# The Psychology of Bipolar Disorders

Sara Tai

There is growing recognition of the importance of understanding bipolar disorder and its underlying mechanisms in psychological terms. This paper reviews the main advances in psychological understanding towards a more integrated biopsychosocial perspective.

Nearly everything written on bipolar disorder has begun by stating how it has long been a neglected area of research within psychopathology, largely conceptualised in biological terms with little understanding of psychological mechanisms. The last ten years has witnessed an increased interest in the disorder so that the picture has slowly been changing and following a surge of research activity within the last five years, this is certainly no longer the case. It would appear that there is a combination of factors; the limited efficacy of pharmacological interventions (Maj, 2003), a need to understand the mechanisms underlying the disorder more (Jones, Sellwood, & McGovern, 2005), and the success of psychological approaches within the field (Colom et al., 2003; Lam et al., 2003; Miklowitz, George, Richards, Simoneau, & Suddath, 2003), which has facilitated recognition of the importance of understanding bipolar disorder in psychological terms. Subsequently, an abundance of psychological research has begun to emerge and radically impact on the way we think about the development and maintenance of the disorder.

This paper begins by providing an overview of the phenomenology of bipolar disorder, followed by a consideration of the concept of a broader spectrum of bipolar disorders. The primary objective is to summarise some of the main advances in psychological understanding of bipolar disorders. Such developments have increased awareness of how the most meaningful way in which to understand this complex disorder is through a more integrated biopsychosocial perspective of the mechanisms involved. Finally this paper discusses how thinking within this field might be shaped within the future utilising an integrative perspective of current models of the disorder.

## Bipolar Disorder

Bipolar disorder involves periods of extreme disruptions to mood, behaviour and cognitive functioning. People with the disorder typically experience periods of severe depression, mania or hypomania, in addition to periods of relatively stable mood. However, even during periods of so-called remission' sufferers experience functional impairment (Fagiolini et al., 2005) and can still display mood swings and subclinical symptoms (Judd & Akiskal, 2003). The majority of sufferers tend to spend significantly more time depressed as opposed to manic (Post et al., 2003). Bipolar II (depression with hypomanic episodes), is the most common expression of bipolar disorder (Judd et al., 2003).

The World Health Organisation (WHO) has identified bipolar disorder as a serious public health problem constituting the sixth leading cause of disability-adjusted life years in the world among people aged 15–44 years (Woods, 2000). It is predicted that mood disorders will continue to be highly prevalent well into the year 2020 with depression as a leading cause of disability, second only to cardiovascular disease (Michaud, Murray, & Bloom, 2001).

Approximately 1–1.5 % of the general population in the US and UK meet the DSMIV criteria for the mental disorder (Bebbington, Bowen, Hirsch, & Kuipers, 1995; Kessler et al., 1994). It is estimated that many more people, around 5–10 % of the population, experience significant symptoms although

they are not severe enough to meet full diagnostic criteria (Akiskal et al., 2000). Diagnosis of the disorder is often delayed, and there is evidence that the age of onset could be much earlier than first thought, with childhood onset in many cases (Jones, Tai, Evershed, Knowles, & Bentall, 2006). Bipolar disorder is a lifelong mental health problem (Chen, Swann, & Johnson, 1998) with less than 1 % of sufferers experiencing an episode as a one off (Angst et al., 1973). Sufferers endure a recurring and relapsing course with numerous studies reporting around 50 % of patients relapsing within one year of recovery from an episode (Gitlin, Swendsen, Heller, & Hammen, 1995; Solomon, 1995). Post et al. (2003) examined the prevalence and persistence of mood episodes in 258 bipolar patients as assessed by clinicians over a twelve month period. Individuals spent 44 % of the year either depressed or manic, and 63 % of the sample experienced four or more episodes per year, despite being in receipt of appropriate psychotropic medication. Similar findings were obtained in a British sample of 204 people with bipolar disorder interviewed weekly for 18 months (Paykel, Abbott, Morris, Hayburst, & Scott, 2006).

Many people with mood disorders experience significant psychotic symptoms during mood episodes (Cassidy, Forrest, Murry, & Carroll, 1998; Cassidy, Murray, Forest, & Carroll, 1998). In a review of 26 studies, Goodwin and Jamison (1990) reported that 58 % of people with bipolar disorder had a lifetime history of at least one of a whole range of psychotic symptoms, such as hallucinations (Baethge et al., 2005), thought disorder (Tai, Haddock, & Bentall, 2004) and delusions (Selva et al., 2006); previously thought to exist only in schizophrenia.

Rates of substance abuse are high, with studies in psychiatric settings reporting rates of 18–75 % (Bernadt & Murray, 1986) and community studies 60–70 % (Cassidy, Ahearn, & Carroll, 2001; Regier et al., 1990). There is also evidence of increased substance abuse in people identified as having an increased risk of developing the disorder both through genetic risk (Jones et al., 2006) and trait markers of hypomania (Kaney, Kinderman, & Bentall, submitted). Maremmani, Perugi, Pacini, and Akiskal (2006) propose a unitary perspective of substance dependency and mood disorders, in which they view the mechanisms involved in bipolar disorders to be similar to those operating within substance abuse so that having bipolar disorder is likely to increase the chances of substance abuse.

Unsurprisingly, there is an elevated risk of attempting or completing suicide in people with bipolar disorder, mainly during phases associated with depression (Valtonen et al., in press). Jamison (2000) identifies bipolar disorder as the most common psychiatric disorder associated with suicide approximating that 25–50 % attempt suicide at least once. Leverich et al. (2003) found that 34 % of 648 bipolar patients had a lifetime history of suicide attempts.

Because of the persistent, recurrent and severe course of bipolar disorders, it has significant impact on family relationships (Speer, 1992; Targum, Dibble, Davenport, & Gershon, 1981) and the physical and psychological wellbeing of care-givers (Perlick et al., 2005). It is understandable therefore that people with bipolar disorder experience a higher rate of divorce and relationship breakdown (Stimmel, 2004), greater levels of social dysfunction even during periods of remission' and a lower quality of life (Vojta, Kinosian, Glick, Alshuer, & Bauer, 2001; Zhang, Wisniewski, Bauer, Sachs, & Thase, 2006). Occupational functioning is also disrupted (Gardner et al., 2006) costing the UK approximately £2 billion per annum (Das Gupta & Guest, 2002). Individuals struggle to find employment; Kupfer et al. (2002) found that despite 60 % of a sample of 2839 North Americans with bipolar disorder having been educated to college level, 64 % were unemployed. Similar unemployment levels have also been reported in the UK (Gareth Hill, Hardy, & Shepherd, 1996).

## Bipolar Spectrum Disorders

Most phenomenological research includes only those people who meet formal DSM-IV diagnostic criteria (American Psychiatric Association, 1994). Such approaches are founded upon the assumption that bipolar disorder is a discrete disorder that is distinguishable from other forms of affective disorder. However, there is an ever growing body of evidence that this is not the case and that the formal DSM IV diagnostic criteria only accounts for a small proportion of wide ranging symptoms that form the basis of a much more variable and broader spectrum of related disorders (Lara, Pinto, Akiskal, & Akiskal, 2006; Valenca et al., 2005). For example, Perugi (2004) estimates that 27 % to 62 % of all major depressions involve some degree of hypomania, and that these are related to premorbid cyclothymic or hyperthymic tem-peramental characteristics. The concept of a bipolar temperament or personality is not a new idea, dating back to Kretschmer (1925) and Wittman and Sheldon (1948). From this perspective, those affected with spectrum disorders is likely to be much greater and often with severe consequences (Dunner, 2003).

Evidence making the case for a spectrum of bipolar disorders (Angst, 2005) is plentiful. Akiskal et al. (2000) estimate that up to 6.4 % of the general population experience soft symptoms' of the condition. Also, there is a growing body of evidence that bipolar spectrum disorders overlap with other syndromes not previously considered to be affective disorders. 13–81 % of people diagnosed with borderline personality disorder (BPD) show bipolarity when using criteria for full mania to soft signs (Mackinnon & Pies, 2006), suggesting it is part of a spectrum in which the same underlying mechanisms could be shared (Deltito et al., 2001; George, Miklowitz, Richards, Simoneau, & Taylor, 2003). Benazzi (2005) found that people with bipolar disorder have more borderline traits than did those with major depression, and Schiavone, Dorz, Conforti, Scarso, and Borgherini (2004) found similar rates of BPD traits in a comparative study of 39 patients with bipolar disorder and 155 patients with unipolar depression. Further evidence for the bipolar spectrum comes from Nardi et al. (2005), who reported considerable overlap between symptoms of schizophrenia and bipolar disorder, coining the term "schizobipolar disorder". They claimed differences between this new "group" and conventionally understood schizophrenia in terms of prescribed drugs but similarities for age of onset, suicide attempts and family history of suicide attempts. Similar findings have also been reported in respect to anxiety disorders, making a case for these being incorporated within a spectrum (Valenca et al., 2005) . Akiskal et al. (in press) interviewed 107 people with bipolar II and found that 46.7 % met DSM criteria for panic disorder and agoraphobia. de Graaf, Bijil, Smit, Vollebergh, and Spijker (2002) report a prospective epidemiological study of 7076 adults aged 18–64, where they found that anxiety-comorbid mood disorder was the most prevalent comorbid disorder; more so than pure mood disorder. Similarly, Valenca et al. (2005) found that people with social anxiety compared to those with bipolar II had a similar number of previous depressive episodes, alcohol abuse, suicide attempts, and family history for mood disorder. Interestingly, they describe a subgroup of patients with social anxiety who, when treated with antidepressants, showed improvement that consisted of a presentation identical to a mild to moderate hypomanic state. Withdrawal of the antidepressants led to the return of the social anxiety symptoms. Masi et al. (2001) also report that in adoles-cents comorbid anxiety is common but often misdiagnosed as pharmacologic (hypo)mania.

Matza, Rajagopalan, Thompson, and de Lissovopy (2005) propose that comorbidity is a common feature of bipolar disorder, but often the reason for misdiagnosis resulting in expensive and ineffective treatments. They recommend that people with mood-related problems but also presenting comorbid

patterns of personality disorder, alcohol abuse, psychotic disorder, generalized anxiety disorder and panic should be screened for bipolar disorder.

Whether bipolar disorder is conceptualised as a specific category of symptoms or as a wider range of related syndromes that make up a spectrum of bipolar disorders, what we are describing is something where the common component would appear to be depression. 90 % of people who experience mania also have depressive episodes (Goodwin & Jamison, 1990). Cassidy and co-workers (Cassidy, Forest et al., 1998; Cassidy, Murray et al., 1998) found depression was the second most reported symptom in 72 % of their sample of people with bipolar disorder; irritability being the first.

It would appear that only a small minority of people experience unipolar mania and even in these cases there is evidence that if full histories are obtained, depressive episodes are apparent (Pfohl, Vasquez, & Nasrallah, 1982). Yazici et al. (2002) argue that many studies documenting unipolar mania have severe methodological limitations; some including people who have had no more than 1 or 2 episodes of mania, or for whom the follow-up time period has not been reported, making it unclear whether sufficient time has lapsed in which further depressive episodes might have occurred. What with uncertainty whether mixed episodes, secondary mania or anti-depressant induced mania are included in the criteria, small samples and the recruitment from multiple sites, reliability of these studies is problematic.

Depression in bipolar disorder appears to resemble unipolar depression, and numerous studies have examined the possible differences between the two. In comparison to unipolar depression bipolar depression is associated with poorer quality of life in terms of social, physical and emotional domains (Yatham et al., 2004), more behavioural symptoms such as hypersomnia, lethargy and apathy (Colom et al., 2003), more suicide attempts (Goodwin & Jamison, 1990), and greater reporting of the co-occurrence of symptoms such as sleep disturbance, loss of energy and anhedonia (Papadimitriou, Dikeos, Daskalopoulou, & Soldatos, 2002). These findings have not been reliably replicated and most studies report contradictory findings (Joffe, Young, & MacQueen, 1999). Hantouche and Akiskal (2005) argue that subtypes of bipolar disorder (I and II) must be considered separately in order to make such distinctions; they define bipolar II depression as having more hypomanic or mixed symptoms than unipolar depression. This adds support to the case for no real difference between the actual depressive component of bipolar and unipolar presentations, an idea that is supported by a wealth of evidence (Benazzi, 1999; Dorz, Borgherini, Conforti, Scarso, & Magni, 2003; Kuhs & Reschke, 1992; Wetzler, Khadivi, & Oppenheim, 1995).

Bipolar disorder is perhaps best conceptualised as depression with (hypo)mania in addition. However, mania and hypomania are complicated and dynamic phenomena, composed of a multitude of dimensions susceptible to change and fluctuation over time (Cassidy, Forest et al., 1998; Cassidy, Murray, et al., 1998; Hantouche, Angst, & Akiskal, 2003). The underlying mechanisms are difficult to explain. The common denominator is the affective components which the main theories of bipolar disorder focus on and attempt to explain.

## BIS/BAS – Reward System

The Behavioural Activation hypothesis of (hypo)mania is based on a model that was independently formulated by Depue (Depue et al.,1981; Depue & Iacano, 1989; Depue, Collins, & Luciana, 1996) and Gray (Gray, 1972, 1987, 1994). The hypothesis states the existence of two neural motivational systems (Behavioural Activation (BAS) and Inhibition Systems (BIS) that regulate approach and withdrawal behaviour.

BAS controls appetitive motivation and moderates approach behaviour in response to environmental cues of reward through dopaminergic activity within the mesolimbic system (Depue & Collins, 1999; Gray, 1994). Meyer and Hautzinger (2001) describe how BAS generates approach related affect in the presence of an incentive stimulus, triggering positive affect, such as hope and excitement when a person is confronted with an incentive stimulus. They conceptualise BAS as essential mechanism for enhancing the chances of actualising incentive acquisition. However, if BAS activation is excessive, it causes increased motor activity, heightened reward responsiveness and related heightened emotions.

BIS is hypothesised as an opposing system that governs the withdrawal of behaviours related to goal attainment and motivation of activity that could result in punishment or adverse consequences. It is sensitive to cues of threat and punishment and activates corresponding threat and avoidance through noradrenergic and serotonergic activity within the septohippocampal system (Depue & Iacano, 1989; Gray, 1982, 1994). This system is responsible for generating behavioural inhibition and associated negative affect, including emotions such as anxiety, fear, disgust and embarrassment.

Of the two systems, it is BAS which is considered to be the essential mechanism for developing bipolar disorders. From this perspective, mania and depression are assumed to be opposites of only one dimension; behavioural activation. Depue and Zald (1993) have further hypothesised a behavioural facilitation system of which regulatory strength' is seen to control BAS reactivity. Carver and White (1994) propose that BAS strength is a stable characteristic which can be measured, for which purpose they devised the BIS/BAS scales.

Analogue studies have demonstrated that BAS strength correlates with mania and depression (Meyer, Johnson, & Winters, 2001).

Considerable evidence for the theory of behavioural activation has come from studies examining the effect of goal-attainment life events on people with bipolar disorder. As predicted, these events believed to trigger the BAS do indeed seem to cause increases in manic symptoms (Johnson, 2005; Johnson, Winett, Meyer, Greenhouse, & Miller, 1999; Johnson et al., 2000; Lozano & Johnson, 2001). Similarly, it is negative life events which seem to play a greater role in depressive symptomatology as opposed to (hypo)manic symptoms (Alloy et al., 2006). Further work examining the role of dysfunctional beliefs relating to extreme goal-attainment, perfectionism and need for approval may have suggested a possible interaction with these life events, increasing risk of an episode (Johnston, Ruggero, & Carver, 2005; Lam, Wright, & Smit, 2004).

The relevance of goal attainment life events in bipolar disorder is fairly well established within the literature and forms the main evidence base upon which the application of the BAS theory is founded. However, it does not provide substantive support for whether an actual biological system exists. Some of the earliest evidence of a behavioural activation system was provided by Rosenthal et al. (1984), whose study of 29 people with seasonal bipolar affective disorder demonstrated that behavioural engagement appears to correspond to levels of light. This work was also supported by Krauss, Depue, Arbisi, and Spoont (1992) and Allen, Iaconi, Depue, and Arbisi (1993). Other than this, BIS/BAS is largely hypothetical and there are a number of difficulties in applying this as a causal explanation of bipolar disorder. BAS alone is an over-simplistic explanation for a complex set of phenomena, and individual differences on BAS reactivity do not adequately explain the inter-individual differences observed for vulnerability to bipolar disorders (Holzwarth & Meyer, 2006). In addition, BAS reactivity cannot fully account for the mechanisms involved in the transition from a manic to a depressive phase and vice versa; if a depressed individual has low BAS, how does that explain a sudden change to mania?

## Depression Avoidance and Self Esteem

Bentall and colleagues (Bentall, 2003; Bentall, Tai, & Knowles, 2006; Lyon, Startup, & Bentall, 1999) have tested an early psychoanalytical hypothesis known as the "manic-defence hypothesis" (Abraham, 1911/1927) conceptualising (hypo)mania as a form of depression avoidance. The main premise is that opposed depression and mania are both essentially forms of depression but manifest themselves as behaviourally different response styles. People become manic as a response to trying to avoid depression (Bentall, 2003). Evidence that depression forms a core part of mania is used to support this hypothesis. In a longitudinal study of 20 people with bipolar disorder Kotin and Goodwin (1972) reported how depression ratings were sometimes higher during manic than depressive episodes and Lyon et al. (1999) demonstrated how manic participants showed depressive-type responding on some psychological tests. Furthermore, Winters and Neale (1985) demonstrated how people suffering from bipolar disorder have a pessimistic attributional style even during remission. Lyon et al. (1999) also presented supporting data demonstrating differences between implicit and explicit attributional style in manic people.

Central to the depression avoidance theory is the compelling evidence that self-esteem has a role within bipolar disorders in the form of abnormal beliefs about the self (Bentall, Kinderman, & Manson, 2005) and instability of beliefs about the self and affect (Knowles, Tai, Jones, Morriss, & Bentall, in press; Kaney, Kinderman, & Bentall, submitted; Jones et al., 2006). Building upon the work of Kernis, Cornell, Sun, Berry, and Harlow (1993), Bentall and colleagues suggest that self-esteem instability is key (Bentall et al., in submission) and this could be due to latent negative self schemas, or dysfunctional attempts to regulate self esteem (Bentall et al., 2006).

There is also sound evidence of abnormal coping styles (Thomas, Knowles, Tai, & Bentall, in submission) where depressed and bipolar patients have a similar ruminative style of coping with depression (Knowles, Tai, Morriss, Jones, & Bentall, in press). However, people with bipolar disorders tend to employ the additional strategy of behavioural risktaking, which contributes to the specific symptoms of mania (Knowles, Tai, Christensen, & Bentall, 2005; Thomas et al., in press; Jones et al., 2006).

The main difficulty with this line of thinking is that although it provides strong evidence for self esteem and coping mechanisms playing a significant role in maintaining the disorder, it does not outline specific mechanisms through which individuals make transition from one phase to another. There is also mixed evidence for mania functioning as a form of depression avoidance.

## Cognitive Vulnerability – Appraisal of Affect

One of the other main areas of psychological research within bipolar disorder has been examining cognitive vulnerability utilising the traditional Beckian style of formulations of depression (Beck, 1967). Originally mania was formulated as the opposite cognitive style to depression (Leahy, 1988), consistent with the view of mania being the polar opposite of depression. Beck postulated that mania constituted a positive cognitive triad as opposed to a negative one. Unfortunately this early formulation does not account for dysphoria and irritability, which are core features of mania; neither does it translate well in light of mixed episodes. Despite the limitations of this model and the absence of sufficient clinical data to support it, there have been few attempts to develop alternatives. There are a number of studies investigating the role of cognitive vulnerability related to bipolar disorder, many indicating that cognitive style is of similar negative characteristics as found in unipolar depression. Numerous studies have found that perfectionism and need for approval from others was more specific

to bipolar disorders as well as poor social problem solving skills (Alloy & Abramson, 2002; Scott, Garland, & Moorhead, 2001; Scott, Stanton, Garland, & Ferrier, 2000). Lam et al. (2004) argue that cognitions around goal attainment and achievement are pertinent and are features considered to be consistent with high BAS sensitivity. These findings have been replicated by other researchers such as Abramson et al. (2005) and Francis-Raniere, Alloy, and Abramson (2006), who found that perfectionism, autonomy, self-criticism as well as performance and high self-standards were central themes in cognitive style. However, Alloy et al. (2006) highlight the need for further research clarifying the role of cognitive styles, summarising that to date findings have yielded mixed results depending on which phase of bipolar disorder an individual is in. Research outlining the role of cognitions in manic and mixed phases is less clear.

Jones (2001) has developed a cognitive framework that builds on the work of Healy and Williams (1989), which emphasised the role of circadian rhythms as a primary causal mechanism in mania. Although Jones's theory is an elaborate model integrating biological and cognitive systems, in essence it postulates that mania and depression are consequences of the way in which individuals interpret altered internal states or activity levels that come about in relation to disruption of social rhythms. In the development of mania, neuropsychological symptoms may be appraised in ways that are positive and self-dispositional as opposed to situational. For example, if a person experiences their thinking as faster than usual, this could be interpreted as a sign of increased intelligence or as evidence of special powers. Subsequently, changes to social rhythms that bring about altered internal states (i.e. changes related to affect or cognition) might lead to behaviours that disrupt rhythms even more. In addition, cognitive distortions about physical state can disrupt social routines necessary for recovery feeding a vicious cycle of escalating symptoms.

As causal models, cognitive theories do not account sufficiently for wide ranging symptoms observed within bipolar disorder. Although they illuminate the importance of stress diathesis approaches with a potential for more integrative biopsychosocial perspectives to bipolar disorder, in doing so there is a heavy emphasis on behavioural components like social coping mechanisms such as rumination and risk taking, disrupted daily rhythms and behavioural activation. Unfortunately there is insufficient specification on the exact nature in which cognitive mechanisms are implicated. Such accounts might justify a plausible pathway into mania, but again there is a lack of clarification on the way in which transition from one phase to another occurs or mixed phases.

In recognition of many of these limitations Mansell, Morrison, Reid, Lowens, and Tai (in submission) have recently developed a cognitive model aimed at understanding mood swings and bipolar disorders. The model hypothesises that an individual has multiple conflicting interpretations of the changes and fluctuations they experience in internal state. They have a tendency to appraise these changes by attaching extreme personal meaning to them; a process influenced by specific sets of beliefs about affect and its regulation and about the self and relations with others. The appraisals of internal state are the central explanatory factor in this model; it is this process which explains how attempts at affect regulation are disturbed through the exaggerated efforts to enhance or exert control over internal states, subsequently causing further internal state changes. Counterproductive attempts at control are classified as either ascent behaviours (increasing activation), or descent behaviours (decreasing activation). This vicious cycle becomes self perpetuating as the process can maintain or exacerbate symptoms producing an interaction that increases vulnerability to relapse. The model incorporates important metacognitive processes and is certainly not inconsistent with to a problem which is multi-faceted, dynamic and variable over time. The model is subject to ongoing investigation in order to establish empirically its utility.

## Conclusions

It would appear that very nature of bipolar disorder presents a confusing picture with some pertinent factors to be considered when interpreting the findings to date. Firstly, there are severe limitations in any attempts to map linear causal mechanisms to relapse. Secondly, fluctuation is likely to be of central importance; factors such as self esteem, behavioural activation and social coping mechanisms are unlikely to be low or high or functional versus dysfunctional; they are naturally fluctuating. Fluctuation is an essential process within normal emotional regulation, but the intensity and extremity is the key to understanding bipolar disorder. Lastly, assumption of bipolarity could simply be incorrect. The evidence available could well indicate that rather than depression and mania existing as polar opposites of the same dimension, they are two separate dimensions that are interrelated and, on occasions, co-dependent. Johnson et al. (in preparation) have recently completed a study investigating the way in which depression and mania covary over time. They argue that if a unidimensional model were plausible, mania and depression would be negatively correlated. However, their longitudinal study of symptom ratings from 236 people with bipolar I over forty weeks showed this not to be the case and depression and mania vary in an independent manner; some individual symptoms of mania might correlate with depression and vice versa, whereas other symptoms might function as opposites.

One possible direction for future research of bipolar disorder is to consider the variety of theories to date not as contradictory or competing, but consistent within a multi-factorial problem. As singular causative models each has limitations, but in conjunction they represent the variety of processes and mechanisms that operate within bipolar disorder. This model of conceptualising psychological processes is also applicable to other mental disorders (Kinderman & Tai, 2007).

It is plausible that numerous factors contribute to psychopathology not only in an additive way, but also through an interactive process. Therefore, at any one point in an individual's life, a person must experience the right combination, amount and interaction of various factors in order to experience an episode. It is possible for example that certain life events might have less or no bearing to an individual on one occasion; while, with the presence and interaction of key factors such as low self esteem and poor coping mechanisms, specific thinking styles are likely to occur which would heighten the probability of disturbed sleep, and activated BAS, so that mania could ensue. The iterative effects of feedback processes may further exacerbate and increase vulnerability to relapse.

Dr. Sara Tai
School of Psychological Sciences
Coupland I, University of Manchester
Oxford Road, Manchester M13 9PL
Email: sara.tai@manchester.ac.uk
Tel: +44 (0) 161 275 2595

**Sara Tai**

Dr. Sara Tai is a Lecturer in Clinical Psychology at the University of Manchester and also works in an adult Community Mental Health Team at Bolton, Salford and Trafford NHS Trust (UK). She completed her training at Manchester University in 2001. Her research areas cover bipolar disorders, psychological processes in psychotic symptoms, emotion and psychosis and thought disorder.

**Sara Tai**

**Key publications**

Jones, S. H., Tai, S., Evershed, K., Knowles, R., & Bentall, R. (2006). Early detection of bipolar disorder: a pilot familial high-risk study of parents with bipolar disorder and their adolescent children. *Bipolar Disorder*, *8,* 362–372.

Kinderman, P., & Tai, S. (2007). Empirically groun-ded clinical interventions. Clinical implications of a psychological model of mental disorder. *Behaviou-ral and Cognitive Psychotherapy, 35,* 1–14.

Knowles, R., Tai, S., Morriss, R., Jones, S., & Bentall, R. P. (In press). Stability of self-esteem in bipolar disorder: Comparison of remitted bipolar patients, remitted unipolar patients and healthy controls. *Bipolar Disorders*.

Tai, S., Haddock, G., & Bentall, R. P. (2004). The effects of emotional salience on thought disorder in patients with bipolar affective disorder. *Psychological Medicine, 34*, 803–809.

# Referanser

References

Abraham, K. (1911/1927). Notes on the psychoanalytic investigation and treatment of manic depressive insanity. In E. Jones (Ed.), *Selected papers of Karl Abraham*. London: Hogarth.

Abramson, L. Y., Alloy, L. B., Walshaw, P. D. et al. (2005). Depressive cognitive styles in bipolar spectrum and normal comparison individuals. *Manuscript in preparation*.

Akiskal, H. S., Akiskal, K. K., Perugi, G., Toni, C., Ruffolo, G., & Tusini, G. (In Press). Bipolar II and anxious reactive "comorbidity": Toward better phenotypic characterization suitable for genotyping. *Journal of Affective Disorders*.

Akiskal, H. S., Bourgeois, M. L., Angst, J., Post, R., Moller, H.-J., & Hirschfeld, R. (2000). Re-evaluating the prevalence of and diagnostic composition within the broad clinical spectrum of bipolar disorders. *Journal of Affective Disorders, 59*, S5–S30.

Allen, J. J., Iacono, W. G., Depue, R. A., & Arbisi, P. (1993). Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biological Psychiatry, 33*(8–9), 642–646.

Alloy, L. B., Abramson, L. Y., Neeren, A. M., Walshaw, P. D., Urosevic, S., & Nusslock, R. (2006). Psychosocial risk factors for bipolar disorder: Current and early environment and cognitive styles. In S. H. Jones & R. Bentall (Eds.), *The psychology of bipolar disorder: New developments and research strategies* (pp. 11–39). Oxford: Oxford University Press.

American Psychiatric Association. (1994). *Diagnostic and statistical manual for mental disorders* (4th edition). Washington, DC: Author.

Angst, J. (2005). The mood spectrum: Improving the diagnosis of bipolar disorder. *Bipolar Disorders, 7*, 4–12

Angst, J., Baastrup, P., Grof, P., Hippius, H., Poldinger, W., & Weis, P. (1973). The course of monopolar depression and bipolar psychoses. *Psychiatria, Neurologia, Neurochirurgia*, *76*, 489–500.

Baethge, C., Baldessarini, R. J., Freudenthal, K., Streeruwitz, A., Bauer, M., & Bshor, T. (2005). Hallucinations in bipolar disorder: Characteristics and comparison to unipolar depression and schizophrenia. *Bipolar Disorders, 7,* 136–145).

Bebbington, P. E., Bowen, J., Hirsch, S. R., & Kuipers, E. A. (1995). Schizophrenia and psychosocial stresses. In S. R. Hirsch & D. R. Weinberger (Eds.), *Schizophrenia* (pp. 587–604). Oxford: Blackwell.

Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects.* New York: Harper and Row.

Benazzi, F. (1999). Bipolar versus psychotic outpatient depression. *Journal of Affective Disorders, 55*, 63–66.

Benazzi, F. (2005). Unipolar depression with racing thoughts: a bipolar spectrum disorder? *Psychiatry and Clinical Neurosciences, 59*(5), 570–575.

Benazzi, F., & Akiskal, H. S. (2003). The dual factor structure of self-rated MDQ hypomania: energized-activity versus irritable-thought racing. *Journal of Affective Disorders, 73*(1–2), 59–64.

Bentall, R. P. (2003). *Madness explained: Psychosis and human nature.* London: Penguin.

Bentall, R. P., Kinderman, P., & Manson, K. (2005). Self-discrepancies in bipolar-affective disorder. *British Journal of Clinical Psychology, 44*, 457–474.

Bentall, R. P., Smith, A., Knowles, R., Jones, S. H., Smith, T., & Tai, S. T. (In submission). Hypomanic personality, stability of self-esteem and response styles to negative mood.

Bentall, R. P., Tai, S. J., & Knowles, R. (2006). Psychological processes and the pathways to mania: exploring he manic defence hypothesis. In S. H. Jones & R. P. Bentall (Eds.), *The psychology of bipolar disorder: New developments and research strategies* (pp. 117–137). Oxford: Oxford University Press.

Bernadt, M. W., & Murray, R. M. (1986). Psychiatric disorder, drinking and alcoholism: what are the links? *British Journal of Psychiatry, 148*, 393–400.

Carver, C., & White, T. (1994). Behavioural inhibition, behavioural activation and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personal and Social Psychology, 67*, 319–333.

Cassidy, F., Ahearn, E. P., & Carroll, B. J. (2001). Substance abuse in bipolar disorder. *Bipolar Disorders, 3*, 181–188.

Cassidy, F., Forest, K., Murry, M., & Carroll, B. J. (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry, 55*, 27–32.

Cassidy, F., Murray, E., Forest, K., & Carroll, B. J. (1998). Signs and symptoms of mania in pure and mixed episodes. *Journal of Affective Disorders, 50*, 187–201.

Chen, Y. R., Swann, A. C., & Johnson, B. A. (1998). Stability of diagnosis in bipolar disorder. *Journal of Nervous and Mental Diseases, 186*, 17–23.

Colom, F., Vieta, E., Martinez-Aran, A., Reinares, M., Goikolea, J. M., Benabarre, A., et al. (2003). A randomized trial on the efficacy of group psychoeducation in the prophylaxis of recurrences in bipolar patients whose disease is in remission. *Archives of General Psychiatry, 60*, 402–407.

Das Gupta, R., & Guest, J. F. (2002). Annual cost of bipolar disorder. *British Journal of Psychiatry, 180*, 227–233.

de Graaf, R., Bijl, R. V., Smit, F., Vollebergh, W. A. M., & Spijker, J. (2002). Risk factors for 12-month comorbidity of mood, anxiety, and substance use Disorders: Findings from the Netherlands Mental Health Survey and Incidence Study. *American Journal of Psychiatry, 159*, 620–629.

Deltito, J., Martin, L., Riefkohl, J., Austria, B., Kissilenko, A., & Corless, C. M. P. (2001). Do patients with borderline personality disorder belong to the bipolar spectrum? *Journal of Affective Disorders, 67*, 221–228.

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion. *The Behavioral and Brain Sciences, 22*(3), 491–517; discussion 518–469.

Depue, R. A., Collins, P. F., & Luciana, M. (1996). A model of neurobiology: Environment interaction in developmental psychopathology. In M. F. Lenzenweger & J. J. Haugaard (Eds.), *Frontiers of developmental psychopathology* (pp. 44–76). New York: Oxford University Press.

Depue, R. A., & Iacano, W. G. (1989). Neurobehavioural aspects of affective disorders. *Annual Review of Psychology, 40*, 457–492.

Depue, R. A., Slater, J. F., Wolfstetter-Kausch, H., Klein, D., Goplerud, E., & Farr, D. (1981). A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: A conceptual framework and five validation studies. *Journal of Abnormal Psychology, 90*, 381–437.

Depue, R. A., & Zald, D. H. (1993). Biological and environmental processes in nonpsychotic psychopathology: A neurobehavioral perspective. In G. Costello (Ed.), *Basic issues in psychopathology* (pp. 127–237). New York: Guilford.

Dorz, S., Borgherini, G., Conforti, D., Scarso, C., & Magni, G. (2003). Depression in inpatients: bipolar vs unipolar. *Psychological Reports, 92*(3 Pt 1), 1031–1039.

Dunner, D. L. (2003). Clinical consequences of under-recognized bipolar spectrum disorder. *Bipolar Disorders, 5*, 456–463.

Fagiolini, A., Kupfer, D. J., Masalehdan, A., Scott, J. A., Houck, P. R., & Frank, E. (2005). Functional impairment in the remission phase of bipolar disorder. *Bipolar Disorders, 7*, 281–285.

Francis-Raniere, E. L., Alloy, L. B., & Abramson, L. Y. (2006). Depressive personality styles and bipolar spectrum disorders: prospective tests of the event congruency hypothesis. *Bipolar Disorders, 8*, 382–399.

Gardner, H. H., Kleinman, N. L., Brook, R. A., Rajagopalan, K., Brizee, T. J., & Smeeding, J. E. (2006). The economic impact of bipolar disorder in an employed population from an employer perspective. *Journal of Clinical Psychiatry, 67*, 1209–1218.

Gareth Hill, R., Hardy, P., & Shepherd, G. (1996). *Perspective on manic depression: A survey of the Manic Depressive Fellowship*. London: Sainsbury Centre for Public Health.

George, E. L., Miklowitz, D. J., Richards, J. A., Simoneau, T. L., & Taylor, D. O. (2003). The comorbidity of bipolar disorder and axis II personality disorders: prevalence and clinical correlates. *Bipolar Disorders, 5*, 115–122.

Gitlin, M. J., Swendsen, J., Heller, T. L., & Hammen, C. (1995). Relapse and impairment in bipolar disorder. *American Journal of Psychiatry, 152*, 1635–1640.

Goodwin, F. K., & Jamison, K. R. (1990). *Manic-depressive illness*. Oxford: Oxford University Press.

Gray, J. (1994). Three fundamental emotion systems. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 243–247). Oxford: Oxford University Press.

Gray, J. A. (1972). The psychophysiological basis of introversion-extraversion: A modification of Eysenck's theory. In V. D. Nebylitsyn & J. A. Gray (Eds.), *The biological bases of individual behaviour* (pp. 182–205). San Diego: CA: Academic Press.

Gray, J. A. (1981). A critique of Eysenck's theory of personality. In H. J. Eysenck (Ed.), *A model for personality* (pp. 246–276). Berlin: Springer-Verlag.

Gray, J. A. (1982). *The neuropsychology of anxiety*. Oxford: Oxford University Press.

Gray, J. A. (1987). Perspectives on anxiety and impulsivity: A commentary. *Journal of Research in Personality, 21*, 493–509.

Hantouche, E. G., & Akiskal, H. S. (2005). Bipolar II vs. unipolar depression: psychopathologic differentiation by dimensional measures. *Journal of Affective Disorders, 84*(2–3), 127–132.

Hantouche, E. G., Angst, J., & Akiskal, H. S. (2003). Factor structure of hypomania: interrelationships with cyclothymia and the soft bipolar spectrum. *Journal of Affective Disorders, 73*(1–2), 39–47.

Healy, D., & Williams, J. M. (1989). Moods, misattributions and mania: an interaction of biological and psychological factors in the pathogenesis of mania. *Psychiatric Developments, 7*(1), 49–70.

Holzwarth, K., & Meyer, T. D. (2006). The dysregulation of the "Behavioural Activation System": An independent dimension. *Personality and Individual Differences, 41*, 319–328.

Jamison, K. R. (2000). Suicide and bipolar disorder. *Journal of Clinical Psychiatry, 61 Suppl 9*, 47–51.

Joffe, R. T., Young, L. T., & MacQueen, G. M. (1999). A two-illness model of bipolar disorder. *Bipolar Disorders, 1*, 25–30.

Johnson, S. L. (2005). Mania and dysregulation in goal pursuit: a review. *Clinical Psychological Review, 25*(2), 241–262.

Johnson, S. L., Morriss, R., Scott, J., Kinderman, P., Paykel, E., & Bentall, R. (In preparation). Testing the bipolar model of depression and mania; understanding how depression and mania covary over time.

Johnson, S. L., Sandrow, D., Meyer, B., Winters, R., Miller, I., Solomon, D., et al. (2000). Increases in manic symptoms after life events involving goal attainment. *Journal of Abnormal Psychology, 109*, 721–727.

Johnson, S. L., Winett, C. A., Meyer, B., Greenhouse, W. J., & Miller, I. (1999). Social support and the course of bipolar disorder. *Journal of Abnormal Psychology, 108*, 558–566.

Johnston, S., Ruggero, C., & Carver, C. (2005). Cognitive, behavioral, and affective responses to reward: Links with hypomanic symptoms. *Journal of Social and Clinical Psychology 24*, 894–906.

Jones, S. H. (2001). Circadian rhythms, multilevel models of emotion and bipolar disorder – an initial step towards integration? *Clinical Psychology Review, 21*, 1193–1209.

Jones, S. H., Sellwood, W., & McGovern, J. (2005). Psychological therapies for bipolar disorder: the role of model-driven approaches to therapy integration. *Bipolar Disorders, 7*, 22–32.

Jones, S. H., Tai, S., Evershed, K., Knowles, R., & Bentall, R. (2006). Early detection of bipolar disorder: a pilot familial high-risk study of parents with bipolar disorder and their adolescent children. *Bipolar Disorders, 8*, 362–372.

Judd, L. L., & Akiskal, H. S. (2003). Depressive episodes and symptoms dominate the longitudinal course of bipolar disorder. *Current Psychiatry Reports, 5*(6), 417–418.

Judd, L. L., Schettler, P. J., Akiskal, H. S., Maser, J., Coryell, W., Solomon, D., et al. (2003). Long-term symptomatic status of bipolar I vs. bipolar II disorders. *The International Journal of Neuropsychopharmacology, 6*(2), 127–137.

Kaney, S., Kinderman, P., & Bentall, R. P. (submitted). Attributional lability in paranoia and depression: Changes in internality following exposure to a mild failure experience.

Kernis, M. H., Cornell, D. P., Sun, C. R., Berry, A., & Harlow, T. (1993). There's more to self-esteem than whether it is high or low: The importance of stability of self-esteem. *Journal of Personality and Social Psychology, 65,* 1190–1204.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., et al. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry, 51*, 8–19.

Kinderman, P., & Tai, S. (2007). Empirically grounded clinical interventions. Clinical implications of a psychological model of mental disorder. *Behavioural and Cognitive Psychotherapy, 35, 1–14.*

Knowles, R., Tai, S., Christensen, I., & Bentall, R. (2005). Coping with depression and vulnerability to mania: a factor analytic study of the Nolen-Hoeksema (1991) Response Styles Questionnaire. *British Journal of Clinical Psychology, 44*, 99–112.

Knowles, R., Tai, S., Jones, S., Morriss, R., & Bentall, R. P. (in submission). Vulnerability to bipolar symptoms: Screening using the Hypomanic Personality Questionnaire and Dysfunctional Attitudes Scale.

Knowles, R., Tai, S., Morriss, R., Jones, S., & Bentall, R. P. (In press). Stability of self-esteem in bipolar disorder: Comparison of remitted bipolar patients, remitted unipolar patients and healthy controls.

Kotin, J., & Goodwin, F. K. (1972). Depression during mania: clinical observations and theoretical implications. *American Journal of Psychiatry, 129*, 679–686.

Krauss, S. S., Depue, R. A., Arbisi, P. A., & Spoont, M. (1992). Behavioral engagement level, variability, and diurnal rhythm as a function of bright light in bipolar II seasonal affective disorder: An exploratory study. *Psychiatry Research, 43*(2), 147–160.

Kretschmer, E. (1925). *Physique and character* (W. J. H. Sprott, Trans.). London: Kegan, Trench and Trubner.

Kuhs, H., & Reschke, D. (1992). Psychomotor activity in unipolar and bipolar depressive patients. *Psychopathology, 25*(2), 109–116.

Kupfer, D. J., Frank, E., Grochocinski, V. J., Cluss, P. A., Houck, P. R., & Stapf, D. A. (2002). Demographic and clinical characteristics of individuals in a bipolar disorder case registry. *Journal of Clinical Psychiatry, 63*(2), 120–125.

Lam, D., Wright, K., & Smith, N. (2004). Dysfunctional assumptions in bipolar disorder. *Journal of Affective Disorders, 79*(1–3), 193–199.

Lam, D. H., Watkins, E. R., P. Hayward, J., Bright, J., Wright, K., Kerr, N., et al. (2003). A randomized controlled study of cognitive therapy for relapse prevention for bipolar affective disorder: outcome of the first year. *Archives of General Psychiatry, 60*, 145–152.

Lara, D. R., Pinto, O., Akiskal, K., & Akiskal, H. S. (2006). Toward an integrative model of the spectrum of mood, behavioral and personality disorders based on fear and anger traits: I. Clinical implications. *Journal of Affective Disorders, 94*(1–3), 67–87.

Leahy, R. L., & Beck, A. T. (1988). Cognitive therapy of depression and mania. In R. Cancro & A. Georgotas (Eds.), *Depression and mania* (pp. 517–537). New York: Elsevier.

Leverich, G. S., Altshuler, L. L., Frye, M. A., Suppes, T., Keck, P. E., Jr., McElroy, S. L., et al. (2003). Factors associated with suicide attempts in 648 patients with bipolar disorder in the Stanley Foundation Bipolar Network. *Journal of Clinical Psychiatry, 64*, 506–515.

Lozano, B. E., & Johnson, S. L. (2001). Can personality traits predict increases in manic and depressive symptoms? *Journal of Affective Disorders, 63*(1–3), 103–111.

Lyon, H., Startup, M., & Bentall, R. P. (1999). Social cognition and the manic defense. *Journal of Abnormal Psychology, 108*, 273–282.

Mackinnon, D. F., & Pies, R. (2006). Affective instability as rapid cycling: theoretical and clinical implications for borderline personality and bipolar spectrum disorders. *Bipolar Disorders, 8*, 1–14.

Maj, M. (2003). The effect of lithium in bipolar disorder: a review of recent research evidence. *Bipolar Disorders, 5*, 180–188.

Mansell, W., Morrison, A. P., Reid, G., Lowens, I., & Tai, S. (In submission) is now in press (Behaviour and Cognitive Psychotherapy. The interpretation of and responses to changes in internal states in bipolar disorder: An integrative cognitive model.

Maremmani, I., Perugi, G., Pacini, M., & Akiskal, H. S. (2006). Toward a unitary perspective on the bipolar spectrum and substance abuse: Opiate addiction as a paradigm. *Journal of Affective Disorders, 93*(1–3), 1–12.

Masi, G., Toni, C., Perugi, G., Mucci, M., Millepiedi, S., & Akiskal, H. S. (2001). Anxiety disorders in children and adolescents with bipolar disorder: A neglected comorbidity. *Canadian Journal of Psychiatry, 46*, 797–802.

Matza, L. S., Rajagopalan, K. S., Thompson, C. L., & de Lissovoy, G. (2005). Misdiagnosed patients with bipolar disorder: comorbidities, treatment patterns, and direct treatment costs. *Journal of Clinical Psychiatry, 66*, 1432–1440.

Meyer, B., Johnson, S. L., & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of Psychopatho-logy and Behavioral Assessment, 23*, 133–143.

Meyer, T. D., & Hautzinger, M. (2001). Hypomanic personality, social anhedonia and impulsive nonconformity: Evidence for familial aggregation? *Journal of Personality Disorders, 15*, 281–299.

Michaud, C. M., Murray, C. J., & Bloom, B. R. (2001). Burden of disease –implications for future research. *The Journal of the American Medical Association, 285*, 535–539.

Miklowitz, D. J., George, E. L., Richards, J. A., Simoneau, T. L., & Suddath, R. L. (2003). A randomized study of family-focused psychoedu-cation and pharmacotherapy in the outpatient management of bipolar disorder. *Archives of General Psychiatry, 60*, 904–912.

Nardi, A. E., Nascimento, I., Freire, R. C., de-Melo-Neto, Valfrido L., Valenca, M, A., et al. (2005). Demographic and clinical features of schizoaffective (schizobipolar) disorder – A 5-year retrospective study. Support for a bipolar spectrum disorder. *Journal of Affective Disorders , 86*(1), 11–18.

Papadimitriou, G. N., Dikeos, D. G., Daskalopoulou, E. G., & Soldatos, C. R. (2002). Co-occurrence of disturbed sleep and appetite loss differentiates between unipolar and bipolar depressive episodes. *Progress in Neuro-psychopharmacology & Biological Psychiatry, 26*, 1041–1045.

Paykel, E. S., Abbott, R., Morriss, R., Hayhurst, H., & Scott, J. (2006). Sub-syndromal and syndromal symptoms in the longitudinal course of bipolar disorder. *British Journal of Psychiatry, 189*, 118–123.

Perlick, D. A., Hohenstein, J. M., Clarkin, J. F., Kaczynski, R., & Rosenheck, R. A. (2005). Use of mental health and primary care services by caregivers of patients with bipolar disorder: A preliminary study. *Bipolar Disorders, 7*, 126–135.

Perugi, G. (2004). Towards a new definition of the new bipolar spectrum disorders. *Clinical Neuropsychiatry: Journal of Treatment Evaluation, 1*, 155–158.

Pfohl, B., Vasquez, N., & Nasrallah, H. (1982). Unipolar vs. bipolar mania: a review of 247 patients. *British Journal of Psychiatry, 141*, 453–458.

Post, R. M., Denicoff, K. D., Leverich, G. S., Altshuler, L. L., Frye, M. A., Suppes, T. M., et al. (2003). Morbidity in 258 bipolar outpatients followed for 1 year with daily prospective ratings on the NIMH life chart method. *Journal of Clinical Psychiatry, 64*, 680–690; quiz 738–689.

Regier, D. A., Farmer, M. E., Rae, D. S., Locke, B. Z., Keith, S. J., Judd, L. L., et al. (1990). Comorbidity of mental disorders with alcohol and other drug abuse. Results from the Epidemiologic Catchment Area (ECA) Study. *Jama, 264*, 2511–2518.

Rosenthal, N. E., Sack, D. A., Gillin, J. C., Lewy, A. J., Goodwin, F. K., Davenport, Y., et al. (1984). Seasonal affective disorder. A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry, 41*, 72–80.

Schiavone, P., Dorz, S., Conforti, D., Scarso, C., & Borgherini, G. (2004). Comorbidity of DSM-IV Personality Disorders in unipolar and bipolar affective disorders: a comparative study. *Psychological Reports, 95*(1), 121–128.

Scott, J., Garland, A., & Moorhead, S. (2001). A pilot study of cognitive therapy in bipolar disorder. *Psychological Medicine, 31*, 459–467.

Scott, J., Stanton, B., Garland, A., & Ferrier, I. N. (2000). Cognitive vulnerability in patients with bipolar disorder. *Psychological Medicine, 30*, 467–472.

Selva, G., Salazar, J., Balanza-Martinez, V., Martinez-Aran, A., Rubio, C., Daban, C., et al. (2007). Bipolar I patients with and without a history of psychotic symptoms: Do they differ in their cognitive functioning? *Journal of Psychiatric Research*, 41(3–4), 265–272.

Solomon, D. A., Keitner, G. I., Miller, I. W., Shea, M. T., & Keller, M. B. (1995). Course of illness and maintenance treatments for patients with bipolar disorder. *Journal of Clinical Psychiatry, 56*, 5–13.

Speer, D. C. (1992). Differences in social resources and treatment history among diagnostic groups of older adults. *Hospital and Community Psychiatry, 43*, 270–274.

Stimmel, G. L. (2004). Economic grand rounds: the economic burden of bipolar disorder. *Psychiatric Services, 55*, 117–118.

Tai, S., Haddock, G., & Bentall, R. P. (2004). The effects of emotional salience on thought disorder in patients with bipolar affective disorder. *Psychological Medicine, 34*, 803–809.

Targum, S. D., Dibble, E. D., Davenport, Y. B., & Gershon, E. S. (1981). The Family Attitudes Questionnaire. Patients' and spouses' views of bipolar illness. *Archives of General Psychiatry, 38*, 562–568.

Thomas, J., Knowles, R., Tai, S., & Bentall, R. P. (in press). Response styles and depressive schemas in bipolar disorder. *Journal of Affective Disorders*.

Valenca, A. M., Nardi, A. E., Nascimento, I., Lopes, F. L., Freire, R. C., Mezzasalma, M. A., et al. (2005). Do social anxiety disorder patients belong to a bipolar spectrum subgroup? *Journal of Affective Disorders, 86*(1), 11–18.

Valtonen, H. M., Suominen, K., Mantere, O., Leppamaki, S., Arvilommi, P., & Isometsa, E. (In press). Suicidal behaviour during different phases of bipolar disorder. *Journal of Affective Disorders*.

Vojta, C., Kinosian, B., Glick, H., Altshuler, L., & Bauer, M. S. (2001). Self-reported quality of life across mood states in bipolar disorder. *Comprehensive Psychiatry, 42*(3), 190–195.

Wetzler, S., Khadivi, A., & Oppenheim, S. (1995). The psychological assessment of depression: Unipolars versus bipolars. *Journal of Personality Assessment, 65*, 557–566.

Winters, K. C., & Neale, J. M. (1985). Mania and low self-esteem. *Journal of Abnormal Psychology, 94*, 282–290.

Wittman, P., & Sheldon, W. (1948). A proposed classification of psychotic disorder reactions. *American Journal of Psychiatry, 105*, 124–128.

Woods, S. W. (2000). The economic burden of bipolar disease. *Journal of Clinical Psychiatry, 61*(Suppl 13), 38–41.

Yatham, L. N., Lecrubier, Y., Fieve, R. R., Davis, K. H., Harris, S. D., & Krishnan, A. A. (2004). Quality of life in patients with bipolar I depression: data from 920 patients. *Bipolar Disorders, 6*, 379–385.

Yazici, O., Kora, K., Ucok, A., Saylan, M., Ozdemir, O., Kiziltan, E., et al. (2002). Unipolar mania: a distinct disorder? *Journal of Affective Disorders, 71*(1–3), 97–103.

Zhang, H., Wisniewski, S. R., Bauer, M. S., Sachs, G. S., & Thase, M. E. (2006). Comparisons of perceived quality of life across clinical states in bipolar disorder: Data from the first 2000 Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) participants. *Comprehensive Psychiatry, 47*(3), 161–168.

There is growing recognition of the importance of understanding bipolar disorder and its underlying mechanisms in psychological terms. This paper reviews the advances in psychological understanding of bipolar disorders and discusses the emerging empirical evidence for the development and maintenance of the disorder; including hypotheses on behavioural activation, depression avoidance,

self esteem instability and cognitive vulnerabilities. This paper also provides an overview of the phenomenology of bipolar disorder and considers the utility of conceptualising affective disorders as a broader spectrum of bipolar disorders. It is suggested that the most meaningful way in which to understand bipolar disorders is through a more integrated biopsychosocial perspective specifying the mechanisms through which multiple factors can impinge on psychological processes.

Keywords: bipolar disorders, psychological processes, behavioural activation system, cognitive vulnerability, depression avoidance

Published: 03.05.2007, Journal of the Norwegian Psychological Association.