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# Mental imagery as an emotional amplifier: Application to bipolar disorder

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ABSTRACT

Cognitions in the form of mental images have a more powerful impact on emotion than their verbal counterparts. This review synthesizes the cognitive science of imagery and emotion with transdiagnostic clinical research, yielding novel predictions for the basis of emotional volatility in bipolar disorder. Anxiety is extremely common in patients with bipolar disorder and is associated with increased dysfunction and suicidality, yet it is poorly understood and rarely treated. Mental imagery is a neglected aspect of bipolar anxiety although in anxiety disorders such as posttraumatic stress disorder and social phobia focusing on imagery has been crucial for the development of cognitive behavior therapy (CBT). In this review we present a cognitive model of imagery and emotion applied to bipolar disorder. Within this model mental imagery amplifies emotion, drawing on Clark's cyclical panic model [(1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*, 461–470]. We (1) emphasise imagery's amplification of anxiety (cycle one); (2) suggest that imagery amplifies the defining (hypo-) mania of bipolar disorder (cycle two), whereby the overly positive misinterpretation of triggers leads to mood elevation (escalated by imagery), increasing associated beliefs, goals, and action likelihood (all strengthened by imagery).

Imagery suggests a unifying explanation for key unexplained features of bipolar disorder: ubiquitous anxiety, mood instability and creativity. Introducing imagery has novel implications for bipolar treatment innovation - an area where CBT improvements are much-needed.



## Introduction

We propose that the presence of "affective, intrusive mental imagery" could prove to be much more prevalent and important than is currently appreciated in bipolar disorder. Our approach supposes a central role for involuntary, distressing mental images about the past or future that come to mind unbidden, typically with high levels of affect. Such images have already provided a distinct and critical Cognitive Behavior Therapy (CBT) treatment target in conditions where the salience of imagery is obvious [PTSD (Ehlers & Clark, 2000) and social phobia (Clark et al., 2006)]. However, to date, the potential roles of imagery have been neglected in other mental disorders with a high prevalence of comorbid anxiety. In particular, the emotional and behavioural disturbance in bipolar disorder seem to us singularly unexplored from this point of view.

Mental imagery has been described as that which occurs when perceptual information is accessed from memory, giving rise to the experience of "seeing with the mind's eye" or "hearing with the mind's ear" (Kosslyn, Ganis, & Thompson, 2001). While perception occurs when information is directly registered from the senses, mental imagery also has neural correspondence with perceived visual stimuli. For example, the same selective neural activation pattern occurs when people simply imagine familiar faces as when they perceive them (O'Craven & Kanwisher, 2000). Thus, mental imagery differs from verbal thought by possessing sensory properties that are both consciously reported and whose representation can be demonstrated at a neural level. Moreover, while drawing on memory, imagination clearly allows us not only to relive events we have actually experienced ("re-experiencing") but to create novel combinations of never-experienced images and to allow mental "time travel" forward to what might happen in the future. That is, we can "pre-experience" future events. The neural processes in autobiographical recall co-localize with those that support imagining the future (Schacter & Addis, 2007; Schacter, Addis, & Buckner, 2007; Schacter, Addis, & Buckner, 2008). Furthermore, we are more likely to act on those events we have simulated in imagination than those we have thought about verbally (Libby, Shaeffer, Eibach, & Slemmer, 2007; Markman, Gavanski, Sherman, & McMullen, 1993; Sanna, 2000).

As imagery has assumed increasing importance in cognition, it is also now appreciated to be a critical process in exacerbating states

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of normal and abnormal emotion. Holmes and Mathews (2005) proposed that emotional processing in the brain is particularly sensitive to imagery (rather than verbal thought) for several reasons. Basic emotions such as fear evolved relatively early in our evolutionary history, prior to language (Öhman & Mineka, 2001). Rapid response to imagery facilitates rapid responding to sensory events signaling danger or reward. Since processes involved in mental imagery overlap with those in perception (Kosslyn et al., 2001), imagined events may be responded to "as if" real. Episodic memories are image-based (Conway, 2001), and this is particularly so for emotional memories (Arntz, de Groot, & Kindt, 2005), thus imagery has enhanced access to previous emotional episodes. Interestingly, individuals prone to acquiring specific fears and phobias, compared to those who are not, have higher trait levels of imagery (Dadds, Hawes, Schaefer, & Vaka, 2004). Thus, imagery susceptibility (that is, the tendency to be a "visualizer" rather than "verbalizer") may be a neglected risk factor for psychiatric disorder.

The goal of this review is to link the evidence that imagery has an amplifying effect on emotion with the still little-understood clinical phenomenology that bipolar is an emotionally volatile disorder. Thus, using anxiety as a starting point, imagery is predicted to serve as an emotional amplifier of a variety of mood states in bipolar disorder. We will review the potential contribution of imagery to key features of bipolarity: anxiety, depression/suicidality, mood elevation and creativity. We will illustrate some of our conclusions by reference to patient experience as reported to us in clinical consultation. We believe that, despite a dearth of direct experimentation in bipolar disorder, there is the beginning of an important message for future investigation of its psychopathology and for the development of potential adjunctive psychological treatments.

## Anxiety in bipolar disorder

As many as 90% of bipolar patients have a comorbid anxiety disorder in their lifetime (Merikangas et al., 2007). This rate appears substantially higher than the prevalence of comorbid anxiety disorder in major depression (50%; Kessler et al., 2003) or non-affective psychosis (63%; Kessler et al., 2005). The comorbidity includes all anxiety diagnoses. Hence it is not surprising that anxiety has been suggested as a core dimension of bipolar disorder (Simon et al., 2004): how and why remains unexplained. Anxiety may have implications for understanding the etiology and expression of bipolar disorder. It certainly has potential implications for its treatment. A recent review (McIntyre et al., 2006) concluded that anxiety symptoms often precede and may hasten the onset of bipolar disorder, and are associated with increased dysfunction (see also Otto et al., 2006). Anxiety comorbidity is most likely in those with Bipolar II disorder (BPD-II), mixed or dysphoric episodes and rapid cycling is associated with poorer treatment response to pharmacological and psychotherapeutic treatments (Feske et al., 2000; McIntyre, Mancini, Parikh, & Kennedy, 2001; Simon et al., 2004). Importantly, anxiety in the context of bipolar disorder is also a risk factor for increased suicide attempts and suicide completion (Dilsaver & Chen, 2003; Simon, Hunkeler, Fireman, Lee, & Savarino, 2007). Given that bipolar disorder poses one of the greatest risks for suicide of all psychiatric disorders (Angst, Angst, Gerber-Werder, & Gamma, 2005; Hawton, Sutton, Haw, Sinclair, & Harriss, 2005), especially when comorbid with anxiety disorders (Simon et al., 2007), anxiety demands further investigation and the development of effective treatment strategies.

While bipolar disorder remains a disorder in which pharmacological treatments predominate, the use of medication to treat anxiety within this disorder is unproven and possibly contraindicated. There are several pharmacological treatments of primary (non-comorbid) anxiety disorders. For example, the SSRIs and related medicines such as venlafaxine have efficacy in anxiety/depression, generalized anxiety disorder, panic, posttraumatic stress disorder and obsessive compulsive disorder (Baldwin et al., 2005). They have not been examined in the specific indication of comorbid anxiety in Bipolar I disorder (BPD-I; characterized by a history of depression and mania) or BPD-II disorder (characterized by a history of depression and hypomania). However, SSRIs and venlafaxine can induce switch to (hypo) mania if used in bipolar disorder (Post et al., 2006). The symptomatic use of benzodiazepines, gabapentin and alcohol appear to be common as anxiolytic strategies in bipolar disorder (Goodwin, 2003).

The absence of evidence on which to base the drug treatment of anxiety in bipolar disorder is matched by a corresponding absence of formal investigation of the effectiveness of psychological treatments targeting bipolar anxiety. CBT has been highly successful, for example for panic disorder (Barlow, 2002; National Institute for Health and Clinical Excellence, 2004). However, the efficacy of CBT in bipolar disorder remains uncertain. Whilst some cognitive programs focusing on education seem to succeed in preventing new episodes (Colom & Lam, 2005; Lam et al., 2003), there is little evidence to support a wider and routine use of generic CBT with bipolar patients (Scott & Colom, 2008). One major trial (Scott et al., 2006) was disappointingly negative (see also National Institute for Health and Clinical Excellence, 2006). However, CBT for bipolar disorder appears to have been rather pragmatic in its development so far. The underlying cognitive models of bipolar disorder have focused on depression and mania and as yet have failed to lead to adequately robust treatments. Moreover, cognitive aspects of anxiety, described below, do not appear to have been adequately explored in the conceptualizations of bipolar disorder. Other evidence-based psychosocial therapies for bipolar disorder such as family focused therapy (Miklowitz, George, Richards, Simoneau, & Suddath, 2003; Miklowitz et al., 2007), interpersonal social rhythm therapy (Frank et al., 2005; Frank, Swartz, & Kupfer, 2000) and psychoeducation (Colom et al., 2003) while not specifically targeting anxiety processes, share an objective of avoiding stressors.

The relationship between bipolar disorder and anxiety can be conceptualized in several different ways.

1) Anxiety may be an irrelevant comorbidity of bipolarity: it may be so common because bipolar disorder itself is severe and 'pulls in' other diagnoses in part by ascertainment bias (a further diagnosis is more likely to be detected), in part via shared risk factors or even as a secondary effect of the primary psychopathology. However, this seems unlikely because the comorbidity is present across samples of bipolar disorder, including those drawn from the community, where severity is often modest. Moreover, schizophrenia does not show the same level of comorbidity, despite its comparable and, on some indices, greater severity.
2) Anxiety may be a developmental accompaniment of bipolar disorder, preceding onset in the life history, but not having a direct role in bipolarity per se. This trajectory might or might not be unique to bipolar disorder, but is compatible with an independent expression and evolution of anxiety disorders on the one hand and bipolarity on the other.
3) Anxiety symptoms may be a precursor required for the development and full expression of bipolarity. On this view anxiety symptoms may mediate some of the characteristic phenomena of bipolar illness course such as increased mood reactivity and intrinsic mood instability. Childhood trauma, for example, appears to be associated both with a high incidence of PTSD and a more severe burden of bipolar episodes (Leverich & Post, 2006).

Currently there is little clinical, observational or experimental work to definitively distinguish these three possibilities. However, we are drawn to the third, quite simply because it could have such