Chapter 1

Bipolar disorder

Moods are so essential to our navigating the world that when they go awry it is only a matter of time until distress and disaster hit. Moods allow us to gauge people and circumstance, alert us to danger and opportunity, and provide us with the means to convey our emotional and physical state to others.

Jamison (2003, p. xv)

Bipolar disorder, also known as manic depression, is a mood disorder that can involve extreme changes in affect, cognition, and behavior. In its extreme form, bipolar disorder can be associated with psychotic symptoms and can require inpatient admission due to disorganization and impulsivity in the manic phase, or due to suicidal ideation or neglect of self-care in the depressive phase. It affects males and females in equal numbers, and has similar rates across all socio-economic groups. Its onset generally occurs during late adolescence or early adulthood, with this having significant implications for the person's developmental trajectory and quality of life. This will be described later in the chapter.

While the *Diagnostic and Statistical Manual of Mental Disorders: Fourth Edition – Text Revision* (DSM-IV-TR) (American Psychiatric Association, 2000) should be consulted regarding diagnosis, a brief summary is as follows:

- A major depressive episode is diagnosed through the presence of depressed mood or loss of interest or pleasure for most of the day, nearly every day for two weeks or more. It must also be accompanied by five or more from nine symptoms, including feelings of worthlessness or guilt, insomnia or hypersomnia, psychomotor agitation or retardation, and fatigue.
- A manic episode is diagnosed through the presence of elevated, expansive, or irritable mood lasting at least one week, and of three or more from seven additional symptoms (or four or more if the mood is only irritable) including inflated self-esteem, increased talkativeness, reduced need for sleep, flight of ideas, and an increase in goal-directed activity.
- Hypomania can be seen as a milder form of mania. It draws from the same list of seven symptoms as mania, but symptoms only need to have been present for four days and cannot include psychotic symptoms. It does not require hospitalization or cause marked impairment in social or occupational functioning.
- A mixed episode occurs when a person meets criteria for both a manic episode and a major depressive episode nearly every day for at least one week, and when the disrupted mood causes a significant level of impairment in functioning.

The DSM-IV-TR defines four main subtypes of bipolar disorder:

- Bipolar I disorder, in which the person must have experienced at least one manic episode.
- Bipolar II disorder, where the person has had one or more depressive episodes, and at least one hypomanic episode, with no manic or mixed episodes.

- Cyclothymic disorder, where for at least two years, the person has had numerous periods of hypomania and depressive symptoms that do not meet criteria for a major depressive episode, and has not been without these for more than two months at a time.
- Bipolar disorder not otherwise specified, which can include very rapid alternation of manic and depressive symptoms, which meet symptom criteria but not minimum duration.

It is notable that clients do not always present with symptoms that fit classic textbook descriptions, and this is especially true in the early stages of bipolar disorder. Many researchers in the field describe the concept of bipolar *spectrum* disorders, which encapsulates the various manifestations of disorders that do not fit the criteria for bipolar I disorder, but nevertheless seem to be part of this diagnostic grouping. As young people often present with variations of the classic symptoms or syndromes (a cluster of symptoms that usually co-occur) of bipolar disorder, this topic will be described further in Chapter 2.

Clinical descriptions of manic and depressive symptoms have a long history. For example, in the first century AD, in his book *On the Causes and Symptoms of Chronic Diseases*, Arataeus gave a description of different types of manic symptoms which could still apply today. He noted: "Some patients with mania are cheerful – they laugh, play, dance day and night, and stroll through the market, sometimes with a garland on their head, as if they had won a game: these patients do not worry their relatives. But others fly into a rage . . . the manifestations of mania are countless. Some manics, who are intelligent and well educated, deal with astronomy, although they never studied it, with philosophy, but autodidactically, they consider poetry the gift of muses" (Marneros & Goodwin, 2005, p. 5).

While bipolar disorder is generally thought to affect around 1.6% of the population (Kessler et al., 1994; Bauer et al., 2002), there has been growing recognition of the concept of a spectrum between unipolar depression and bipolar disorder (Phelps et al., 2008). Using a broader definition, between 3.5% and 6% of people may experience some form of the disorder (Elgie & Morselli, 2007; Merikangas et al., 2007).

Given its prevalence, persistence, and the degree of impairment that can be associated with bipolar disorder, it should not come as a surprise to note that it is rated the sixth leading cause of disability among all types of physical or mental health disorders in people aged 19–40 (Murray & Lopez, 1996), a higher position than schizophrenia. In financial terms, Begley et al. (2001) calculated that the lifetime cost of all people developing bipolar disorder in the United States in 1998 was $24 billion, with average costs ranging from $11 720 for a person experiencing a single manic episode, to $624 785 for a person with multiple episodes. Furthermore, it has been estimated that an adult developing bipolar disorder in his/her mid-twenties effectively loses 9 years of life expectancy, 12 years of normal health, and 14 years of work activity (Prien & Potter, 1990).

Historically it has been suggested that whilst bipolar disorder has the potential to be recurrent with high rates of relapse, most individuals experience good inter-episode recovery. However, this was challenged by a prospective study of 146 people with bipolar I disorder, which found that participants were symptomatic for over 47% of the 13-year follow-up period (Judd et al., 2002). The percentage of time spent in each phase of the disorder is illustrated in Figure 1.1. Joffe et al. (2004) reported similar findings in their study, which found that bipolar participants experienced depressive symptoms for 40.9% and manic symptoms for 6% of the time during a follow-up period of almost 3 years.

In addition to poor inter-episode recovery, bipolar disorder has one of the highest lifetime risks for suicide associated with any psychiatric disorder, with research indicating that between 15% and 20% of people with the disorder take their own lives (Goodwin & Jamison, 1990). Notably, the suicide rate in bipolar disorder has been found to be almost

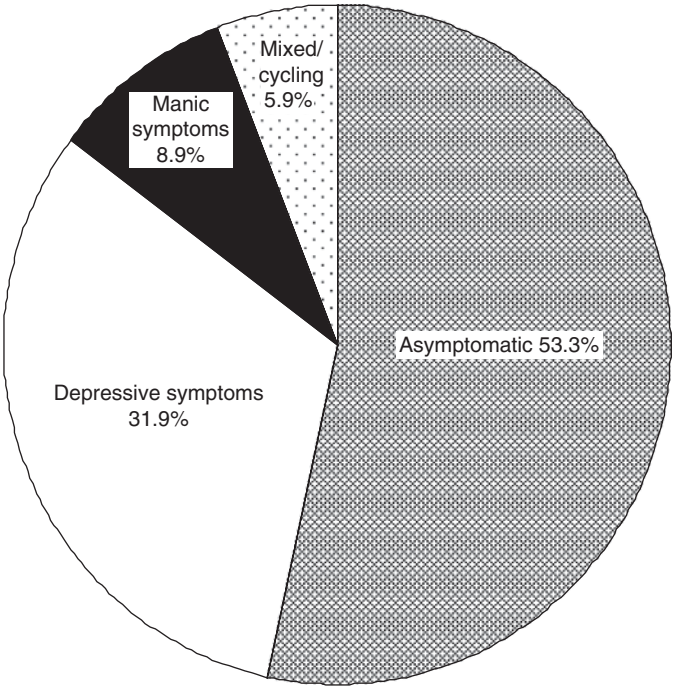

Figure 1.1 Breakdown of time spent in various phases of bipolar disorder. Data taken from Judd et al., 2002.

double that of unipolar depression (Chen & Dilsaver, 1996; Mitchell & Malhi, 2004). Encouragingly, however, a long-term naturalistic follow-up demonstrated that receiving treatment is associated with lower suicide rates and lower mortality rates from all causes compared with not receiving treatment (Angst et al., 2002).

Difficulties associated with bipolar disorder

A number of difficulties have been found to be associated with bipolar disorder, including:

- Adjustment problems have been noted to affect a number of people in the initial phase of bipolar disorder. For example, Goodwin and Jamison (1990) found that on discovering the disorder may be chronic and recurrent, many people reported experiencing ambivalence, anxiety, disappointment, denial, and anger.
- Financial difficulties have been reported by 70% of people with bipolar disorder and their partners, with these often remaining long-term (Targum et al., 1981).
- Poor self-esteem is highly prevalent, with evidence that people with bipolar disorder may view themselves as different or defective, even when asymptomatic (Rush, 1988).
- Even when in remission, people with bipolar disorder have been found to have poorer social adjustment when compared with control participants (Blairy et al., 2004).
- Coryell et al. (1993) summarized: "The psychosocial impairment associated with mania and major depression extends to essentially all areas of functioning and persists for years, even among individuals who experience sustained resolution of clinical symptoms" (p. 720). While Coryell and colleagues may be describing the more severe end of the bipolar spectrum, it is nevertheless notable that the disorder can have a negative impact on a significant number of people with the diagnosis.

However, even with mounting evidence of the potential difficulties associated with bipolar disorder, many people remain untreated, with a recent World Health Organization

bulletin estimating that the "treatment gap" for bipolar disorder (i.e. the difference between those experiencing the disorder and those receiving treatment) was 56% (Kohn et al., 2004).

Etiology of bipolar disorder

The search for causes of bipolar disorder has existed since the emergence of the disorder itself. The ancient Greeks believed that mood and behavioral disturbances were caused by imbalances in our essential body fluids or "humors" (Evans et al., 2003), with a similar concept being promoted by the physician Alcmaeon (around 500 BC), who described sadness as being related to the interaction between bile and the brain (Angst & Marneros, 2001).

Until recently, the dominant models of bipolar disorder have continued to focus on biological factors, specifically neurotransmitters and neuroendocrine theories as well as considerable debate about the role of genetic factors. However, the last few decades have also seen a greater emphasis placed on stress-diathesis models of bipolar disorder (Prien & Potter, 1990; Scott, 1995; Scott & Colom, 2005). Like the literature on schizophrenia, there has been an increasing acknowledgement that genetics and biological factors alone cannot account for the emergence of bipolar disorder and that psychosocial factors, such as personality or cognitive style, the experience of significant life events, and aspects of family environment such as negative affective style or high levels of expressed emotion, can increase the risk of relapse in bipolar disorder or adversely affect the prognosis of an episode. The current consensus is that a multi-factorial model offers the most robust explanation of the causes of bipolar disorder, no single gene is the "primary cause," and no psychological or social factor can fully explain why some individuals develop bipolar disorder whilst others, with the same genetic predisposition, do not.

As individuals who develop bipolar disorder and their families often ask about the possible causes, we now offer brief comments on some key issues in current biological research. We then highlight areas of interest in psychosocial research that are relevant to the clinical sections later in the book. This is not meant to be an exhaustive review of every current model, and we recognize that new findings will be published that will overtake some of the ideas discussed here, and that the psychobiosocial model will continue to evolve over the coming decades. Most importantly, the key "take-home" message is that individuals appear to inherit a "risk" of developing bipolar disorder and whether that risk is expressed or not (i.e. whether they manifest the symptoms of the disorder) will be determined by a number of psychobiosocial factors.

Genetic and biological models

Current research on etiology tends to maintain a strong biological focus, and a range of biological factors have been implicated in the causation of bipolar disorder. The strong familial clustering of cases gives an indication that inheritance (and therefore genes) plays a role in the causes of bipolar disorder, and research has identified several candidate genes which may be associated with the disorder, as well as specific loci that may confer risk (Abou Jamra et al., 2007; Kato, 2007; Sklar et al., 2008).

Twin studies have generally shown that if one twin has bipolar disorder, the risk in the co-twin is much higher than expected by chance. For example, Berrettini (2000) reported that concordance rates were 14% for dizygotic twins (who have half their genes in common) and 65% for monozygotic twins (who are genetically identical). However, whilst the levels of concordance clearly indicate that genetic factors are important, the fact that in many pairs only one identical twin manifests the disorder indicates that genes alone cannot explain the variance. As Bauer and McBride (2003) succinctly concluded, "Thus, it is impossible . . .

that manic depressive disorder is totally genetically determined" (p. 38). Furthermore, results of studies investigating genetics in bipolar disorder are generally not consistent with the notion of a single causative gene for the disorder. Therefore, it appears likely that the contribution of genetics to etiology is through a complex interaction of a number of genes and the environment (Hyman, 1999).

As in other mood disorders, neurotransmitter abnormalities and dysfunctional neuroendocrine stress responses have also been implicated in the onset and/or outcome of bipolar disorder. The main weakness of such models is the absence of a robust theory that explains why changes lead to mania in some circumstances and depression in others. Research focusing on neurotransmitters has shown a deficiency in norepinephrine in people with depressed mood, and changes in serotonin and dopamine have been linked with manic and psychotic symptoms (Zubieta et al., 2000).

Recent promising work on neural pathways has identified reduced activation of the dorsolateral prefrontal cortex and orbitofrontal cortex, and increased activity in the amygdala in adults with bipolar disorder (for a review see Pavuluri, 2004). Similarly, a review by Malhi et al. (2004) cited research indicating reduced prefrontal and subgenual cingulate volumes, and enlargement of subcortical and medial temporal structures such as the basal ganglia and amygdala, in people with bipolar disorder. These circuits are important, as changes in their functioning may explain some of the changes in mood and activation observed in bipolar disorder and perhaps tentatively help us understand why atypical antipsychotic medications (which act predominantly on dopamine pathways) appear to be useful in treating acute bipolar episodes and stabilizing an individual's mental state. Furthermore, they form part of the behavioral activation system, which, through its role in rewards and goal-directed behavior, is now seen as an important potential pathway linking life events, cognitive style, and brain activity with onset of mood episodes.

Neurobiological research in bipolar disorder has also found increases in the volume of the lateral brain ventricles (Swayze et al., 1990), while other studies have noted increased binding cells in the thalamus and ventral brain stem (Zubieta et al., 2000). Inconsistent results have been observed in temporal lobe studies, and there has been some indication that hippocampal volume may also contribute to the etiology of bipolar disorder (Frey et al., 2007). A difficulty with much of this research is whether these changes are a cause or a consequence of the mental disorder.

Psychosocial models of bipolar disorder

Despite the strong historical research focus on exploring biological and genetic etiological pathways to bipolar disorder, there is emerging evidence that life events, stress (including family stress and high expressed emotion), and cognitive style can influence the course of bipolar disorder, especially the likelihood of relapse. The exact role of these factors in the onset of the first episode is less clear cut, but there is increasing evidence that psychosocial stressors can precipitate the onset of symptoms so that an underlying vulnerability to bipolar disorder becomes manifest in those at risk (Scott, 2003).

There is currently a complex literature describing potential psychosocial mechanisms relating to bipolar disorder, and a detailed analysis of each theory is beyond the scope of this manual. We will therefore focus on a brief description of the evolution of the concept of the "manic defense" and its recent reformulations and then highlight some more recent cognitive models that together inform the integrative model we use in our clinical work. Interested readers should also refer to Healy and Williams (1989) or Scott (2003) for a review of cognitive models, and Power's (2005) excellent overview of currently dominant

psychosocial models. These models generally share the view that specific cognitive styles, when interacting with life events and biological vulnerability, may increase the likelihood of developing manic or depressive symptoms, and that exacerbation of these symptoms is in turn further driven by underlying beliefs and attributions about the meaning of the early symptoms experienced by an individual.

The "manic defense," self-esteem and bipolar disorder

Research describing the contribution of psychological factors to the onset and course of bipolar disorder has a long history. In 1911, Karl Abraham suggested that rather than being polar opposites, mania and depression "are dominated by the same complexes, and that it is only the patient's attitudes to these complexes that is different" (Bentall, 2003, p. 277). At this time, the concept of the "manic defense" emerged. Klein (1974) provided a succinct definition that "mania is a massive defence arising from the failure of the containing processes which normally occur in the infant-mother relationship, and which results in a catastrophic fragmentation of the ego" (p. 261). Bateman et al. (1954) provided another psychoanalytic conceptualization of mania, which focused on the role of trauma. They suggested that past or present trauma can result in guilt, which is avoided or defended against by "desperate vigilance and by a psychologic counteroffensive which is no longer consistent with reality" (p. 353). They further commented: "It is not sufficient for the manic merely to avoid painful recollections or stimuli. He must actively evade, repudiate, or counter-attack them. Therefore, the manic mood is one of extreme tenseness" (p. 353). They concluded: "All the symptoms, in addition to serving the essential manic aim, also serve a secondary aim of protecting the patient from external interference while he is engaged in his attempt at regaining psychic equilibrium" (p. 356).

In the 1960s, the rise of the biological model of bipolar disorder and the emphasis on treatment with medications overshadowed psychological theories, and the potential implications of the manic defense received less attention. This was compounded by the revolution in psychology with cognitive and behavioral models that were amenable to empirical testing being favored over psychoanalytic theories, which at times also defied easy comprehension (e.g. Morgenson, 1996). Studies that explored self-esteem and, later, social desirability and self-representations, also began to appear. For example, Winters and Neale (1985) wrote a highly influential article in which they hypothesized that although research participants with remitted bipolar disorder did not usually *report* impaired self-esteem, they may nevertheless have *experienced* cognitive schema relating to low self-esteem. Winters and Neale used the Pragmatic Inference Test (PIT), an implicit measure of self-esteem in which participants are given ambiguous stories (such as a person becoming unemployed or a first date going badly) and are then asked to attribute why the event occurred. They found that, even when in remission, people with a history of bipolar disorder or unipolar depression were significantly more likely than control participants to attribute unsuccessful outcomes to factors relating to the person. One implication of this finding is that people prone to bipolar disorder or unipolar depression may be more likely to blame themselves following a negative event rather than look to external environmental factors. Winters and Neale concluded that participants with bipolar disorder might have negative feelings about the self that were not revealed on the typical, explicit self-report measures employed in research settings.

The findings of Winters and Neale (2005) have been replicated by Lyon et al. (1999), who also observed that while manic participants were more likely to *endorse* positive words as being descriptive of themselves (e.g. "successful," "capable," and "valuable"), similarly to

bipolar depressive participants, they were more likely to *recall* negative words (e.g. "deficient," "unloved," and "weak"). The authors suggested that these findings – which utilized implicit rather than explicit measures – also appear to support the concept of mania serving a defensive function against poor self-esteem, and occurring beyond the person's awareness.

Neale (1988) proposed that *unstable* self-esteem coupled with unreasonable standards for success may be predisposing factors for bipolar disorder. Although there is little empirical support for this hypothesis, Pardoen and colleagues' (1993) study of self-esteem confirmed the presence of social conformism in people with bipolar disorder. Furthermore, recent research has shown that people with bipolar disorder often indicate lower self-esteem on more subtle or implicit measures, possibly as some core beliefs or schemas occur outside the person's conscious awareness (Timbremont & Braet, 2004). A key methodological lesson from these studies is the need to consider the use of implicit as well as explicit measures of cognitive style (Bentall, 2003).

Recent studies on levels of self-esteem in bipolar disorder suggest variations may also be a function of lability as well as differences between implicit and explicit ratings (Scott, 2003). Scott and colleagues have identified that labile self-esteem, rather than a fixed low level of self-esteem, may potentially differentiate people with bipolar disorder from people with severe unipolar disorders in the depressed or euthymic phases of disorder. However, unstable self-esteem and low levels of self-esteem are both known to confer similar levels of risk for depressive relapse (Kernis et al., 1993).

This research has led to a number of theorists, including Bentall (2003), revisiting the concept of the manic defense and adapting it to a more contemporary cognitive model, whereby the person may be protected from a poor or fluctuating self-esteem and a strong need for approval from others by grandiose beliefs and elevated mood.

Thomas et al. (2007) also supported the hypothesis that mania can result from a coping style which attempts to avoid negative emotion. They found that manic participants – compared with depressed bipolar participants, remitted bipolar participants, and non-bipolar controls – showed higher levels of risk-taking and active coping. They summarized: "Although the findings provide support for a version of the depression-avoidance hypothesis, the mechanism proposed here is simpler than that proposed by psychoanalytic theorists" (p. 251). They concluded that excessive distraction may overload the behavioral activation system, and that disruption of circadian rhythms may contribute to the etiology of mania.

Stressful life events, "daily hassles" and circadian rhythm disruption in bipolar disorder

Several studies have implicated stress and life events in the initial onset and relapse of bipolar disorder. For example, Bebbington et al. (1993) found that, compared with control participants, people developing bipolar disorder experienced significantly more critical life events, particularly in the three months prior to their first episode. This pattern remained even when incidents that may have been related to the disorder itself were removed. Similar findings have been reported by Kennedy et al. (1983), Frank et al. (2000), and Hammen and Gitlin (1997). A prospective study by Ellicott et al. (1990) found that individuals with bipolar disorder who had experienced a high number of stressful life events were in excess of four times more likely to relapse than those who had not encountered similar stressors. A meta-analysis by Altman et al. (2006) concluded: "The majority of research in the field, in both small and large studies, supports the notion that stressful life events have an overall

negative impact on outcomes in bipolar disorder, both in terms of contributing to relapse and in lengthening time to recovery" (p. 273). Furthermore, research in both circadian rhythms (Hlastala et al., 2000) and neurobiology (Post, 1992) has indicated that people who have had fewer episodes, and young people specifically, may be even more vulnerable to bipolar disorder in response to life events.

A large study involving 1565 individuals with bipolar disorder and 16 200 age- and gender-matched controls (Kessing et al., 2004) was able to elucidate the relative importance of some specific life events in bipolar disorder. This study found that the suicide of a mother or sibling significantly increased the risk of a first admission for a manic or mixed episode by 5.75 and 4.7 times respectively. Recent unemployment and divorce both increased the likelihood of a first admission by 1.5 times, and marriage in the previous year almost doubled the risk of first admission.

However, rather than focusing solely on the importance of significant life events, some recent research on young people has examined "daily hassles" – including everyday annoyances such as minor arguments or disagreements – and their influence on mood and behavior. Dumont and Provost (1999) suggested that focusing on daily hassles may allow for more subjective analysis, which more closely reflects people's experiences than objective measures. Secondly, they noted that "life events are relatively rare, whereas daily hassles are common and show a greater interindividual variance" (p. 345). Finally, they suggested that research has shown that daily hassles have a greater influence on mental health difficulties than life events. They concluded: "This implies that daily hassles might be better predictors of the psychological health of young adolescents than are life events" (p. 345). This is supported in research by Thompson et al. (2007), who measured plasma cortisol levels in young people at high risk of developing psychotic disorders, as this has been found to correlate with symptoms of anxiety and depression. They reported that while there was no correlation between cortisol levels and significant stressful life events, there was a positive correlation between cortisol levels and the number of day-to-day hassles reported by the young person.

There has been some debate as to the mechanism by which stressful life events or daily hassles could lead to onset and relapse in bipolar disorder. One suggestion is that this occurs through the impact of such events on circadian rhythms, with disruption to these having long been suspected as playing a part in the development of bipolar disorder and relapse. For example, Malkoff-Schwartz et al. (1998) reported that stressful life events that involved social disruption were associated with the onset of manic episodes, regardless of the episode's severity. Interestingly, however, *severely* stressful life events, regardless of their level of social disruption, have been correlated with the onset of manic and depressive episodes (Frank et al., 2000). As Satterfield (1999) succinctly described in a paper on rapid-cycling bipolar disorder, "life events disrupt critical social rhythms and push biologically and affectively vulnerable ... patients into a cascading state of biological and affective dysregulation" (p. 359).

A specific aspect of circadian rhythms that has attracted significant attention has been the area of sleep. Colombo et al. (1999) found that 10% of people with bipolar disorder developed manic or hypomanic symptoms after induced sleep deprivation. A recent prospective study by Meyer and Maier (2006) specifically examined the role of sleep patterns in young people. They found that those at risk of bipolar disorder did not differ in their *amount* of sleep, compared to control participants and people at risk of unipolar depression, but did differ on their *regularity* of sleep and daily activities. Meyer and Maier concluded: "From a theoretical as well as empirical point of view, there is reason to assume that

circadian rhythms play a central role in the origin and course of bipolar disorders" (p. 104). Even subtle changes in sleep and biological rhythms may be clinically meaningful, with Berk et al. (2008a) having found a significant increase in suicide rates in a national sample after transition to daylight saving, which alters sleep rhythms by only one hour.

Johnson (2005) reported that life events significantly affected speed of relapse and length of time for recovery from both manic and depressive episodes. However, Johnson differentiated between the type of events experienced by the person and the polarity of their mood. Specifically, manic episodes were more likely to be affected by events that upset circadian rhythms and involved goal attainment, while depressive episodes tended to be related to negative life events.

While circadian rhythm disruption offers one possible etiological factor, the mechanism by which life events affect mood has been subject to considerable discussion. Scott (2003) noted that cognitive changes and sometimes mood elevation may occur following sleep disruption, but whether isolated manic symptoms then cascade into a full-blown episode depends on whether the individual makes dispositional or situational attributions. Healy and Williams (1989) provided the example, "this efficiency and speed is due to my natural intuition and intelligence," leading to actions that increase rather than decrease the risk of manic relapse. Other researchers conceptualize bipolar disorder as occurring due to individuals having "impaired shock absorbers," in which the person's ability to "cushion" the impact of positive or negative events is less effective than that of people who do not have bipolar disorder. A literature review by Johnson (2005) concluded: "Even when they are euthymic, people with bipolar disorder appear to experience frequent and intense emotions in response to environmental conditions" (p. 251).

Bentall (2003) reported a model similar to the "impaired shock absorber" theory, describing an "excitability hypothesis" in which people with bipolar disorder experience a dysregulation of behavioral activation, and crave social contact, excitement, and motor activity. He found that people with bipolar disorder endorsed questionnaire items including "when good things happen to me, it affects me strongly" and "I will often do things for no other reason than that they might be fun" (p. 289). Bentall (2003) elaborated by suggesting that when people with bipolar disorder experience negative life events, this interacts with a basic underlying dysphoria and a pessimistic attributional style, which may lead to depression. However, if combined with distraction or high levels of behavioral activation – such as becoming involved in risk-taking behavior and drive to succeed – the resulting excitement and circadian rhythm disruption (i.e. changes in eating patterns and sleep loss) can lead to mania.

It is also important to note the importance of the bidirectional nature of stress, coping, and mood, as stressors can impact on coping style, which may in turn result in the experience of more stress and altered mood. Most notably, coping strategies such as substance use or social withdrawal – while potentially an attempt to reduce short-term stress – appear likely to result in further difficulties and exacerbation of mood symptoms.

Cognitive style and bipolar disorder

While the relationship between life events and mood is complex, a mediating factor appears to be attributional style, and a number of researchers (e.g. Lam et al., 1999; Bentall, 2003; Johnson & Leahy, 2004; Ramirez-Basco & Rush, 2007) have described models for understanding bipolar disorder within a cognitive behavioral framework. Pioneering work was conducted by Aaron Beck (1976), who noted that many people with depression appeared to have characteristic ways of thinking which were either inaccurate or unhelpful. Specifically,

Beck defined the "negative cognitive triad," in which a person with depression will selectively attend to, and recall more easily, information which fits with their negative views of themselves, the world, and the future. According to Beck's model, this way of thinking impacts on the person's emotional state and behavior, which in turn impacts further on the person's negative thinking. Beck further described the bidirectional interaction between this thinking style and the environment, as people may actually find themselves failing at tasks or relationships, being criticized or withdrawing socially as a result of their impaired performance, which in turn could lead to further negative cognitions in a downward spiral of mood, behavior, and cognition. More recently, Lyon et al. (1999) similarly concluded: "Bipolar depression, like unipolar depression, appears to be characterised by a negative self-schema, a corresponding tendency to blame the self for negative experiences, and selective attention to depression-related stimuli" (p. 279).

Beck et al. (2006) noted that, similar to the negative cognitive triad common in people with depression, people prone to mania tend to hold clusters of *positive* beliefs about the self ("I am strong"), relationships ("everybody loves me"), pleasure/excitement ("I have to live for today"), and activity ("I have enough energy to do anything I want to") (p. 239). In Beck's model, mania may be a mirror image of depression, being associated with cognitive distortions such as jumping to positive conclusions, underestimating risks, minimizing problems, and overvaluing immediate gratification. In addition, positive experiences may be selectively attended to and recalled more easily, with negative events being minimized or ignored.

However, as elated mania is only one form of mania, it is also clear that further elaboration of cognitive models is required to explain dysphoric mania and mixed states (Scott, 2003). Furthermore, some researchers have suggested that rather than manic and depressive cognitions being polar opposites, they in fact share some similarities at the level of core beliefs or schemas, particularly in relation to the experience of poor self-esteem (Scott et al., 2000; Schwannauer, 2003). Ginsberg (1979) also suggested that the two disorders are similar, stating: "Mania does not end depression, it interrupts it" (p. 8).

Johnson (2005) observed that, compared with control participants, people with bipolar I disorder and students who were vulnerable to hypomania tended to have higher expectations of success, and were more likely to choose more difficult goals when given a choice of tasks. Furthermore, Johnson found that when experiencing a manic episode, people were more able to recall positive events, and were more likely to pursue difficult tasks and ignore danger cues after experiencing small successes. Johnson noted: "High goal setting appears to be a stable characteristic among persons with bipolar disorder" (p. 254). One clinical implication of this is that if people with bipolar disorder hold high expectations, and are more likely to be encouraged by small successes, they may become more driven to complete tasks that would be abandoned by people without the disorder. On occasions when success does eventuate, this could contribute to an upward spiral of energy, confidence, and disruption to sleep, which could precipitate a manic episode.

A further cognitive factor which has been implicated in the development and maintenance of manic or depressive episodes is that of memory bias. A review by Johnson (2005) stated that mood state could provoke recall for specific memories, such as low mood being related to a preoccupation with memories of negative past experiences. Similar findings emerged in studies by Eich et al. (1994) and Weingartner et al. (1977), who found that when depressed, people could recall equal numbers of positive and negative memories, whereas when hypomanic, they would recall more than three times as many positive as negative experiences. Clearly, such biases can be seen to have a role in maintaining manic or depressive symptoms.