$ 18–60 Brazil.	Continuous anxiety symptoms. BP-I & II between episodes.	Currently in episode excluded; "euthymic, mildly depressed or mildly hypomanic" at intake included defined using ≥35 BDI, ≥20 YMRS. Mean BDI baseline score: 19.52 CBT, 11.67 Control ($p = .083$). Mean YMRS score 9.68 CBT, 1.33 Control (ns).	Group CBT for BP (Basco & Rush). 14× 2 h weekly sessions, psychoed with family. Groups of 5–6.	RCT vs. medication only (TAU). 6 month f'up
Docteur et al. (2013)	$N = 73$ ($n = 53$ CBT, 20 TAU) 18–65, mean age 45 France.	Continuous anxiety symptoms. BP-I between episodes.	Patients in acute episode excluded (not operationalised). Mean scores at intake: HAM-D 8.23 (mild), MAS 1.51.	Group CBT for BP (Lam, Jones, Hayward, & Bright, 1999). 20× 2 h weekly sessions.	Controlled study vs. WL (TAU). Non-blinded, non-randomised, clinician-report only. 5–9 month f'up; CBT m = 8.62 months, SD = 2.66 vs. WL m = 5.78 months, SD = 2.63.
Docteur et al. (2007)	$N = 12$ total, but data on $n = 7$ 18–65, mean age 42 France.	Continuous anxiety symptoms. BP-I between episodes.	Patients with significant symptoms mood symptoms excluded, not operationalised. No min/max scores specified. Mean baseline scores: HAM-D 9 (mild), MAS 2 (no mania), BDI-13 14.5.	Group CBT for BP (Lam et al., 1999). 20× 2 h weekly sessions.	Pilot. No comparison group, clinician-report only. No follow-up
Proudfoot et al. (2012)	$N = 407$ 18–75 Australia.	Continuous anxiety symptoms. BPSD type not specified. Recruited within 1 year of diagnosis.	Diagnosed with BPSD (type not specified) by a health professional within the past 12 months. Confirmed by MSQ-27. Mean GADS depression score 6.5. Mania scores not assessed.	Psychoed: Online Bipolar Education Program, with/without email contact with Informed Supporters. 8× 30–40 min weekly modules and workbooks.	RCT vs. active control (weekly emails with links to simple BP-Info). Participants blind to condition. High dropout. 6 month f'up.
Totterdell et al. (2012)	$N = 1$ 35 UK.	Continuous anxiety symptoms. Cyclothymia.	Current phase not specified. Low mood/depression not assessed. Mean scores during baseline using subjective 1–9 scales: "energetic" 4.73, "happy" 3.57.	CBT for BPSD (adapted Basco & Rush, 2005). 19 sessions weekly. 4× daily monitoring.	AB single case multiple baseline experimental design. 11 week f'up.
Van Gent et al., 1988; Van Gent & Zwart, 1993a	$N = 34$[a] Under 60, mean age 40 Netherlands.	Continuous anxiety symptoms. BPSD type not specified	Severe depression and acute psychosis excluded (not operationalised). Phase of BPSD not specified. Mean scores at intake: Depression subscale SCL-90, 31.3 group therapy, 25.4 control; cheerful subscale ML 9.6 group therapy, 8.9 control, dispirited subscale 7.1 group therapy, 7.1 control.	Psychoed "group therapy" (Powell, Othmer, & Sinkhorn, 1977). 10–13× 1.5 h weekly groups of 6–8.	Controlled study vs. TAU. Non-randomised, non-blinded? 15 month f'up (no anxiety data at 5 year f'up).
Van Gent and Zwart (1993b), Van Gent, 2000	$N = 35$–41[a] Mean age 42 Netherlands.	Continuous anxiety symptoms. BPSD type not specified.	Severe depression and acute psychosis excluded (not operationalised). Phase of BPSD not specified. Mean scores at intake: Depression subscale SCL-90, 31.3 group therapy, 28.5 education group; high mood subscale ML, 9.6 group therapy, 10.2 education group, low mood subscale ML, 7.1 group therapy, 2.2 education group.	Psychoed "group therapy" (Van Gent et al., 1988), vs. Ultra-short psychoed group. 10–13× 1.5 h weekly groups of 6–9. vs. 5× 2.5 h weekly groups of 6–8.	Controlled study, two active treatment groups. Non-blinded, non-randomised. 15 month f'up.
Williams et al. (2008)	$N = 68$ ($n = 17$ BP). Data presented for $N = 55$ ($n = 14$ BP) 18–65 UK.	Continuous anxiety symptoms. BPSD type not specified.	History of depression "with serious suicidal ideation or behaviour" currently in recovery. <1 week "minimal depression" in last 8 weeks, no manic episode within 6 m (NIMH definition). Min/max scores not specified. Baseline BPSD scores BDI 12.7 MBCT, 11.4 control. No mania measures.	Group MBCT (Segal et al., 2002). 8× 2 h weekly groups of 12–15, plus full day meditation practice.	RCT vs. WL (TAU). Randomisation concealed via envelopes. Small subgroup analysis. No follow-up.

Note. BDD = body dysmorphic disorder, BP-I = bipolar I disorder, BPSD = bipolar spectrum disorders, CBT = cognitive behaviour therapy, psychoed = psychoeducation, ERP = exposure and response prevention, F'up = follow-up, GAD = generalised anxiety disorder, MBCT = mindfulness-based cognitive therapy, ns = non significant, NOS = not otherwise specified, OCD = obsessive compulsive disorder, PTSD = post-traumatic stress disorder, RCT = randomised control trial, TAU = treatment as usual, WBT = wellbeing therapy.

Measures: BDI = Beck Depression Inventory (Beck, Ward, & Mendelson, 1961); BDI-13 = Beck Depression Inventory short version (Beck & Beamesderfer, 1974); BPRS = Brief Psychiatric Rating Scale (Lukoff, Nuechterlein, & Ventura, 1986); CID = Clinical Interview for Depression (Guidi, Fava, Bech, & Paykel, 2010); GADS = Goldberg Anxiety and Depression Scale (Goldberg, Bridges, Duncan-Jones, & Grayson, 1988), HAM-D = Hamilton Depression Rating Scale (Hamilton, 1960); MADRS = The Mongomery Åsberg Depression Scale (Montgomery & Åsberg, 1979); MAS = Mania Scale (Bech, Kastrup, & Rafaelsen, 1986); MINI = Mini International Neuropsychiatric Interview (Sheehan et al., 1998); ML = Mood List (Zwart & Spooren, 1983), MRS = Mania Rating Scale (Bech, Rafaelsen, Kramp, & Bolwig, 1978); MSQ-27 = Mood Swings Questionnaire (Parker, Hadzi-Pavlovic, & Tully, 2006); NIMH = National Institute of Mental Health; SCL = Symptom Checklist (Arrindell & Ettema, 1986; Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974), SCID = Structured Clinical Interview for DSM (First, Spitzer, Gibbon, & Williams, 1996), YMRS = Young Mania Rating Scale (Young, Biggs, Ziegler, & Meyer, 1978).

[a] Van Gent et al. (1988) compare $n = 20$ patients in three therapy groups lasting 10 sessions to $n = 16$ WL controls. Van Gent and Zwart (1993a) report an additional 5 year follow-up to the previous study, and present within-group analyses for $n = 26$ (no control condition). This paper described four groups sessions lasting 10–13 sessions. Van Gent and Zwart (1993b) compare $n = 20$ described in Van Gent et al. (1988) to $n = 15$ from a more recent ultra-short condition. Van Gent (2000) compares $n = 26$ to $n = 15$ from the ultra-short condition. It appears as if much of the data is common to the papers, but it is not clearly explained which data is included/excluded in which papers and why.

Table 2
Summary of study findings.

	Results	Scores	Dropouts
Treatments that include components targeting both anxiety and BPSD (N = 7)			
Dusser et al. (2009)	↑STAI-S, ↓STAI-T, ↓HAM-A**, ↓PSS14 stress*	WG: self-reported stress [PSS14] pre 36; post 32, $p < .05$. HAM-A pre 12; post 4, $p < .01$. STAI-S pre 27; post 29, ns. STAI-T pre 43; post 41, ns	2 dropped out
Fava et al. (2011)	↓SADS diagnosis*	BG: *n* with Axis 1 comorbidities Pre 23 CBT/WBT vs. 17 CM, $\chi^2 = 2.536$, ns; post: 8 vs. 16, $\chi^2 = 4.351$; 1 yr 8 vs. 16, $\chi^2 = 4.351$; 2 yr 6 vs. 15, $\chi^2 = 5.833$, all $p < 0.05$.	CBT/WBT 5; CM 6.
González-Isasi, et al. (2010) and González Isasi, et al. (2012)	↓STAI-S***	BG: STAI-S pre: 21.30 CBT vs. 16.80 TAU; post 16.0 vs. 22.35, ns trend $p = 0.062$; 6 m 16.50 vs. 26.35, $p = 0.019$; 1 yr 8.85 vs. 32.78, $p < 0.001$; 5 yr 8.80 vs. 28.55, $p < 0.001$.	2 died in control group. No other dropouts
Miklowitz et al. (2009)	↓BAI	WG: BAI pre, 15.38; post 12.80 post, Cohen's $d = .23$ small effect size. No tests of significance are reported due to the low numbers.	6/22 didn't complete treatment. Complete data for 14/16 treatment completers and 3/6 dropouts.
Perich et al. (2013)	↓STAI-S*, ↓STAI-T∞, ↓DASS stress∞, =DASS anxiety, CIDI diagnosis: ↓WG, =BG	BG: STAI-S pre, 47 MBCT vs. 49 TAU; post, 41 vs. 44; 12 m, 39 vs. 43; F = 2.158, $p = 0.048$. Diagnosis: pre 69% MBCT vs. 77% TAU; 12 m 46% vs. 33% $\chi^2 = .083$, $p = .773$	Total dropout 14/48 (29%) MBCT. Defined as missing more than half the sessions. MBCT: 10/48 (21%) dropped out. TAU: 18/47 (38%) dropped out. A further 4 (8%) in each group did not complete f'up. Anxiety outcome data available for 22 MBCT and 12 TAU.
Reilly-Harrington et al. (2007)	↓BAI (ns)	WG: BAI pre 24.83, moderate; mid 17.33, moderate; post 15.33, mild; 2 m 17.83, moderate. Cohen's $d = .55$.	Total 4/10 (40%). 3/10 dropped out after 4 or fewer sessions (of 20 total), 1/10 after 12 and did not complete f'up.
Satterfield (1999)	↓BAI	WG: BAI pre-CBT 10, mild; 0 for the last four months of therapy, (no multiple/stable baseline using BAI, raw data not presented)	None.
Treatments that target specific anxiety disorders in a BPSD population (N = 7)			
Baer et al. (1985)	Reported ↓obsessions, ↓compulsions	No raw scores reported.	None
Hamblen et al. (2004)	↓CAPS, ↓BPRS	Total CAPS score, pre 57; post 8; 3 m f'up 9.	None
Lu et al. (2009)	↓PCL***, ↓PDS**, ↓BPRS***	WG: PCL diagnosis rates, pre 100%; post 69%; 3 m 33%; 6 m, 58%. PDS diagnosis rates, pre 100%; post 89%, χ^2 ns; 75%, $p = .01$; 67%, $p = .007$. Total PDS score pre, 35; post, 24, $p = .002$; 3 m, 19, $p < .001$; 6 m, 17, $p < 001$. Total PCL score pre, 59; post, 51, $p = .02$; 3 m, 44, $p = .004$; 6 m, 48, $p = .01$.	Defined as missing more than half the sessions. 5/19 (26%) dropped out. 2 (11%) further lost to f'up; one due to incarceration, and another due to service discontinuation
Mueser et al. (2007)	↓PCL diagnosis*** ↓PCL total***	WG: mean PCL score, pre 64; post 52; 3 m 52; last follow-up 53, all $p < .001$. Diagnosis rates pre 100%; post 76%; 3 m 77%; 6 m 80%, all $p < .05$	Defined as missing more than half the sessions.40/80 (50%) dropped out of which 12 attended no sessions. Outcome data available for 18–31 treatment completers, and 0–9 dropouts.
Mueser et al. (2008)	↓CAPS diagnosis, ↓CAPS total**, ↓BAI**	BG: diagnosis rates pre 100% CBT vs. 100% TAU; post 67.7% vs. 77.8%; 3 m 63.3% vs. 77.1%; 6 m 72.7% vs. 85.0% ns. Participants with CAPS > 65 at intake ($N = 78$) post 70.8% CBT vs. 90.0% TAU; 3 m 68.2% vs. 92.3%; 6 m 78.3% vs. 88.2%, $p = .02$, large effect size. Total CAPS score pre 74 CBT vs. 76 TAU; post 56 vs. 68; 3 m 55 vs. 65; 6 m 57 vs. 71, $p = .005$ large effect size. BAI pre 48, CBT vs. 50, TAU; post 43 vs. 46; 3 m 41 vs. 48; 6 m 44 vs. 48, $p = .03$, large effect size	Defined as missing more than half the sessions. CBT: 10/54 (19%) dropped out of which 2 attended no sessions after randomisation. A further 11 (20%) in each group lost to f'up. Outcome data available for 20–35/54 TAU and 30–33/54 CBT.
Rosenberg et al. (2004)	↓CAPS total***, ↓CAPS diagnosis, ↓BPRS*	WG: Total CAPS score; pre, 71; post 53; 3 m 48; $t(11) = 4.87$, $p < .001$. Diagnosis rates pre 100%; post 64%; 3 m 50%.	Total dropout 3/22 (14%). Defined as missing more than half the sessions. A further 6/22 (27%) were lost to f'up. Full data available for 12 participants.
Thienot et al. (2013)	↓PSWQ, ↓QIA, ↓BAI	WG: SCID diagnosis rates pre 100%; post 25%. BAI scores baseline 25, moderate; pre 20; post 10; 4 m 7 non-clinical. Somatic symptoms QIA: baseline 31; pre 26; post 23; 4 m 21. Cognitive symptoms PSQW: baseline 60; pre 55; post 49; 4 m 54.	None
Treatments that target BPSD symptoms only and report anxiety outcomes (N = 8)			
Da Costa et al. (2011)	↓BAI***	BG: baseline 18.6 CBT, moderate, vs. 8.0 TAU, non-clinical, $p = 0.096$. WG: CBT pre 19, moderate; post 7, non-clinical, $p < 0.0005$. Control pre 8, non-clinical; post 6, non-clinical, $p = .234$.	4 dropped out (2 from each group); 3 due to hospitalisation, 1 preferred individual therapy. Data for 4 dropouts not complete.
Docteur et al. (2013)	↓HAM-A*	WG: CBT mean HAM-A pre 9.6; post 8.21 Cohen's $d = .38$, $t = 2.36$, df = 40, $p = .012$ (whether pre-post or pre-f'up not specified) TAU $p > .25$, U value not stated. Raw scores not provided for TAU.	11 lost to f'up, all CBT group.

Table 2 *(continued)*

	Results	Scores	Dropouts
Treatments that target BPSD symptoms only and report anxiety outcomes (N = 8)			
Docteur et al. (2007)	=HAM-A	WG: pre 8.62; post 9.00 ns	2 did not attend the group, 3 dropped out early. 5 dropouts were not reassessed. $n = 7$ analysed.
Proudfoot et al. (2012)	GADS: ↓WG, =BG	WG: significant improvement in GADS across the whole sample ($p < .01$), but no significant between-group differences or interactions. Raw scores were not presented. Peer support increased adherence, but this did not affect outcome.	Of $N = 419$ randomised, 118 (28%) dropped out/withdrew and a further 132–142 (32–34%) lost to f'up. BEP 45/141(32%) dropped out/withdrew, and a further 48–52 (34-37%) lost to f'up. BEP + IS 32/139 (23%) dropped out/withdrew, 42–44 (30–32%) lost to f'up. Control 41/139 (29%) dropped out/withdrew, 42–46 (30–33%) lost to f'up.
Totterdell et al. (2012)	↓Anxious 1-9 scale**.	Anxiety and variation fell: baseline, 5.08, sd 1.33; therapy, 3.87, sd 1.36; follow-up, 2.23, sd 0.63. ANCOVAs significant differences baseline vs. during/after therapy; partial $\eta^2 = .12$, $F(2, 1195) = 81.51$, $p < .01$. Significant interaction between time-of-day and stage indicated less deviation each day (partial $\eta^2 = .05$, $F(6, 1195) = 10.10$, $p < .01$); and less within-day variability (partial $\eta^2 = .13$, $F(2, 318) = 23.79$, $p < .01$)	None
Van Gent et al., 1988; Van Gent & Zwart, 1993a	=STAI-T, =STAI-S, =SCL anxiety, =SCL ag'phob	STAI-S; pre, 45 psychoed, vs. 41 WL; post, 43 vs. 39; 3 m 44 vs. 42; 15 m, 43 vs. 39. STAI-T; pre, 48 vs. 42; post, 44 vs. 41; 3 m 44 vs. 41; 15 m, 43 vs. 42. SCL ag'phob; pre, 9 vs. 9; post — no data; 3 m, 9 vs. 9; 15 m, 9 vs. 9. SCL anxiety subscale; pre, 16 vs. 14; post — no data; 3 m, 16 vs. 14; 15 m, 15 vs. 14	Defined as attending less that 8 group sessions, stopped taking lithium, or were admitted. 4/34 (12%) dropped out; 2/20 from the treatment group (due to a vacation and feeling too well/sufficiently informed) and 2/14 dropped out of the control group (one was admitted, the other stopped taking lithium). Further participants lost to f'up not clearly reported.
Van Gent and Zwart (1993b), Van Gent (2000)	↓STAI-T	STAI-T pre 48 10-session group vs. 45 five-session group; 3 m 44 vs. 49; 15 m 42 vs. 45	Defined as stopping more than one group session before the end. 3/35 (9%) dropped out, of which 2/20 (10%) from group therapy and 1/15 (7%) from education group. Further participants lost to f'up not clearly reported.
Williams et al. (2008)	WG: =BAI MBCT, ↑BAI TAU	Time * group * condition interaction, $F(1,41) = 7.55$, $p = .009$. Group * condition interaction, $F(1,41) = 5.63$, $p = .032$. WG: (BPSD patients, MBCT condition) BAI, pre: 13, mild; post, 7, non-clinical, ns. (TAU condition) pre, 11, mild; post, 21, moderate, $p = .004$	3 BPSD participants started treatment did not complete final follow-up. Timing of dropout not specified.

Note. ↑Increase in anxiety scores, =No change, ↓Decreased anxiety.
ns = non significant.
ANCOVA = analysis of covariance, BEP = bipolar education program, BG = between-groups, CBT = cognitive behavioural therapy, F'up = follow-up, MBCT = mindfulness-based cognitive therapy, psychoed = psychoeducation, WBT = wellbeing therapy, WG = within-groups, WL = Waiting list control.
Measures: BAI = Beck Anxiety Inventory, BPRS = Brief Psychiatric Rating Scale, CAPS = Clinician Administered PTSD Scale, CIDI-AUTO = computerised Composite International Diagnostic Interview (Peters & Andrews, 1995; World Health Organisation, 1995); DASS = Depression Anxiety Stress Scales (Crawford & Henry, 2003; Lovibond & Lovibond, 1995); GADS = Goldberg Anxiety and Depression Scale, HAM-A = clinician-rated Hamilton Anxiety Rating Scale, MADRS = Montgomery Åsberg Depression Rating Scale (Hamilton, 1959); PCL = PTSD Checklist (Blanchard et al., 1996); PDS = Posttraumatic Diagnostic Scale (Foa, 1995); PSS14 = Perceived Stress Scale (Paulhan & Bourgeois, 1998); PSWQ = Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990); QIA = Worry and Anxiety Questionnaire (French) (Dugas et al., 2001); SADS = Schedule for Affective Disorders and Schizophrenia (Spitzer & Endicott, 1979); SCID = Structured Clinical Interview for DSM, SCL = Symptom Checklist, Ag'phob = Agoraphobia subscale, STAI-S/T = State Trait Anxiety Inventory — State / Trait version (van der Ploeg, 1982).
° Trend level $p < .1$, pertaining to the significance of last follow-up reported.
* Significant to $p < .05$, pertaining to the significance of last follow-up reported.
** Significant to $p < .01$, pertaining to the significance of last follow-up reported.
*** Significant to $p < .001$, pertaining to the significance of last follow-up reported.

3.2.2. Uncontrolled/non-randomised pilot studies

Three uncontrolled, open studies were identified. Two had follow-ups; 6 months (Dusser et al., 2009), and 8 weeks, (Reilly-Harrington et al., 2007), and one did not (Miklowitz et al., 2009).

Dusser et al.'s (2009) $N = 10$ pilot study applied a novel "stress management" therapy for BP-I and II, using CBT and decentring principles, including regular relaxation, breathing, self-soothing, and "concentration and directed attention" exercises. Within-group reductions in clinician-reported anxiety were significant. Self-reported stress improved slightly and significantly, whereas self-reported state anxiety did not.

Reilly-Harrington et al.'s (2007) $N = 10$ pilot applied CBT to rapid cycling BP-I and included five sessions of CBT anxiety management. Seven of those who started had comorbid anxiety, but diagnostic interviews were not repeated post-therapy. Data are presented only for the six treatment completers. Self-rated anxiety symptoms improved, and were maintained at eight week follow-up, but the mean decrease was not statistically significant.

Miklowitz et al. (2009) piloted (with $N = 22$) an adapted eight week MBCT course, similar to Perich et al. (2013). The target was BPSD relapse prevention (I and II), with anxiety symptoms as a secondary outcome measure. There were small effect size improvements from pre- to post-treatment in self-reported anxiety symptoms, but no tests of statistical significance due to the low numbers, and no follow-up.

3.2.3. Single-case experimental design studies

One study (Satterfield, 1999) is a single-case, experimental design in rapid cycling BP-I, however anxiety data were only collected pre- and during the treatment phase (no baseline). It applied a novel CBT intervention for BPSD symptoms, which included a) prediction and monitoring, b) prevention, and c) treatment/stabilisation. Therapy also included identifying idiosyncratic sources of stress, and subsequent systematic desensitisation and anxiety training, with the aim of preventing BPSD episode onset. Self-reported anxiety symptoms reduced over the course of therapy.

3.2.4. Summary of treatments that target both anxiety and BPSD symptoms

The addition of an anxiety component to existing CBT treatments for BPSD appears effective in reducing anxiety scores and comorbid anxiety diagnosis rates. Two large, good quality RCTs with extensive follow-ups found that comorbid anxiety rates in an unmedicated cyclothymia population (Fava et al., 2011), and anxiety symptoms in refractory BPSD (González-Isasi, et al., 2010; González Isasi, et al., 2012), reduced significantly following CBT. Results of a single-case (Satterfield, 1999) and pilot study (Reilly-Harrington et al., 2007) are also promising for CBT for rapid-cycling BP. Generalisability may be limited by the rapid-cycling population, however studies of rapid cycling BPSD may also be helpful for assessing the impact of treatment. When episodes are less frequent a much longer follow-up is needed to capture improvement in terms of acute relapse. When episodes are infrequent, inter-episode variability can also be monitored (Bonsall, Wallace-Hadrill, Geddes, Goodwin, & Holmes, 2012). These small *N* and pilot studies warrant replication in other BPSDs.

CBT for non-specific "stress" has some positive outcomes, but these appear less clear than those derived from a specific cognitive model of anxiety. As yet, the evidence appears hard to interpret for adapted MBCT although suggestive of small effect size improvements.

The evidence gathered suggests that the addition of CBT anxiety components to standard treatments for BPSD may provide a useful route for treatment development. However, it is not possible to draw firm conclusions about clinical efficacy from the small number of studies currently available. Although it has not been possible to systematically compare these here, the studies reviewed also found positive outcomes for mood symptoms; the potential benefit on depressive and manic symptoms of anxiety treatment would be worth exploring further.

3.3. Treatments that target specific anxiety disorders in a BPSD population (N = 7)

3.3.1. Randomised controlled trials

Only one RCT was identified (Mueser et al., 2008), which applied CBT for PTSD in a "severe mental illness" [SMI] population, which includes major mood disorders (including BPSD), psychotic disorders, and axis II personality disorders. The adapted CBT protocol emphasises integration with case management and cognitive restructuring techniques rather than trauma exposure, thought to be more palatable and safe for this client group (Mueser, Rosenberg, Jankowski, Hamblen, & Monica, 2004; Mueser, Rosenberg, & Rosenberg, 2009). Of $N = 108$, 25 participants had BPSD (type not specified). The between-group change on diagnosis between CBT and TAU was not significant for the whole sample using the Clinician-Administered PTSD Scale [CAPS] (Blake et al., 1995), although reached statistical significance for those with CAPS > 65 ($n = 78$) at intake. PTSD symptoms improved with a large effect size, as did self-reported anxiety symptoms, although these remained in the severe range. Results were not presented separately for the different diagnostic groups.

3.3.2. Uncontrolled/non-randomised pilot studies

Three uncontrolled studies provide further preliminary evidence for Mueser et al.'s (2004) adapted CBT protocol for PTSD also in SMI populations. Rosenberg et al.'s (2004) pilot of $N = 13$ includes two treatment completers with BPSD (type not specified). Using the CAPS, there were statistically significant within-subjects improvements in self-reported PTSD symptoms and reductions in diagnosis rates. In an ethnically and culturally diverse urban population, Lu et al. (2009) reported statistically significant within-subjects improvements in PTSD symptoms and diagnosis rates. The proportion (of $N = 14$) with BPSD was not reported. In a group format, Mueser et al. (2007) reported statistically significant within-subjects improvements on PTSD symptoms, and the slight reduction in diagnosis rates was statistically significant. Seven (of $N = 80$) had BPSD (type not specified) but results were not presented separately for the different diagnostic groups.

3.3.3. Single-case experimental design studies

One study uses an $N = 4$, multiple-baseline, single-case experimental design (Thienot et al., 2013). Three of the four participants with BP-I, II or NOS no longer met diagnostic criteria for GAD (one met criteria for anxiety NOS with high worry) following an established CBT manual (Dugas, 2004). There were reductions in self-reported symptoms of anxiety, somatic symptoms, and worry (albeit the latter not consistently maintained).

3.3.4. Case studies

Two case studies were identified, but they lack a baseline and cannot be considered experimental. Hamblen et al. (2004) described an $N = 1$ case study from Rosenberg et al.'s (2004) pilot (above), who recovered from PTSD by three month follow-up. Baer et al. (1985) reported an $N = 2$ case study of ERP for OCD with BP-I and mania-only in an inpatient setting. The author emphasised the medical stabilisation of mood symptoms required prior to the intensive ERP for OCD. Checking behaviour and obsessions reportedly reduced, and functioning and activities increased, however no formal measures or outcomes (e.g. time spent) are reported.

3.3.5. Summary of treatments for a specific co-morbid anxiety disorder

Adapted CBT for PTSD in BPSD appears promising. The only RCT (Mueser et al., 2008) finds modest reductions in diagnosis rates, perhaps explained by the control condition being a specialist multidisciplinary intervention that may include medication and "supportive counselling". However, it is noted that only approximately a quarter of this sample were BPSD, and results are not reported by diagnostic group, although the authors found no significant differences in improvement between the diagnostic groups (Mueser, personal communication, 22nd December 2011). Further research with BPSD-only populations would be welcome. Uncontrolled studies found significant reductions in self-reported symptoms. These studies benefit from fidelity checks, and generalization is aided by the treatment having been delivered by local trained clinicians, rather than specialists.

One preliminary study suggests that CBT treatment for GAD in euthymic BPSD patients may be effective, and larger feasibility studies are indicated. The results benefit from the experimental design, which provides a within-subject control. Interestingly, the participant who benefitted least had an additional diagnosis of social anxiety. Traditional ERP for OCD may be helpful, although the results should be interpreted with caution given the methodological limitations. Given developments in OCD treatment, a more recent CBT approach – which incorporates cognitive elements in addition to ERP – may be more appropriate (NICE, 2005a). Studies that apply evidence-based protocols for other anxiety disorders to people with BPSD would be welcome, such as panic with/without agoraphobia, social anxiety, and phobias. Further research should also seek to gather information on the subsequent effect of anxiety treatment on mood symptoms during an extended follow-up period since none of the current studies report on this.

3.4. Treatments that target BPSD symptoms only and report anxiety outcomes (N = 8)

3.4.1. Randomised controlled trials

Three RCTs were identified, although only one includes an active control condition, and participants blind to condition (Proudfoot et al., 2012). The other two use TAU as a comparison. Two include a 6 month follow-up (Da Costa et al., 2011; Proudfoot et al., 2012).

Proudfoot et al.'s (2012) $N = 407$ RCT applied computerised psychoeducation in a sample of recently diagnosed BPSD (type not specified) participants, and collected data on anxiety symptoms. The Bipolar Education Program [BEP] is a computerised, non-interactive, audio-visual, psychoeducation programme with no therapist contact. There were three groups; BEP alone was compared to BEP plus emails from "Informed Supporters" [IS] and to an active control. ISs were

trained and supervised "expert patients" who sent up to 2 × 300 word emails per week. Participants were recruited within a year of diagnosis (BPSD type not specified), and therefore not necessarily between episodes; only 48.16% were euthymic at intake. Information on comorbidities was not gathered. Drop-out was high, with 66% providing data at post-treatment and six-month follow-up. Descriptive statistics showing mean scores on outcome measures are not provided. There were significant within-subject improvements in anxiety and depression, but no significant between-group differences or interactions. Peer support increased adherence, but this did not affect outcome. The authors posit that non-specific factors and self-monitoring common to all three conditions (i.e. use of an active control), lack of power due to drop-out, or the failure to exclude those in episode masked any potential effect. The negative finding in this large RCT suggests that computer-based psychoeducation following diagnosis, with or without support, does not appear to reduce anxiety or other symptoms compared to reading information online.

Da Costa et al.'s (2011) $N = 41$ RCT compares group CBT, which included family members in psychoeducation sessions, to TAU. Thirty percent of participants (BP-I or II) had an axis I comorbidity (not specified), although axis I "severe psychiatric disorder" (not operationalised) and axis II comorbidities were excluded, as were participants who required a change of medication during the study. Within-group pre-post improvements in self-reported anxiety symptoms were highly significant in the CBT group and non-significant for the control group. Data from the 6-month follow-up were not reported. This study suggests that standard CBT for BPSD may also reduce anxiety but direct comparison to TAU is lacking. Thus, interpretation must be cautious due to methodological and reporting issues, critically the presentation of only within-group changes (i.e. no formal between-group comparisons of outcome), small and uneven group numbers, and lack of blinding and follow-up data.

Of $N = 55$ mood disorder participants in Williams et al.'s (2008) RCT, $n = 14$ had BPSD (type not specified) and the rest (unipolar) depression, distributed evenly between conditions. The standard 8 week MBCT group treatment was compared to TAU using a waitlist [WL] condition, and anxiety symptom data were collected. Researchers were blind to assignment, and all outcome data were by self-report. Amongst the participants with BPSD, there was a significant increase in self-reported anxiety symptoms from pre- to post-treatment in the TAU condition, but no significant change in the MBCT condition. The authors suggest that this may indicate a protective effect of MBCT on anxiety. However, the study was designed to establish feasibility of MBCT in remitted patients with mood disorders and a history of suicidal ideation or behaviour, rather than efficacy of MBCT in BPSD specifically. Thus the sample size is small and the results must be regarded as preliminary.

3.4.2. Uncontrolled/non-randomised pilot studies

Four studies were identified; one was uncontrolled (Docteur et al., 2007), two compared treatment with a WL control condition (Van Gent et al., 1988; Docteur et al., 2013), and one compared two treatment groups without random allocation (Van Gent, 2000; Van Gent & Zwart, 1993b). Two have a follow-up of 15 months (Van Gent et al., 1988; Van Gent, 2000) and one of 5–9 months (Docteur et al., 2013).

Docteur et al.'s (2007) $N = 12$ uncontrolled pilot study presents complete data for $n = 7$. Treatment was group CBT and participants had BP-I according to diagnostic criteria (assessment method not specified). Comorbid anxiety and personality disorders were allowed but numbers are not reported. There was no significant pre-post change in clinician-rated anxiety, and no follow-up.

Docteur et al.'s (2013) $N = 73$ study compared group CBT in BP-I to a WL control group without random allocation. Improvement in clinician-rated anxiety was statistically significant in the CBT group but not in TAU. This paper suggests that anxiety reduces following group CBT, but the results must be interpreted with caution due to methodological limitations: time to follow-up differs; group sizes are unequal; only within-group differences are reported (i.e. no between-group comparison), using different analytic strategies (parametric vs. non-parametric); and assessor blinding (for clinician-rated assessment) is not specified.

Van Gent, Vida, & Zwart's (1988) $N = 34$ study compares group psychoeducation to a WL control condition, method of allocation not specified. Anxiety symptoms were unchanged, and psychoeducation did not reduce anxiety compared to WL control condition.

Data from the treatment described in the above study is then compared in two papers (Van Gent, 2000; Van Gent & Zwart, 1993b) to data from a condensed "ultra-short" version with additional written materials (five sessions), precluding random allocation. There was no significant difference in anxiety outcomes between the 10-session and ultra-short conditions. Overall, these psychoeducation groups do not appear to improve anxiety significantly.

3.4.3. Single-case experimental design studies

Totterdell et al.'s (2012) $N = 1$ "intensive time-sampling," multiple-baseline, single-case experimental design adapted Basco and Rush's (2005) CBT protocol for cyclothymia. Anxiety was measured using a scale of one (not at all) to nine (a great extent) (Matthews, Jones, & Chamberlain, 1990) four times daily for 51 weeks; five weeks baseline, 35 weeks therapy, and 11 weeks follow-up. It was hypothesised that anxiety would improve, although it was not directly targeted in treatment. Anxiety and variation therein – as assessed by standard deviations – improved significantly during/after therapy compared to baseline. Improvement on all measures was mediated by a subjective measure of cognitive control, consistent with the Mansell et al. (2007) model of BP. This study suggests that targeting mood stability can improve anxiety, and identifies a possible mechanism for change.

3.4.4. Summary of treatments that target BPSD symptoms only

There is no evidence from the studies reviewed here that psychoeducation alone reduces anxiety in BPSD. Individual CBT for cyclothymia appeared to reduce anxiety in a single-case design and warrants further research. One RCT found individual CBT for BP-I and II that included family members significantly reduced anxiety scores. Results from non-controlled and/or non-randomised studies find at best modest results for group CBT for BPSD. The subgroup of BPSD patients in an RCT of MBCT showed no significant reduction in symptoms of anxiety following MBCT, but did not show the increase in anxiety found in BPSD patients in the TAU arm.

4. Discussion

This paper collates and critically evaluates existing research into psychological treatments for anxiety in BPSDs. Anxiety symptoms and comorbidity are common in BPSD and confer poor outcomes in several domains. Thus, treating anxiety in BPSD may reduce distress and improve outcomes. Clearly the literature is in its infancy, but nevertheless offers useful insights for clinicians and researchers.

All studies but one (Proudfoot et al., 2012) appear to apply treatment inter-episode (i.e. between acute episodes of depression/mania), but the definition of euthymia varies and a direct comparison between studies is not possible. This review focuses only on anxiety symptoms, and the relationship between anxiety and residual mood symptoms remains to be examined. Future studies should seek to gather parallel data to elucidate the links between anxiety, inter-episodic mood symptoms, and treatment. This could be pertinent to the development of specific models of BPSD anxious distress, and further specification of the time course of mood instability and its link to anxiety may aid the development of effective treatments (Bonsall et al., 2012).

4.1. Clinical implications

The studies reviewed above are extremely heterogeneous, in terms of patient characteristics, treatments applied, design, and outcome measurement, and this limits the extent to which clear clinical implications can be drawn. Therefore we may have to conclude that it can be useful to target anxiety in bipolar disorder via a psychological treatment approach, but that at present it is not clear how best to do so. Based on the small number of available RCTs, CBT with specific additional components targeted at managing anxiety appears to have the most support.

The possibility of reducing anxiety in BPSD via a specific anxiety component is promising given the paucity of pharmacological alternatives to treat anxiety in this patient group (see earlier). Indeed, pharmacological management of anxiety symptoms in BPSD can be complex and include the use of multiple drugs, thus increasing the risk of physical side effects (Goodwin et al., 2009). This could be minimised if anxious distress was instead reduced by effective psychological interventions. Therefore successful psychological treatment approaches for anxiety in BPSD are likely to have a positive impact on the general long term management of the disorder. In fact, they could be a crucial advance in the treatment of populations presenting with particularly high levels of anxiety and for whom pharmacological interventions are most controversial, such as youth with BPSD.

All the studies presented here applied treatment between mood episodes and in the presence of medication, suggesting that a psychological approach to anxiety could be added to an existing treatment regime. As the presence of anxiety has been shown to also have a direct negative impact on adherence to pharmacological treatment (Perlis et al., 2010), targeted psychological interventions for anxiety might also improve medication compliance and thus lead to better prognosis, although this remains to be assessed.

4.2. Research implications

While small *N* studies are appropriate at an early stage in treatment development, future studies should consider the use of more formal study designs and analytic strategies. More rigorous studies are required to further the field. For example, data on safety, acceptability and feasibility has not been systematically collected except by a handful of studies (e.g. Rosenberg et al., 2004); future studies should collect and report this (Dimidjian & Hollon, 2010).

The phenomenology and treatment of anxiety in different stages of BPSD may be distinct. Although it is not stated, most studies included in this review can be thought of as treating residual phase symptoms (Cosci & Fava, 2012) rather than prodromal or acute phases. Future studies could use these definitions to specify their target population, and to compare the effectiveness of applying treatments during different phases. As already mentioned, research is especially warranted for prodromal stages of BPSD and youth populations, where anxiety is prominent and there is a particular lack of available interventions (Kapczinski et al., 2014).

It would be valuable for treatment research into BPSD to include measures of anxiety as standard. Measures should be repeated at regular intervals to allow for the detection of change. Given the research suggesting a potentially reciprocal relationship between anxiety and mood symptoms in BPSD (Deckersbach et al., 2013; Holmes et al., 2011), it would be useful to also research the effect of a specific anxiety-focussed treatment and its impact on mood symptoms. Anxiety may also be a key mediator of instability in BPSD (Holmes et al., 2008), and statistical analyses would be useful to explore how mood and anxiety symptoms may reciprocally affect each other (c.f. Maguire, McCusker, Meenagh, Mulholland, & Shannon, 2008).

Given the considerable heterogeneity of treatment approaches available, an important goal for future research would be to identify which specific treatment components or techniques may be effective in reducing BPSD anxiety, and their mechanisms of change, as this would allow development of briefer, more focussed, interventions (cf. Geddes & Miklowitz, 2013). The available evidence suggests that including a treatment component with a specific anxiety focus into CBT approaches is helpful. CBT approaches may have several key core features, such as having a structured, problem-focused and action-oriented approach, formulation, and the systematic application of cognitive and behavioural treatment techniques. However, there is also considerable heterogeneity, for example in the kinds of techniques that can be used within this framework, and future work should seek to detail the specific anxiety-focussed techniques used. Currently it is unclear how to maximize the effectiveness of an anxiety-focussed approach in BPSD. It may be important to develop and test techniques designed specifically around the phenomenology of BPSD anxiety, rather than only importing modules from formulations of other axis 1 disorders, as has been suggested for BP depression (Mansell, Colom, & Scott, 2005). Such a treatment development process would benefit from the identification of BPSD-specific cognitive processes related to anxiety in this population, in order to provide targets for a focussed therapy.

For example, there is emerging evidence for a bias towards elevated mental-imagery based processing in BPSD (e.g. Gregory, Brewin, Mansell, & Donaldson, 2010; Gruber, Harvey, & Johnson, 2009; Hales, Deeprose, Goodwin, & Holmes, 2011; Holmes et al., 2011; Ivins, Di Simplicio, Close, Goodwin, & Holmes, 2014), which has been hypothesised to play a key role in BPSD anxiety and mood instability (Holmes et al., 2008). For anxiety disorders in which problematic mental images are thought to play a central role in maintenance of distress (e.g. flashbacks in PTSD; Ehlers & Clark, 2000), working with these images to reduce their impact is a key part of many successful treatment protocols. Thus, one possibility is that mental imagery-based treatment techniques developed in the context of CBT for anxiety disorders (e.g. Holmes, Arntz, & Smucker, 2007) may prove useful for anxiety BPSD. However, these techniques may also (as noted above) require adapting for people with BPSD and the nature of their problematic imagery. One example might be to specifically tackle vivid intrusive imagery that has been found to be elevated in bipolar disorder and linked to mood instability (Holmes et al., 2011), and to which those with hypomanic experience are vulnerable (Malik, Goodwin, Hoppitt, & Holmes, 2014). There are likely to be other cognitive processes that could also offer targets for treatment innovation. Identifying and characterising such cognitive processes in BPSD and their relation to anxiety and mood instability could present a useful step forward in guiding development of more effective BPSD-tailored approaches (Geddes & Miklowitz, 2013).

4.3. Limitations

In the current review, there is a risk of selection bias due to searching only titles and abstracts for “anxiety” or related terms; studies that found significant results may have been more likely to report anxiety measures in the abstract, whereas studies with negative findings on anxiety measures may have omitted to mention anxiety in the abstract.

This review aimed to reduce bias by including papers published in any language; though relied on a supplementary abstract or title in English. Four studies in French are included (Docteur et al., 2007; Dusser et al., 2009; Thienot et al., 2013; Van Gent, 2000). This review aimed to collate all relevant studies, without limitation on year of publication, methodology, sample size, or other stipulations, which allows as thorough a review as possible. However, it prevents formal synthesis of the data.

Investigating the effect of treatments for anxiety on the stability of BPSD symptoms and on long-term relapse rates was outside the scope of this review, and would be a valuable contribution for future research (e.g. preliminary studies such as Fava et al., 2001).

5. Conclusions

Research investigating psychological treatment for anxiety in BPSD is much needed yet still in its infancy; few studies exist and the field would

benefit from more RCTs with adequate power. However, studies to date that have applied psychological therapy inter-episode (i.e. between acute episodes of mania or depression) have found benefits for anxiety, even if modest. There is no empirical basis to suggest that psychological treatment is harmful or ineffective in this population. These findings help to establish a role for existing psychological interventions for anxiety, and the need for development of tailored psychological interventions for anxious distress in BPSD. Future research should focus on identifying the specific treatment components responsible for reducing anxiety in BPSD, and the key psychological processes to target via therapy.

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Conflict of interest

The authors declare that they have no conflicts of interest

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Appendix A. Systematic search strategy and results

Date searched 16/05/2013.

1	AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, HEALTH BUSINESS ELITE	("bipolar disorder" OR "bipolar affective disorder" OR "manic depression" OR cyclothymia OR "rapid cycling" OR "bipolar I" OR "bipolar II" OR "bipolar 2" OR "bipolar NOS" OR "bipolar spectrum disorder").ti,ab	58586
2	AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, HEALTH BUSINESS ELITE	(anxiety OR anxious).ti,ab	409893
3	AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, HEALTH BUSINESS ELITE	(psychoeducation OR psychotherapy OR "psychological therapy" OR "cognitive therapy" OR "cognitive behavio* therapy" OR "CBT" OR "family therapy").ti,ab	192661
4	AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, HEALTH BUSINESS ELITE	1 AND 2 AND 3	288
5	AMED, EMBASE, HMIC, MEDLINE, PsycINFO, BNI, CINAHL, HEALTH BUSINESS ELITE	Duplicate filtered: [1 AND 2 AND 3]	288 109 unique results 179 duplicate results

References

Albert, U., Rosso, G., Maina, G., & Bogetto, F. (2008). Impact of anxiety disorder comorbidity on quality of life in euthymic bipolar disorder patients: Differences between bipolar I and II subtypes. *Journal of Affective Disorders*, *105*, 297–303. http://dx.doi.org/10.1016/j.jad.2007.05.020.

American Psychiatric Association (2013a). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

American Psychiatric Association (2013b). *Highlights of changes from DSM-IV-TR to DSM-5.* Washington, DC: American Psychiatric Association.

Arrindell, W. A., & Ettema, J. H. M. (1986). *SCL-90 Handleiding bij een multidimensionele psychopathologie-indicator [Manual for a Multidimensional Psychopathology Indicator].* Lisse, The Netherlands: Swets en Zeitlinger.

Baer, L., Minichiello, W. E., & Jenike, M. A. (1985). Behavioral treatment in two cases of obsessive–compulsive disorder with concomitant bipolar affective disorder. *American Journal of Psychiatry*, *142*, 358.

Balon, R. (2013). Benzodiazepines revisited. *Psychotherapy and Psychosomatics*, *82*, 353–354. http://dx.doi.org/10.1159/000353599.

Barlow, D. H., & Craske, M. G. (2006). *Mastery of your anxiety and panic, workbook* (4th ed.). New York: Graywinds.

Basco, M. R., & Rush, A. J. (1996). *Cognitive-behavioral therapy for bipolar disorder.* New York: Guilford Press.

Basco, M. R., & Rush, A. J. (2005). *Cognitive behaviour therapy for bipolar disorder* (2nd ed.). New York: Guilford Press.

Bech, P., Kastrup, M., & Rafaelsen, O. J. (1986). Mini-compendium of rating scales for states of anxiety, depression, mania, schizophrenia with corresponding DSM III syndromes. *Acta Psychiatrica Scandinavica*, *73*(Suppl. 326), 1–37.

Bech, P., Rafaelsen, O. J., Kramp, P., & Bolwig, T. G. (1978). The mania rating scale: Scale construction and inter-observer agreement. *Neuropharmacology*, *17*, 430–431. http://dx.doi.org/10.1016/0028-3908(78)90022-9.

Beck, A. T., & Beamesderfer, A. (1974). *Assessment of depression: The depression inventory.* Basel, Switzerland: S. Karger.

Beck, A. T., & Steer, R. A. (1993). *BAI, Beck anxiety inventory: Manual.* San Antonio, TX: Psychological Corporation.

Beck, A. T., Ward, C., & Mendelson, M. (1961). Beck depression inventory (BDI). *Archives of General Psychiatry*, *4*, 561–571. http://dx.doi.org/10.1001/archpsyc.1961.01710120031004.

Berk, M., Berk, L., Dodd, S., Cotton, S., Macneil, C., & Daglas, R. (2013). Stage managing bipolar disorder. *Bipolar Disorders*, *16*, 471–477. http://dx.doi.org/10.1111/bdi.12099.

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., et al. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, *8*, 75–90. http://dx.doi.org/10.1002/jts.2490080106.

Blanchard, E. P., Jones-Alexander, J., Buckley, T. C., & Forneris, C. A. (1996). Psychometric properties of the PTSD Checklist. *Behavior Therapy*, *34*, 669–673. http://dx.doi.org/10.1016/0005-7967(96)00033-2.

Bonsall, M. B., Wallace-Hadrill, S. M. A., Geddes, J. R., Goodwin, G. M., & Holmes, E. A. (2012). Non-linear time series approaches in characterising mood stability and mood instability in bipolar disorder. *Proceedings of the Royal Society B: Biological Sciences*, *279*(1730), 916–924. http://dx.doi.org/10.1098/rspb.2011.1246.

Cochrane Collaboration (2011). *The Cochrane handbook for systematic reviews of interventions: Version 5.1.0.* (Retrieved from http://www.cochrane-handbook.org).

Cosci, F., & Fava, G. A. (2012). Staging of mental disorders: Systematic review. *Psychotherapy and Psychosomatics*, *82*, 20–34. http://dx.doi.org/10.1159/000342243.

Crawford, J. R., & Henry, J. D. (2003). The Depression Anxiety Stress Scales (DASS): Normative data and latent structure in a large non-clinical sample. *British Journal of Clinical Psychology*, *42*, 111–131. http://dx.doi.org/10.1348/014466503321903544.

Da Costa, R. T., Cheniaux, E., Rosaes, P. A. L., Carvalho, M. R., Freire, R. C. R., Versiani, M., et al. (2011). The effectiveness of cognitive behavioral group therapy in treating bipolar disorder: A randomised controlled study. *Revista Brasileira de Psiquiatria*, *33*, 144–149. http://dx.doi.org/10.1590/S1516-44462011000200009.

Deckersbach, T., Peters, A. T., Sylvia, L., Urdahl, A., Magalhães, P. V., Otto, M. W., et al. (2013). Do comorbid anxiety disorders moderate the effects of psychotherapy for bipolar disorder? Results from STEP-BD. *American Journal of Psychiatry*, *171*, 178–186. http://dx.doi.org/10.1176/appi.ajp.2013.13020225.

Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behavioral Science*, *19*, 1–15. http://dx.doi.org/10.1002/bs.3830190102.

Dimidjian, S., & Hollon, S. D. (2010). How would we know if psychotherapy were harmful? *American Psychologist*, *65*, 21. http://dx.doi.org/10.1037/a0017299.

Docteur, A., Mirabel-Sarron, C., Guelfi, J. -D., Rouillon, F., & Gorwood, P. (2013). The role of CBT in explicit memory bias in bipolar I patients. *Journal of Behavior Therapy and Experimental Psychiatry*, *44*, 307–311. http://dx.doi.org/10.1016/j.jbtep.2012.12.003.

Docteur, A., Mirabel-Sarron, C., Urdapilleta, I., Siobud-Dorocant, E., Guelfi, J. D., & Rouillon (2007). Group therapy with bipolar patients: A clinical and cognitive assessment. [Original Title: Evaluation clinique et cognitive d'une therapie comportementale et cognitive de groupe chez des patients bipolaires I.]. *Journal de Thérapie Comportementale et Cognitive*, *17*, 79–83. http://dx.doi.org/10.1016/S1155-1704(07)89709-6.

Duffy, A., Horrocks, J., Doucette, S., Keown-Stoneman, C., McCloskey, S., & Grof, P. (2013). The developmental trajectory of bipolar disorder. *British Journal of Psychiatry*, *204*, 122–128. http://dx.doi.org/10.1192/bjp.bp.113.126706.

Dugas, M. J. (2004) *Manuel du thérapeute pour le traitement du trouble d'anxiété généralisée.* [Treatment manual for generalised anxiety disorder] Unpublished treatment manual, Université Concordia, Montréal.

Dugas, M. J., Freeston, M. H., Provencher, M. D., Lachance, S., Ladouceur, R., & Gosselin, P. (2001). Le questionnaire sur l'inquiétude et l'anxiété: Validations dans des échantillons non cliniques et cliniques [The Worry and Anxiety Questionnaire: Validation in non-clinical and clinical samples]. *Journal de Thérapie Comportementale et Cognitive*, *11*, 31–36.

Dusser, I., Romo, L., & Leboyer, M. (2009). Élaboration et évaluation d'un programme de gestion du stress pour patients souffrant de troubles bipolaires [Construction and evaluation of a stress management group for patients with bipolar disorder]. *Journal de Thérapie Comportementale et Cognitive*, *19*, 56–60. http://dx.doi.org/10.1016/j.jtcc.2009.08.002.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319–345. http://dx.doi.org/10.1016/S0005-7967(99)00123-0.

El-Mallakh, R. S., & Hollifield, M. (2008). Comorbid anxiety in bipolar disorder alters treatment and prognosis. *Psychiatric Quarterly*, *79*, 139–150. http://dx.doi.org/10.1007/s11126-008-9071-5.

Fava, G. A. (1999). Well-being therapy: Conceptual and technical issues. *Psychotherapy and Psychosomatics*, *68*, 171–179. http://dx.doi.org/10.1159/000012329.

Fava, G. A., Bartolucci, G., Rafanelli, C., & Mangelli, L. (2001). Cognitive-behavioral management of patients with bipolar disorder who relapsed while on lithium prophylaxis. *Journal of Clinical Psychiatry*, *62*, 556–559. http://dx.doi.org/10.4088/JCP.v62n07a10.

Fava, G. A., Rafanelli, C., Tomba, E., Guidi, J., & Grandi, S. (2011). The sequential combination of cognitive behavioral treatment and well-being therapy in cyclothymic disorder. *Psychotherapy and Psychosomatics*, *80*, 136–143. http://dx.doi.org/10.1159/000321575.

Fava, G. A., & Tomba, E. (2009). Increasing psychological well-being and resilience by psychotherapeutic methods. *Journal of Personality*, *77*, 1903–1934. http://dx.doi.org/10.1111/j.1467-6494.2009.00604.x.

Feske, U., Frank, E., Mallinger, A. G., Houck, P. R., Fagiolini, A., Shear, M. K., et al. (2000). Anxiety as a correlate of response to the acute treatment of bipolar I disorder. *American Journal of Psychiatry*, *157*, 956–962. http://dx.doi.org/10.1176/appi.ajp.157.6.956.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV Axis I disorders, clinician version (SCID-CV).* Washington, D.C.: American Psychiatric Press, Inc.

Foa, E. B. (1995). *Posttraumatic stress diagnostic scale manual.* Minneapolis, MN: National Computer Systems, Inc.

Geddes, J. R., & Miklowitz, D. J. (2013). Treatment of bipolar disorder. *The Lancet*, *381*, 1672–1682. http://dx.doi.org/10.1016/S0140-6736(13)60857-0.

Ghaemi, S. N., Boiman, E. E., & Goodwin, F. K. (1999). Kindling and second messengers: An approach to the neurobiology of recurrence in bipolar disorder. *Biological Psychiatry*, *45*, 137–144. http://dx.doi.org/10.1016/S0006-3223(98)00256-X.

Goldberg, D., Bridges, K., Duncan-Jones, P., & Grayson, D. (1988). Detecting anxiety and depression in general medical settings. *British Medical Journal*, *297*, 897.

Goldberg, D., & Fawcett, J. (2012). The importance of anxiety in both major depression and bipolar disorder. *Depression and Anxiety*, *29*, 471–478. http://dx.doi.org/10.1002/da.21939.

González Isasi, A., Echeburúa, E., Limiñana, J. M., & González-Pinto, A. (2012). Psychoeducation and cognitive-behavioral therapy for patients with refractory bipolar disorder: A 5-year controlled clinical trial. *European Psychiatry*, *29*, 134–141. http://dx.doi.org/10.1016/j.eurpsy.2012.11.002.

González-Isasi, A., Echeburúa, E., Mosquera, F., Ibáñez, B., Aizpuru, F., & González-Pinto, A. (2010). Long-term efficacy of a psychological intervention program for patients with refractory bipolar disorder: A pilot study. *Psychiatry Research*, *176*, 161–165. http://dx.doi.org/10.1016/j.psychres.2008.06.047.

Goodwin, G. M. (2003). Evidence-based guidelines for treating bipolar disorder: Recommendations from the British Association for Psychopharmacology. *Journal of Psychopharmacology*, *17*, 149–173. http://dx.doi.org/10.1177/0269881103017002003.

Goodwin, G. M., & Consensus Group of the British Association for Psychopharmacology (2009). Evidence-based guidelines for treating bipolar disorder: Revised second edition-recommendations from the British Association for Psychopharmacology. *Journal of Psychopharmacology*, *23*, 346–388. http://dx.doi.org/10.1177/0269881109102919.

Goodwin, G., Fleischhacker, W., Arango, C., Baumann, P., Davidson, M., De Hert, M., et al. (2009). Advantages and disadvantages of combination treatment with antipsychotics: ECNP Consensus Meeting, March 2008, Nice. *European Neuropsychopharmacology*, *19*, 520–532. http://dx.doi.org/10.1016/j.euroneuro.2009.04.003.

Gregory, J. D., Brewin, C. R., Mansell, W., & Donaldson, C. (2010). Intrusive memories and images in bipolar disorder. *Behaviour Research and Therapy*, *48*, 698e703. http://dx.doi.org/10.1016/j.brat.2010.04.005.

Grimes, D. A., & Schulz, K. F. (2002). An overview of clinical research: The lay of the land. *The Lancet*, *359*, 57–61. http://dx.doi.org/10.1016/S0140-6736(02)07283-5.

Gruber, J., Harvey, A. G., & Johnson, S. L. (2009). Reflective and ruminative processing of positive emotional memories in bipolar disorder and healthy controls. *Behaviour Research and Therapy*, *47*, 697e704.

Guidi, J., Fava, G. A., Bech, P., & Paykel, E. (2010). The Clinical Interview for Depression: A comprehensive review of studies and clinimetric properties. *Psychotherapy and Psychosomatics*, *80*, 10–27. http://dx.doi.org/10.1159/000317532.

Hales, S. A., Deeprose, C., Goodwin, G. M., & Holmes, E. A. (2011). Cognitions in bipolar affective disorder and unipolar depression: Imagining suicide. *Bipolar Disorders*, *13*, 651–661. http://dx.doi.org/10.1111/j.1399-5618.2011.00954.x.

Hamblen, J. L., Jankowski, M. K., Rosenberg, S. D., & Mueser, K. T. (2004). Cognitive-behavioral treatment for PTSD in people with severe mental illness: Three case studies. *American Journal of Psychiatric Rehabilitation*, *7*, 147–170. http://dx.doi.org/10.1080/15487760490476192.

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology*, *32*, 50–55. http://dx.doi.org/10.1111/j.2044-8341.1959.tb00467.x.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, *23*, 56. http://dx.doi.org/10.1136/jnnp.23.1.56.

Hans, E., & Hiller, W. (2013). A meta-analysis of nonrandomised effectiveness studies on outpatient cognitive behavioural therapy for adult anxiety disorders. *Clinical Psychology Review*, *33*, 954–964. http://dx.doi.org/10.1016/j.cpr.2013.07.003.

Henry, C., Van den Bulke, D., Bellivier, F., Etain, B., Rouillon, F., & Leboyer, M. (2003). Anxiety disorders in 318 bipolar patients: Prevalence and impact on illness severity and response to mood stabilizer. *Journal of Clinical Psychiatry*, *64*, 331–335. http://dx.doi.org/10.4088/JCP.v64n0316.

Hirschfeld, R. M. A., Bowden, C. L., Gitlin, M. J., Keck, P. E., Suppes, T., & Thase, M. E. (2004). *Treatment of patients with bipolar disorder.* American Psychiatric Association Practice Guidelines for the treatment of psychiatric disorders Compendium. Arlington, VA: American Psychiatric Association, 525–612.

Hoertel, N., Le Strat, Y., Blanco, C., Lavaud, P., & Dubertret, C. (2012). Generalisability of clinical trial results for generalized anxiety disorder to community samples. *Depression and Anxiety*, *29*, 614–620. http://dx.doi.org/10.1002/da.21937.

Hofmann, S. G., Asnaani, A., Vonk, I. J. J., Sawyer, A. T., & Fang, A. (2012). The efficacy of cognitive behavioral therapy: A review of meta-analyses. *Cognitive Therapy and Research*, *36*, 427–440. http://dx.doi.org/10.1007/s10608-012-9476-1 (0147-5916;1573-2819).

Holmes, E. A., Arntz, A., & Smucker, M. R. (2007). Imagery rescripting in cognitive behaviour therapy: Images, treatment techniques and outcomes. *Journal of Behavior Therapy and Experimental Psychiatry*, *38*, 297–305. http://dx.doi.org/10.1016/j.jbtep.2007.10.007.

Holmes, E. A., Deeprose, C., Fairburn, C. G., Wallace-Hadrill, S. M. A., Bonsall, M. B., Geddes, J. R., et al. (2011). Mood stability versus mood instability in bipolar disorder: A possible role for emotional mental imagery. *Behaviour Research and Therapy*, *49*, 707–713. http://dx.doi.org/10.1016/j.brat.2011.06.008.

Holmes, E. A., Geddes, J. R., Colom, F., & Goodwin, G. M. (2008). Mental imagery as an emotional amplifier: Application to bipolar disorder. *Behaviour Research and Therapy*, *46*, 1251–1258. http://dx.doi.org/10.1016/j.brat.2008.09.005.

Ivins, A., Di Simplicio, M., Close, H., Goodwin, G. M., & Holmes, E. (2014). Mental imagery in bipolar affective disorder versus unipolar depression: Investigating cognitions at times of 'positive'mood. *Journal of Affective Disorders*, *166*, 234–242. http://dx.doi.org/10.1016/j.jad.2014.05.007.

Johnson, S. L. (2005). Life events in bipolar disorder: Towards more specific models. *Clinical Psychology Review*, *25*, 1008–1027. http://dx.doi.org/10.1016/j.cpr.2005.06.004.

Jones, S. H. (2001). Circadian rhythms, multilevel models of emotion and bipolar disorder—An initial step towards integration? *Clinical Psychology Review*, *21*, 1193–1209. http://dx.doi.org/10.1016/S0272-7358(01)00111-8.

Jones, S. H., McGrath, E., Hampshire, K., Owens, R., Riste, L., Roberts, C., et al. (2013). A randomised controlled trial of time limited CBT informed psychological therapy for anxiety in bipolar disorder. *BMC Psychiatry*, *13*, 54. http://dx.doi.org/10.1186/1471-244X-13-54.

Kapczinski, F., Magalhães, P. V. S., Balanzá-Martinez, V., Dias, V. V., Frangou, S., Gama, C. S., et al. (2014). Staging systems in bipolar disorder: An International Society for Bipolar Disorders Task Force Report. *Acta Psychiatrica Scandinavica*, *130*, 354–363. http://dx.doi.org/10.1111/acps.12305.

Kauer-Sant Anna, M., Frey, B. N., Andreazza, A. C., Cereser, K. M., Gazalle, F. K., Tramontina, J., et al. (2007). Anxiety comorbidity and quality of life in bipolar disorder patients. *Canadian Journal of Psychiatry*, *52*, 175.

Lam, D. H., Hayward, P., Watkins, E. R., Wright, K., & Sham, P. (2005). Relapse prevention in patients with bipolar disorder: Cognitive therapy outcome after 2 years. *American Journal of Psychiatry*, *162*, 324–329. http://dx.doi.org/10.1176/appi.ajp.162.2.324.

Lam, D. H., Jones, S. H., & Hayward, P. (2010). *Cognitive therapy for bipolar disorder: A therapist's guide to concepts, methods and practice.* West Sussex: Wiley-Blackwell.

Lam, D. H., Jones, S., Hayward, P., & Bright, J. (1999). *Cognitive therapy for bipolar disorder: A therapist's guide to concepts, methods and practice.* New York: John Wiley & Sons.

Lee, J. H., & Dunner, D. L. (2008). The effect of anxiety disorder comorbidity on treatment resistant bipolar disorders. *Depression and Anxiety*, *25*, 91–97. http://dx.doi.org/10.1002/da.20279.

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the depression anxiety stress scales* (2nd ed.). Sydney: Psychology Foundation.

Lu, W., Fite, R., Kim, E., Hyer, L., Philip, T. Y., Mueser, K. T., et al. (2009). Cognitive-behavioral treatment of PTSD in severe mental illness: Pilot study replication in an ethnically diverse population. *American Journal of Psychiatric Rehabilitation*, *12*, 73–91. http://dx.doi.org/10.1080/15487760802615863.

Lukoff, D., Nuechterlein, K. H., & Ventura, J. (1986). Manual for the expanded brief psychiatric rating scale (BPRS). *Schizophrenia Bulletin*, *12*, 594–602.

Lynch, D., Laws, K. R., & McKenna, P. J. (2010). Cognitive behavioural therapy for major psychiatric disorder: Does it really work? A meta-analytical review of well-controlled trials. *Psychological Medicine*, *40*, 9–24. http://dx.doi.org/10.1017/S003329170900590X.

Maguire, C., McCusker, C. G., Meenagh, C., Mulholland, C., & Shannon, C. (2008). Effects of trauma on bipolar disorder: The mediational role of interpersonal difficulties and alcohol dependence. *Bipolar Disorders*, *10*, 293–302. http://dx.doi.org/10.1111/j.1399-5618.2007.00504.x.

Malik, A., Goodwin, G. M., Hoppitt, L., & Holmes, E. A. (2014). Hypomanic experience in young adults confers vulnerability to intrusive imagery after experimental trauma: Relevance for bipolar disorder. *Clinical Psychological Science*, *2*, 675–684. http://dx.doi.org/10.1177/2167702614527433.

Mansell, W. (2007). An integrative formulation-based cognitive treatment of bipolar disorders: Application and illustration. *Journal of Clinical Psychology*, *63*, 447–461. http://dx.doi.org/10.1002/jclp.20369.

Mansell, W., Colom, F., & Scott, J. (2005). The nature and treatment of depression in bipolar disorder: A review and implications for future psychological investigation. *Clinical Psychology Review*, *25*, 1076–1100. http://dx.doi.org/10.1016/j.cpr.2005.06.007.

Mansell, W., Morrison, A. P., Reid, G., Lowens, I., & Tai, S. (2007). The interpretations of, and responses to, changes in internal states: An integrative cognitive model of mood swings. *Behavioural and Cognitive Psychotherapy*, *35*, 515–539.

Mansell, W., Powell, S., Pedley, R., Thomas, N., & Jones, S. A. (2010). The process of recovery from bipolar I disorder: A qualitative analysis of personal accounts in relation to an integrative cognitive model. *British Journal of Clinical Psychology*, *49*, 193–215. http://dx.doi.org/10.1348/014466509X451447.

Marks, I. M. (1987). *Fears, phobias, and rituals: Panic, anxiety, and their disorders.* New York, NY: Oxford University Press.

Matthews, G., Jones, D. M., & Chamberlain, A. G. (1990). Refining the measurement of mood: The UWIST mood adjective checklist. *British Journal of Psychology*, *81*, 17–42. http://dx.doi.org/10.1111/j.2044-8295.1990.tb02343.x.

McIntyre, R., Konarski, J., Soczynska, J., Wilkins, K., Panjwani, G., Bouffard, B., et al. (2006). Medical comorbidity in bipolar disorder: Implications for functional outcomes and health service utilization. *Psychiatric Services*, *57*, 1140–1144. http://dx.doi.org/10.1176/appi.ps.57.8.1140.

McIntyre, R. S., Soczynska, J. K., Bottas, A., Bordbar, K., Konarski, J. Z., & Kennedy, S. H. (2006). Anxiety disorders and bipolar disorder: A review. *Bipolar Disorders*, *8*, 665–676.

Merikangas, K. R., Akiskal, H. S., Angst, J., Greenberg, P. E., Hirschfeld, R. M., Petukhova, M., et al. (2007). Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey replication. *Archives of General Psychiatry*, *64*, 543–552. http://dx.doi.org/10.1001/archpsyc.64.5.543.

Merikangas, K. R., Jin, R., He, J. P., Kessler, R. C., Lee, S., Sampson, N. A., et al. (2011). Prevalence and correlates of bipolar spectrum disorder in the World Mental Health Survey Initiative. *Archives of General Psychiatry*, *68*, 241–251. http://dx.doi.org/10.1001/archgenpsychiatry.2011.12.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*, 487–496. http://dx.doi.org/10.1016/0005-7967(90)90135-6.

Miklowitz, D. J., Alatiq, Y., Goodwin, G. M., Geddes, J. R., Fennell, M. J., Dimidjian, S., et al. (2009). A pilot study of mindfulness-based cognitive therapy for bipolar disorder. *International Journal of Cognitive Therapy*, *2*, 373–382. http://dx.doi.org/10.1521/ijct.2009.2.4.373.

Miklowitz, D. J., Otto, M. W., Frank, E., Reilly-Harrington, N. A., Kogan, J. N., Sachs, G. S., et al. (2007). Intensive psychosocial intervention enhances functioning in patients with bipolar depression: Results from a 9-month randomized controlled trial. *American Journal of Psychiatry*, *164*, 1340. http://dx.doi.org/10.1176/appi.ajp.2007.07020311.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D. G., & The PRISMA Group (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *PLoS Medicine*, *6*(6), e1000097. http://dx.doi.org/10.1371/journal.pmed1000097 (www.prisma-statement.org).

Montgomery, S. A., & Åsberg, M. (1979). A new depression scale designed to be sensitive to change. *The British Journal of Psychiatry*, *134*, 382–389. http://dx.doi.org/10.1192/bjp.134.4.382.

Mueser, K. T., Bolton, E., Carty, P. C., Bradley, M. J., Ahlgren, K. F., DiStaso, D. R., et al. (2007). The trauma recovery group: A cognitive-behavioral program for post-traumatic stress disorder in persons with severe mental illness. *Community Mental Health Journal*, *43*, 281–304. http://dx.doi.org/10.1007/s10597-006-9075-2.

Mueser, K. T., Rosenberg, S. D., Jankowski, M. K., Hamblen, J. L., & Monica, D. (2004). A cognitive-behavioral treatment program for posttraumatic stress disorder in persons with severe mental illness. *American Journal of Psychiatric Rehabilitation*, *7*, 107–146. http://dx.doi.org/10.1080/15487760490476183.

Mueser, K. T., Rosenberg, S. D., & Rosenberg, H. J. (2009). *Treatment of posttraumatic stress disorder in special populations: A cognitive restructuring program.* Washington, DC: American Psychological Association.

Mueser, K. T., Rosenberg, S. D., Xie, H., Jankowski, M. K., Bolton, E. E., Lu, W., et al. (2008). A randomised controlled trial of cognitive-behavioral treatment for posttraumatic stress disorder in severe mental illness. *Journal of Consulting and Clinical Psychology*, *76*, 259–271. http://dx.doi.org/10.1037/0022-006X.76.2.259.

National Institute for Health and Clinical Excellence (2005a). *Post-traumatic stress disorder (PTSD): The management of PTSD in adults and children in primary and secondary care.* London: Author.

National Institute for Health and Clinical Excellence (2005b). *Obsessive compulsive disorder (OCD) and body dysmorphic disorder (BDD). CG31.* London: Author.

National Institute for Health and Clinical Excellence (2011). *Generalised anxiety disorder and panic disorder (with or without agoraphobia) in adults: Management in primary, secondary and community care CG113.* London: Author.

National Institute for Health and Clinical Excellence (2013). *Social anxiety disorder: Recognition, assessment and treatment of social anxiety disorder, CG159.* London: Author.

National Institute for Health and Clinical Excellence (2014). *Bipolar disorder: The assessment and management of bipolar disorder in adults, children and young people in primary and secondary care CG185.* London: Author.

Newman, C. E., Leahy, R. L., Beck, A. T., Reilly-Harrington, N. A., & Gyulal, L. (2001). *Bipolar disorder: A cognitive therapy approach.* Washington, DC: American Psychological Association.

Offidani, E., Fava, G. A., Tomba, E., & Baldessarini, R. J. (2013). Excessive mood elevation and behavioral activation with antidepressant treatment of juvenile depressive and anxiety disorders: A systematic review. *Psychotherapy and Psychosomatics*, *82*, 132–141. http://dx.doi.org/10.1159/000345316.

Offidani, E., Guidi, J., Tomba, E., & Fava, G. A. (2013). Efficacy and tolerability of benzodiazepines versus antidepressants in anxiety disorders: A systematic review and meta-analysis. *Psychotherapy and Psychosomatics*, *82*, 355–362. http://dx.doi.org/10.1159/000353198.

Otto, M. W., Simon, N. M., Wisniewski, S. R., Miklowitz, D. J., Kogan, J. N., Reilly-Harrington, N. A., et al. (2006). Prospective 12-month course of bipolar disorder in out-patients with and without comorbid anxiety disorders. *British Journal of Psychiatry*, *189*, 20–25. http://dx.doi.org/10.1192/bjp.bp.104.007773.

Pacchiarotti, I., Bond, D. J., Baldessarini, R. J., Nolen, W. A., Grunze, H., Licht, R. W., et al. (2013). The International Society for Bipolar Disorders (ISBD) Task Force Report on Antidepressant Use in Bipolar Disorders. *American Journal of Psychiatry*, *170*, 1249–1262. http://dx.doi.org/10.1176/appi.ajp.2013.13020185.

Parikh, S. V., Zaretsky, A., Beaulieu, S., Yatham, L. N., Young, L. T., Patelis-Siotis, I., et al. (2012). A randomized controlled trial of psychoeducation or cognitive-behavioral therapy in bipolar disorder: A Canadian Network for Mood and Anxiety treatments (CANMAT) study. *The Journal of Clinical Psychiatry*, *73*, 803–810. http://dx.doi.org/10.4088/JCP.11m07343.

Parker, G., Hadzi-Pavlovic, D., & Tully, L. (2006). Distinguishing bipolar and unipolar disorders: An isomer model. *Journal of Affective Disorders*, *96*, 67–73. http://dx.doi.org/10.1016/j.jad.2006.05.014.

Paulhan, I., & Bourgeois, M. (1998). *Stress and coping. Coping strategies in the face of adversity.* Paris: PUF.

Perich, T., Manicavasagar, V., Mitchell, P. B., Ball, J. R., & Hadzi-Pavlovic, D. (2013). A randomised controlled trial of mindfulness-based cognitive therapy for bipolar disorder. *Acta Psychiatrica Scandinavica*, *127*, 333–343. http://dx.doi.org/10.1111/acps.12033.

Perlis, R. H., Ostacher, M. J., Miklowitz, D. J., Hay, A., Nierenberg, A. A., Thase, M. E., et al. (2010). Clinical features associated with poor pharmacologic adherence in bipolar disorder: Results from the STEP-BD study. *Journal of Clinical Psychiatry*, *71*, 296–303. http://dx.doi.org/10.4088/JCP.09m05514yel.

Perlis, R. H., Ostacher, M. J., Patel, J. K., Marangell, L. B., Zhang, H., Wisniewski, S. R., et al. (2006). Predictors of recurrence in bipolar disorder: Primary outcomes from the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). *FOCUS: The Journal of Lifelong Learning in Psychiatry*, *4*, 553–561.

Peters, L., & Andrews, G. (1995). Procedural validity of the computerized version of the Composite International Diagnostic Interview (CIDI-AUTO) in the anxiety disorders. *Psychosomatic Medicine*, *25*, 1269–1280. http://dx.doi.org/10.1017/S0033291700033237.

Post, R. M., Altshuler, L. L., Leverich, G. S., Frye, M. A., Nolen, W. A., Kupka, R. W., et al. (2006). Mood switch in bipolar depression: Comparison of adjunctive venlafaxine, bupropion and sertraline. *British Journal of Psychiatry*, *189*, 124–131. http://dx.doi.org/10.1192/bjp.bp.105.013045.

Powell, B. J., Othmer, E., & Sinkhorn, C. H. (1977). Pharmacological aftercare for homogeneous groups of patients. *Hospital and Community Psychiatry*, *28*, 125–127.

Proudfoot, J., Parker, G., Manicavasagar, V., Hadzi-Pavlovic, D., Whitton, A., Nicholas, J., et al. (2012). Effects of adjunctive peer support on perceptions of illness control and understanding in an online psychoeducation program for bipolar disorder: A randomised controlled trial. *Journal of Affective Disorders*, *142*, 98–105 (0165-0327; 1573-2517).

Provencher, M. D., Hawke, L. D., & Thienot, E. (2011). Psychotherapies for comorbid anxiety in bipolar spectrum disorders. *Journal of Affective Disorders*, *133*, 371–380. http://dx.doi.org/10.1016/j.jad.2010.10.040.

Rakofsky, J. J., & Dunlop, B. W. (2011). Treating nonspecific anxiety and anxiety disorders in patients with bipolar disorder: A review. *The Journal of Clinical Psychiatry*, *72*, 81–90. http://dx.doi.org/10.4088/JCP.09r05815gre.

Reilly-Harrington, N. A., Deckersbach, T., Knauz, R., Wu, Y., Tran, T., Eidelman, P., et al. (2007). Cognitive behavioral therapy for rapid-cycling bipolar disorder: A pilot study. *Journal of Psychiatric Practice*, *13*, 291. http://dx.doi.org/10.1097/01.pra.0000290667.02484.3d.

Rickels, K. (2013). Should benzodiazepines be replaced by antidepressants in the treatment of anxiety disorders fact or fiction. *Psychotherapy and Psychosomatics*, *82*, 351–352. http://dx.doi.org/10.1159/000353502.

Rosenberg, S. D., Mueser, K. T., Jankowski, M. K., Salyers, M. P., & Acker, K. (2004). Cognitive-behavioral treatment of PTSD in severe mental illness: Results of a pilot study. *American Journal of Psychiatric Rehabilitation*, *7*, 171–186. http://dx.doi.org/10.1080/15487760490476200.

Ryff, C. D. (1989). Happiness is everything, or is it? Explorations on the meaning of psychological well-being. *Journal of Personality and Social Psychology*, *57*, 1069. http://dx.doi.org/10.1037/0022-3514.57.6.1069.

Satterfield, J. M. (1999). Adjunctive cognitive-behavioral therapy for rapid-cycling bipolar disorder: An empirical case study. *Psychiatry*, *62*, 357–369.

Scott, J., Leboyer, M., Hickie, I., Berk, M., Kapczinski, F., Frank, E., et al. (2013). Clinical staging in psychiatry: A cross-cutting model of diagnosis with heuristic and practical value. *The British Journal of Psychiatry*, *202*, 243–245. http://dx.doi.org/10.1192/bjp.bp.112.110858.

Scott, J., Paykel, E. S., Morriss, R., Kinderman, P., Johnson, T., Abbott, R., et al. (2006). Cognitive-behavioural therapy for severe and recurrent bipolar disorders: Randomised controlled trial. *British Journal of Psychiatry*, *188*, 313–320. http://dx.doi.org/10.1192/bjp.188.4.313.

Searson, R., Mansell, W., Lowens, I., & Tai, S. (2012). Think Effectively About Mood Swings (TEAMS): A case series of cognitive–behavioural therapy for bipolar disorders. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*, 770–779. http://dx.doi.org/10.1016/j.jbtep.2011.10.001.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse.* New York: Guildford Publications.

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., et al. (1998). The Mini-International Neuropsychiatric Interview (MINI): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, *59*, 22–33.

Simon, N. M., Otto, M. W., Wisniewski, S. R., Fossey, M., Sagduyu, K., Frank, E., et al. (2004). Anxiety disorder comorbidity in bipolar disorder patients: Data from the first 500 participants in the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). *American Journal of Psychiatry*, *161*, 2222–2229. http://dx.doi.org/10.1176/appi.ajp.161.12.2222.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *The state trait anxiety inventory.* Palo Alto, CA: Consulting Psychologist Press.

Spitzer, R. L., & Endicott, J. (1979). *Schedule for Affective Disorders and Schizophrenia, SADS.* New York, NY: New York State Psychiatric Institute, Department of Research Assessment and Training.

Stewart, R. E., & Chambless, D. L. (2009). Cognitive-behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*, *77*, 595. http://dx.doi.org/10.1037/a0016032.

Strawn, J. R., Adler, C. M., McNamara, R. K., Welge, J. A., Bitter, S. M., Mills, N. P., et al. (2013). Antidepressant tolerability in anxious and depressed youth at high risk for bipolar disorder: A prospective naturalistic treatment study. *Bipolar Disorders*, *16*, 523–530. http://dx.doi.org/10.1111/bdi.12113.

Szentagotai, A., & David, D. (2010). The efficacy of cognitive-behavioral therapy in bipolar disorder: A quantitative meta-analysis. *Journal of Clinical Psychiatry*, *71*, 66–72. http://dx.doi.org/10.4088/JCP.08r04559yel.

Thienot, E., Provencher, M. D., & St-Amand, J. (2013). Traitement du trouble d'anxiété généralisée chez des individus présentant un trouble bipolaire: un protocole à cas unique [Treatment of generalized anxiety disorder for patients with bipolar disorder: A single case experimental design.]. *Journal de Thérapie Comportementale et Cognitive*, *23*, 9–16. http://dx.doi.org/10.1016/j.jtcc.2012.11.003.

Tondo, L., Vázquez, G., & Baldessarini, R. J. (2010). Mania associated with antidepressant treatment: Comprehensive meta-analytic review. *Acta Psychiatrica Scandinavica*, *121*, 404–414. http://dx.doi.org/10.1111/j.1600-0447.2009.01514.x.

Totterdell, P., Kellett, S., & Mansell, W. (2012). Cognitive behavioural therapy for cyclothymia: Cognitive regulatory control as a mediator of mood change. *Behavioural and Cognitive Psychotherapy*, *40*, 412–424. http://dx.doi.org/10.1017/S1352465812000070.

Treuer, T., & Tohen, M. (2010). Predicting the course and outcome of bipolar disorder: A review. *European Psychiatry*, *25*, 328–333. http://dx.doi.org/10.1016/j.eurpsy.2009.11.012.

van der Ploeg, H. M. (1982). The development and validation of a Dutch inventory for the measurement of anxiety: "de Zelf-Beoordehngs Vragenhjst (STAY-DY)". *Tijdschrift voor Psychiatrie*, *24*, 576–588.

Van Gent, E. M. (2000). Une étude suivie de 3 ans de thérapies de groupe additives au traitement au lithium [Follow-up study of 3 years group therapy with lithium treatment]. *L'Encéphale*, *26*, 76.

Van Gent, E. M., Vida, S. L., & Zwart, F. M. (1988). Group therapy in addition to lithium therapy in patients with bipolar disorders. *Acta Psychiatrica Belgica*, *88*, 405.

Van Gent, E. M., & Zwart, F. M. (1993a). Five year follow-up after educational group therapy added to lithium prophylaxis: Five years after group added to lithium. *Depression*, *1*, 225–226. http://dx.doi.org/10.1002/depr.3050010406.

Van Gent, E. M., & Zwart, F. M. (1993b). Ultra-short versus short group therapy in addition to lithium. *Patient Education and Counselling*, *21*, 135–141. http://dx.doi.org/10.1016/0738-3991(93)90071-4.

Vázquez, G. H., Baldessarini, R. J., & Tondo, L. (2014). Co-ocurrence of anxiety and bipolar disorders: Clinical and therapeutic overview. *Depression and Anxiety*, *31*, 196–206. http://dx.doi.org/10.1002/da.22248.

Williams, J. M. G., Alatiq, Y., Crane, C., Barnhofer, T., Fennell, M. J. V., Duggan, D. S., et al. (2008). Mindfulness-based cognitive therapy (MBCT) in bipolar disorder: Preliminary evaluation of immediate effects on between-episode functioning. *Journal of Affective Disorders*, *107*, 275–279. http://dx.doi.org/10.1016/j.jad.2007.08.022.

World Health Organisation (1995). *CIDI-auto version 2.1. Administrator's guide.* Sydney: Training and Reference Centre for WHO CIDI.

Young, R. C., Biggs, J. T., Ziegler, V. E., & Meyer, D. A. (1978). A rating scale for mania: Reliability, validity and sensitivity. *British Journal of Psychiatry*, *133*, 429–435. http://dx.doi.org/10.1192/bjp.133.5.429.

Zwart, F. M., & Spooren, J. (1983). *Een Stemmingslijst voor Depressie [A mood list for depression].* Utrecht: Department of Psychiatry.