CHAPTER

10 Mania and Bipolar Spectrum Disorders

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> When you're high it's tremendous. The ideas and feelings are fast and frequent like shooting stars, and you follow them until you find better and brighter ones. Shyness goes, the right words and gestures are suddenly there, the power to captivate others a felt certainty. . . . The fast ideas are far too fast, and there are far too many; overwhelming confusion replaces clarity. Memory goes. Humor and absorption on friends' faces are replaced by fear and concern.
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> —*Jamison (2004, p. 67)*

Bipolar disorder (or BD) is a serious and recurrent psychological disorder. As described in the above quote by Kay Redfield Jamison, BD is characterized by episodic and prolonged mood episodes that range between abnormally and persistently elevated mood phases lasting a week or longer (mania) and frequently periods of dysphoria mood (depression) lasting 2 weeks or longer (*DSM-5*). Importantly, BD is associated with significant and dire consequences including occupational, social, and even mortality costs (e.g., Coryell et al., 1993; Dilsaver, 2011; Romans & McPherson, 2002). BD has been associated with significant increases in suicidality and is rated as one of the leading causes of disability worldwide (e.g., Murray & Lopez, 1996; Schaffer et al., 2015). This chapter (a) provides an overview of the phenomenology and assessment of BD; (b) reviews associated psychobiological processes across cognitive, affective, neural, and circadian rhythm dimensions; (c) considers the influence of context and social environment on BD; (d) synthesizes empirically supported interventions to prevent and treat BD; and (e) highlights future directions in the study of BD that focus on increasing diversity and representation of marginalized communities and addressing stigma about BD and related psychiatric disorders.

Phenomenology and Diagnostic Criteria

In the *DSM-5*, bipolar spectrum disorders (BSDs) refer to a broad umbrella of mood-related difficulties that progress along a spectrum that encompasses varying degrees of depression and mania or hypomania-like severity and duration. This typically includes four categories: bipolar I (BD I, defined by the occurrence of at least one manic episode; bipolar II (BD II), which requires a lifetime combination of a major depressive episode and at least one hypomanic episode; cyclothymic disorder, characterized by hypomanic and depressive symptoms not severe enough to warrant a manic or major depressive label, yet still impairing and often more persistent. The last category, which had been referred to as BD Not

Abbreviations

BD	Bipolar disorder
BSDs	Bipolar spectrum disorders
FFT	Family-focused treatment
HPS	Hypomanic Personality Scale
ICM	Integrative cognitive model (of mood dysregulation)
IDAS	Inventory for Depression and Anxiety Symptoms
IPSRT	Interpersonal and social rhythm therapy
MDQ	Mood Disorder Questionnaire
OFC	Orbitofrontal cortex
SCN	Suprachiasmatic nucleus

Otherwise Specified (BD-NOS) is a residual group for cases where there are mood symptoms that are clearly a change from baseline and that are associated with impairment, but which fail to meet strict criteria for one of the other mood disorders.

Both the current *DSM-5* and *ICD* nosologies note that the depressed phases of BSDs have the usual symptoms of unipolar depression and also a lot of anxiety (Youngstrom & Van Meter, 2013). The hypomanic and manic presentations have high energy, rapid speech, and distractibility (shading into flight of idea) that can look similar to attention deficit hyperactivity disorder (ADHD); and the impulsive, goal-directed, rule-breaking behavior can lead to substantial interpersonal conflict and conduct problems. Periods of unusually elated, goofy mood or decreased need for sleep without fatigue (or even with increased energy) are less likely to be the focus of a clinical referral, but are more suggestive of hypomania than irritability alone, for example. The depressive phases of BSDs can have acute or gradual onset (more typical of persistent depressive or cyclothymic presentations) and often have a mix of increased energy and other manic symptoms, appearing as an "agitated depression" with an irritable mood. Hypomania also may be irritable as well as elated or euphoric, as might manic episodes. These, too, can have mixed presentations where anxious or depressive symptoms may be juxtaposed. Some data indicate that mixed presentations are even more common in youth than in later life. Data are inconsistent about whether the first episode is more likely to be a depressive or hypomanic episode (Van Meter, Burke, Youngstrom et al., 2016). The most helpful way to differentiate mood disorders from other issues is if the behaviors are a change from typical functioning, wax and wane, or manifest sometimes without an obvious environmental trigger (Youngstrom et al., 2008).

Epidemiological data indicate that cyclothymic disorder and otherwise specified bipolar and related disorders (OS-BRDs) are three to four times more common than BD I or II (Moreira et al., 2017; A. Van Meter et al., 2019), also consistent with the dimensional statistical models and our emerging understanding of etiology as involving both multiple genes and environmental risk factors. All the BSDs are associated with substantial impairment, and cyclothymia and OS-BRD have high rates of progression to BD I or II. Some data suggest that remission may be possible in a subset of cases, particularly with early and titrated intervention (e.g., Cicero et al., 2009). Other differential diagnoses to consider include all major depression and persistent depressive disorders (which will not have a history of hypomanic or manic episodes), anxiety disorders (which also will not have the hypomanic/manic history, though they may have the motor agitation and poor concentration), oppositional and conduct problems and ADHD (which will tend to be more chronic and less likely to show fluctuations in sleep or energy), and trauma or abuse (which could also be linked with an acute change in functioning). Because comorbidity is common, it is possible to have both BSDs and any of these, and BSDs may be a trigger as well as an outcome of some of these issues (Youngstrom & Algorta, 2014).

Assessment of Bipolar Disorders

To accurately determine a mood disorder diagnosis requires a longitudinal perspective. Because the course can be intermittent and with different polarities of episode, any single snapshot of clinical presentation provides an incomplete view. Only after gathering a careful developmental history, not just asking about current mood and functioning, but also looking for past episodes, are we ready to proceed with diagnostic formulation. The clinical encounter in which BD is assessed can be divided into four phases: preparation, prediction, prescription, and process/progress, described below.

PREPARATION

Before first meeting the patient, a psychologist or clinician can prepare to do a rapid yet accurate evaluation by having a good framework in place. This includes having a set of benchmarks for common issues, helping calibrate where BD ranks compared to other presenting problems. Reviews have gathered these across a variety of clinical settings (e.g., Youngstrom et al., 2020). Looking at the clinical prevalence of disorders in outpatient mental health clinics shows that conduct problems, ADHD, depression, and anxiety are the most common problems, with BSDs falling in a tier similar to the prevalence of posttraumatic stress disorder (PTSD) or conduct disorder. These in turn are more common than autistic spectrum or schizophrenia in the child and adolescent population typically coming to general purpose outpatient clinics. Such benchmarks provide a helpful anchor for clinical evaluations and decision-making. Having a rating scale toolkit[1]

[1] https://en.wikiversity.org/wiki/Evidence-based_assessment/Bipolar_disorder_in_youth_(assessment_portfolio)

along with semi-structured interview modules gathered ahead of time allows clinicians to rapidly follow up on clues to arrive at a case conceptualization.

PREDICTION

Several rating scales and checklists are well-suited for screening or rapid information gathering before the first appointment. These could be mailed ahead of time, completed in the waiting room, or even done online with automated scoring. A "core battery" should gather data about common issues (anxiety, depression, trauma, externalizing and attention problems, substance use), ideally from more than one informant's perspective. Less obviously, this is an opportunity to also gather family history (Algorta et al., 2013), and there are free, brief measures to get an indication of pubertal stage. There are more than a dozen scales focused specifically on manic symptoms for youths (Youngstrom et al., 2015), and more than 40 for use with adults (Youngstrom et al., 2018). Free PDFs of these in English, Spanish, and several other languages are available online, and some are available with free administration and scoring guides (https://www.hgaps.org/for-clinicians.html).

DURING PRESCRIPTION

During the prescription phase, the clinician will review the presenting problem, look at the results from the scales used in the prediction phase, and revise the probabilities attached to our list of hypotheses. They will use the interview to probe for confirming or disconfirming evidence and arrive at a working diagnosis and case formulation. The information-gathering here is in service of coming up with a treatment plan that addresses the key problems and guides our intervention selection to best meet the needs of the patient. Using more structured methods increases reliability and improves detection of comorbidity (Jensen-Doss et al., 2020).

PROCESS AND PROGRESS

During the next phase, assessment shifts to seeing how treatment is going. Process measures include keeping track of no-shows and short cancellations versus kept visits, whether the patient is doing "homework," and other indicators of engagement. Process measures could also include brief, direct ratings of therapeutic alliance, knowledge acquisition (especially with more psychoeducational modalities), sleep tracking apps, life charts, and mood records. There are a large and growing number of options for tracking whether we are "doing the work" together. Progress measures, in contrast, focus on "is treatment helping?" These measures can include brief symptom checks (e.g., severity ratings on mood charts), short forms of symptom scales, or nomothetic benchmarks for clinically significant change (Freeman & Young, 2020). A plan for long-term monitoring and early detection of relapse would be an excellent component of treatment termination planning, given the high recurrence risk associated with BSDs (Youngstrom et al., 2020).

Contextualizing Assessment Within Dimensional Frameworks

When diagnosing and assessing BSDs, recent attention has been given to considering alternative and more dimensional approaches to symptom severity and diagnosis. Specifically, decades of research indicate limitations of disorder definitions as described in the *DSM-5*, including poor interrater reliability, high levels of disorder co-occurrence, and within-disorder heterogeneity, among other issues (Kotov et al., 2020). As an example of within-disorder heterogeneity in the context of BSD diagnosis, two individuals could report mutually exclusive histories of specific hypomanic symptoms, but both could be diagnosed with BD II. For instance, the first individual could report a history of expansive mood, grandiosity, decreased need for sleep, and a marked increase in goal-directed activities, whereas the second could report a history of irritable mood, pressured speech, racing thoughts, distractibility, and engagement in risky or dangerous activities, yet both individuals would receive the same diagnostic label. Issues concerning interrater reliability and diagnostic comorbidity complicate treatment planning by making it difficult for clinicians to determine which presenting issues are primary and most impairing (e.g., individuals diagnosed with BD often also meet criteria for substance use disorders, personality disorders, and other disorders). Many *DSM-5* disorder descriptions also remain largely agnostic regarding etiological factors and mechanisms accounting for symptom onset and maintenance. Relatedly, many of these descriptions provide limited consideration of how social and contextual factors influence symptom presentation, symptom course, and treatment.

Accurate BSD diagnosis can be challenging because many individuals with a hypomania/mania history often present for treatment at times when depressed mood, anxiousness, interpersonal

difficulties, or other issues are present, rather than when hypomanic/manic symptoms are most prominent. Researchers have raised concerns about BSD underdiagnosis (Carta & Angst, 2016) because hypomania/mania symptom histories may go undetected if clinicians fail to adequately assess for a hypomania/mania history in individuals presenting to treatment for other issues. Measures such as the Mood Disorder Questionnaire (MDQ; Hirschfeld et al., 2000) have become commonly used to retrospectively assess for a hypomania/mania history. Still, many individuals may have difficulty accurately recalling their symptom histories.

Other researchers have cautioned that BSDs are *overdiagnosed* rather than underdiagnosed because some individuals may have symptom histories better accounted for by disorders such as borderline personality disorder (Zimmerman et al., 2019). These issues may arise as a result of the hypomania/mania criteria seemingly overlapping with internalizing (e.g., irritability with anxiety disorders), externalizing (e.g., impulsivity with personality disorders), and psychotic disorders (e.g., grandiose views with schizophrenia). For example, it may be challenging for diagnosticians to determine whether experiences of intense irritability are indicative of BSDs because elevated levels of irritability are listed as a criterion for many disorders and are commonly reported by many individuals seeking treatment irrespective of diagnosis (Stanton et al., 2019).

As a result of these challenges associated with diagnosing BSDs and other disorders from a *DSM*-based perspective, the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2020) has been proposed as an alternative to the *DSM* for improving diagnosis and understanding how various symptom experiences are interconnected. The HiTOP framework adopts a dimensional approach to conceptualizing psychopathology, recognizing that most symptoms are continuous rather than categorical in nature (e.g., experiences of irritability range from minor to very severe rather than present or absent). For example, from the HiTOP perspective, many symptom types defining *DSM-5* depressive, anxiety, and other disorders characterized by experiences of intense and/or persistent negative affect (e.g., sadness, pervasive worry, feelings of guilt) would be classified under a broad internalizing spectrum. The "hierarchical" descriptor for the HiTOP reflects that internalizing also is defined by sub spectra labeled fear and distress. Considering even more nuanced levels of specificity, the fear and distress sub spectra are defined by even more specific symptom dimensions (e.g., trembling, racing heart, and sweating are indicators of fear; worry and sadness are indicators of distress).

As an example illustrating the application of this approach, a clinician using the HiTOP model would identify dysphoric mood dimensionally as indicating distress-based problems associated with internalizing psychopathology (Ruggero et al., 2019); with the *DSM*-based approach, clinicians would seek to determine which *DSM* label or labels (e.g., different depressive disorders, PTSD) best reflect an individual's experiences of dysphoric mood, which can prove challenging. In addition to internalizing, other HiTOP spectra include thought disorder (e.g., positive symptoms of psychosis), antagonistic externalizing (e.g., callousness), disinhibited externalizing (e.g., irresponsibility), and detachment (e.g., aloofness).

Hypomania/mania is provisionally identified as a component of thought disorder due to its phenotypic and genetic overlap with *DSM* disorders such as schizophrenia. However, hypomania/mania (a) also overlaps strongly with the internalizing spectrum and (b) has features that distinguish it from other forms of psychopathology defining thought disorder (e.g., a more episodic course; being linked to reward hypersensitivity; Kotov et al., 2020). Thus, it is possible that some hypomanic/manic dimensions do not align neatly with the existing HiTOP structure and may reflect a hypomania/mania spectrum distinct from other spectra such as internalizing and thought disorder. Most studies examining the classification of hypomania/mania within the HiTOP have focused on composite, diagnostic ratings (i.e., *present* or *not present* ratings) which has precluded examinations of the degree to which different hypomania/mania symptoms converge to define the same spectrum of symptoms (e.g., irritability, but not other hypomania/mania symptoms, may define internalizing). Therefore, future efforts explicating the classification of hypomanic/manic symptom dimensions within comprehensive dimensional models are needed to inform clinical assessment.

Existing Measures for the Dimensional Assessment

Although research is needed to inform hypomania/mania assessment in the ways described, existing measures are available for dimensionally assessing hypomania/mania in a manner consistent with the HiTOP. For example, the Expanded Version of the Inventory for Depression and Anxiety Symptoms (IDAS-II; Watson et al., 2012) includes Euphoria

(5 items, e.g., "had so much energy") and Mania (5 items, e.g., "thoughts raced") scales that efficiently assess interrelated but distinct hypomania/mania dimensions. The Euphoria scale assessing high-arousal positive emotional experiences may be particularly useful for distinguishing hypomania/mania from overlapping internalizing or personality disorders (Stanton et al., 2019). Other relevant measures include the 48-item Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986; Schalet, Durbin, & Revelle, 2011), which assesses traits (e.g., excitability, self-perceived charisma) relevant to BD risk. Researchers frequently use HPS total score cutoffs to identify individuals at risk for hypomania/mania. More specific facet scales of Social Vitality (e.g., "persuade and inspire others"), Mood Volatility (e.g., "moods change easily"), and Excitement (e.g., "am a hyper person") also can be scored with the HPS items, with these facet scales appearing to show distinctive correlates in many ways (e.g., Social Vitality associates strongly with measures of social dominance, but Mood Volatility does not; Schalet et al., 2011).

It also should be noted that the HPS is only one example of dimensionally oriented assessment measures, as several other well-validated assessment tools also are available for efficiently assessing BD risk, such as the General Behavior Inventory (GBI; Depue et al., 1989), and current hypomania/mania symptoms in a dimensional manner (see Meyer et al., [2020] for a review).

Developmental Considerations

The onset of BD most commonly occurs in childhood or adolescence (Leverich et al., 2007; Perlis et al., 2004). BD in childhood and adolescence often presents with a severe and protracted course, including severe depressive symptomatology, mixed episodes, longer episode durations, psychosis, co-occurring psychopathology (e.g., substance use disorders, anxiety disorders, etc.), and psychosocial adversity (e.g., legal problems, academic impairment) (Birmaher et al., 2006; Cosgrove et al., 2013; Geller et al., 2002; Perlis et al., 2004; Saxena et al., 2020; Yapıcı Eser et al., 2020) compared with adults. More than 50% of adults with BD report onset of illness before age 18, while more than 20% identify age at onset before 13 (Perlis et al., 2004). Symptoms in youth may include dysregulation of emotion, heightened arousal when presented with emotional stimuli, and hypersensitivity to criticism (Peters et al., 2018; Weintraub et al., 2014). Major depressive episodes and cyclothymia are often harbingers of symptoms of mania. About half of all adults with BD I or II report that their first episode was major depression (Lish et al., 1994).

Issues surrounding the commonness of early-onset BD have been contentious in recent decades, associated with dramatic increases in reported community prevalence (Carlson & Glovinsky, 2009; Luby & Navsaria, 2010; Moreno et al., 2007). However, meta-analyses have repeatedly confirmed that rates of BD in youth are not increasing over time and are indeed similar across Western countries (A. R. Van Meter et al., 2011, 2019). While appreciable debate has persisted in recent years (Parry et al., 2018; A. Van Meter et al., 2019) regarding the legitimacy of the diagnosis, longitudinal studies have clearly documented the recurrent course of early-onset BD and its effect on functioning and quality of life (Birmaher et al., 2009; Chang, 2010)). It is clear that without early intervention, the socioemotional and intellectual growth of youth with BD may be jeopardized (Miklowitz, Schneck et al., 2020).

Among youth who have a parent with BD I or BD II, clinical presentation is often subthreshold, with some symptoms emerging many years prior to the onset of threshold BD (Shaw et al., 2005). Subthreshold symptomatology, in conjunction with a family history of BD, exacerbates child or adolescent vulnerability for progression to BD I or II unless interrupted by pharmacological and/or psychosocial intervention (West & Pavuluri, 2009). Risk of conversion from BD-NOS to BD I or II during a 4-year follow-up is substantial (i.e., 58%) for youth with a family history, compared to 32% for youth with no family history. BD in youth results from a complex interplay of genetic vulnerability, individual personality, and characteristics of a youth's family environment. For example, emotional dysregulation may intensify conflict within families and raise levels of expressed emotion in caregivers (i.e., high criticism, hostility, negative communication cycles), which can in turn exacerbate symptomatology in youth with BD (Miklowitz, Wisniewski et al., 2005). Adolescents with BD in low-conflict families experience more rapid symptom improvement during treatment than do those from high-conflict families (Sullivan et al., 2012; Sullivan & Miklowitz, 2010). In one seminal study, adolescents with BD from families high in expressed emotion showed longer times to symptom remission and were more symptomatic during a 12-month follow-up period than those from families low in expressed emotion (Miklowitz et al., 2013). Such

impairments have been proposed to be associated with deficits in social cognition (Keenan-Miller et al., 2012), with potential downstream implications for the development and maintenance of important peer relationships.

Adolescents with BD who experience chronic stress in family, peer, or romantic relationships tend to experience poorer symptomatic outcomes than those with lower levels of chronic stress (Kim et al., 2007). Although exposure to stressful environments is a risk factor for mood disorders, less is known about how adolescents with BD respond to stress. In both healthy and clinical samples of youth, responses to stress (i.e., active problem-solving and acceptance) have been found to be associated with positive mental health outcomes and academic and social competence (Compas et al., 2001).

Relevant Psychobiological Processes

In this section, we review relevant cognitive, emotional, neural, and circadian rhythm processes that are relevant to the onset, course, and profile of BSD.

Cognitive Processes

Aaron Beck's seminal cognitive model of emotional disorders (see Beck & Haigh, 2014, for a review) was one of the first theoretical frameworks to place cognitive styles (e.g., beliefs, ways of interpreting experiences) as central to the development and maintenance of psychopathology. Specifically, negative beliefs about the self, world, and others were linked to depression. This includes *dysfunctional assumptions*, which are negative "if-then" beliefs (e.g., "If I fail, then I am useless"). These can shape the way that someone with depression thinks about everyday situations and interacts with the world. As depressed mood is a common experience among people with BD, psychologists postulate that negative dysfunctional attitudes may also play a role in BD.

But what about mania (i.e., a diagnosis of BD)? This was traditionally viewed as "opposite" to depression (*bi*-polar). As such, mania has been theoretically linked to overly *positive* cognitive styles. For example, people with BD who had a higher "sense of hyper-positive self," characterized by excessive confidence and productivity, were more likely to relapse even after receiving cognitive therapy—potentially because they viewed the emerging symptoms of a (hypo)manic episode as desirable and due to their own positive attributes (Lam, Wright, & Sham, 2005). Heightened goal attainment beliefs (e.g., believing you need to be "outstanding") have been associated with current manic symptoms (Atuk & Richardson, 2020). In line with this, a body of work has focused on *goal dysregulation* and BD. This theory postulates that the behavioral activation system (BAS), which controls reward responsivity, is overly sensitive in BD. This oversensitivity means that people with BD find it difficult to regulate response to reward and goal-related events. Goal-relevant cognitive styles, including extreme aspirations for the future (particularly popular fame and financial success) and greater expectation of success, have been linked to BD (Johnson, Carver et al., 2012; Johnson et al., 2017).

Taken together, results suggest that both negative and positive cognitive styles are implicated in BD. Cognitive models of BD need to address the experience of mood fluctuation (highs and lows) that are part of BD. Cognitive theories of BD further suggest that the symptoms of BD manifest from making self-referent interpretations (or *internal* appraisals) of the mood changes linked to disrupted sleep (Jones et al., 2006). These appraisals can be negative (e.g., interpreting low mood as indicating failure) or positive (e.g., interpreting heightened mood and increased energy as indicating success and productivity). These themes overlap with dysfunctional attitudes (negative appraisals) and goal-relevant cognitions (positive appraisals). For example, if someone experienced increased energy and alertness after disrupted sleep and interpreted this experience as evidence of their own strengths (e.g., "I am full of good ideas and others are too slow"), they might then start to do more and sleep less, prompting an escalation of manic symptoms. There is evidence that internal appraisals are elevated in people with BD and relate to mood symptoms (Banks et al., 2016; Jones et al., 2006).

Building on these findings, the *integrative cognitive model* (ICM) of mood dysregulation (Mansell et al., 2007) proposed that positive and negative appraisals of internal experiences are central to the mood dysregulation characteristic of BD. In the ICM, these appraisals of changes to internal state are extreme, with personally significant meaning (e.g., "I have the energy to do anything I want"). To address the mood fluctuations seen in BD, the ICM contends that appraisals of high mood are not always positive, and appraisals of low mood may not necessarily be negative—in fact, those vulnerable to mood fluctuation have multiple, conflicting appraisals of how they are feeling. For example, high energy could be interpreted positively, with

excessive expectations of goal attainment and success, self-confidence, and optimism. However, the same internal state could also be appraised negatively, such as feeling the need to make excessive effort to avoid failure or taking a self-critical or catastrophic perspective (e.g., losing control). These beliefs are elevated in BD compared to both nonclinical and clinical controls and associated with mood symptoms (Dodd et al., 2011; Kelly et al., 2017). However, there is limited research on extreme appraisals of low mood derived from the ICM, especially around more positive appraisals of these experiences.

ATTENTION AND MEMORY

We have already seen that how people interpret their experience is important—but how do they come to notice certain aspects of their surroundings and situation in the first place? As with cognitive styles, do people with BD tend to notice particularly negative and positive aspects of their environment over more neutral aspects? Research findings are mixed and may depend on the context in which attention biases are measured as well as the current mood or clinical state of the BD participant. For example, some research indicates that people with BD who were not currently manic or depressed were no more drawn to positive or negative emotional stimuli than nonclinical controls (Purcell et al., 2018). However, another study of young adults at risk for developing BD did demonstrate an attentional bias for positive emotional stimuli (e.g., Gruber et al. 2021). It may be that there is an attentional bias to reward-relevant stimuli (Mason et al., 2016), and attentional biases may be present only during mood episodes (e.g., processing positive information faster when manic) but not a characteristic of BD more generally (Garcia-Blanco et al., 2013).

With respect to emotional memory, there is some evidence that people with BD have difficulties remembering both personal experiences and external events across childhood, adolescence, and adulthood (Bozikas et al., 2019). These deficits in episodic memory have been replicated across studies alongside impaired working memory—and there is some evidence that these difficulties are pronounced in more severe manifestations of BD (Cotrena et al., 2020). Furthermore, there is evidence that people with BD recall emotional stimuli more than do nonclinical controls (Fijtman et al., 2020). Linked to biases in attention and memory, vivid and highly emotive mental imagery may play a role in the mood dysregulation seen in BD. Rich, personally relevant images of the future could directly amplify mood or indirectly amplify mood by influencing thoughts and behavior, particularly in the context of goals and reward (Ivins et al., 2014; Di Simplicio et al., 2016).

In sum, the interplay between *negative* cognitive processes and overly *positive* cognitive processes is a defining feature of BD. The cognitive processes outlined here are not mutually exclusive. In fact, these processes are often studied together (e.g., Fletcher et al., 2014). In cognitive models of BD, appraisals of *current* affect are influenced by underlying beliefs about the self, world, and others which can be considered analogous to the dysfunctional attitudes outlined by Aaron Beck for depression. Cognitive styles are linked to cognitive emotion regulation strategies, which can amplify or dampen affect. Much in the same way as thoughts, personally meaningful, overly positive or negative mental imagery may prompt cognitive and behavioral responses that exacerbate mood symptoms. Goal-relevant appraisals could prompt increased goal-directed activity and sleeping less, which could in turn destabilize circadian rhythms and the BAS, intensifying the cycle of mood instability. Overall, there are multiple cognitive processes linked to BD, with a particular emphasis on how individuals appraise and respond to their experiences.

Emotional Processes

BD is centrally a disorder of emotion. Emotional processes in BSD have been characterized by heightened and persistent positive emotion *reactivity*, difficulty with positive and negative *emotion regulation*, and altered *emotional understanding* or perceptions of others people's emotions. With respect to emotion reactivity, prior work suggests that BSDs are associated with increased positive emotion reactivity that appears to persist across different types of emotional and nonemotional contexts (e.g., Gruber, 2011; Gruber et al., 2019). Specifically, individuals diagnosed with a clinical history of BD by a trained interviewer or who are at self-reported risk for future BD onset self-report greater positive feelings in response to a variety of laboratory stimuli, including short film clips, static pictures or images, and also monetary reward and positive feedback (e.g., Gruber et al., 2008; Gruber, 2011; Johnson, 2007; M'Bailara et al., 2009). In addition, young adults at self-reported risk for BD as well as adults with a *DSM* diagnosis of BD I have been found to exhibit elevated levels of parasympathetic activity

(as measured via respiratory sinus arrhythmia, for example) in laboratory studies while watching emotional film clips, interpreted as a physiological correlate of positive mood (Gruber et al., 2008; Gruber, Harvey, & Purcell, 2011). Some neuroimaging studies further suggest that people with BSDs exhibit greater neural reactivity in reward-related brain regions (such as the ventral striatum) and networks in response to rewarding stimuli (e.g., Dutra et al., 2015; Nusslock et al., 2011). More detail on neural processes in BSDs are described further below.

With respect to emotion regulation, it has been defined as the processes individuals engage to influence the intensity, duration, display, and type of emotions experienced, and these can occur consciously or automatically (e.g., Gross, 2015); research suggests that individuals with BSDs demonstrate difficulties with emotion regulation in the laboratory and everyday life when spontaneously (or naturalistically) regulating their emotions. Specifically, BD adults who are not currently manic or depressed (i.e., inter-episode) report less success effectively regulating their momentary emotions in everyday life using a 1-week experience-sampling period (e.g., Gruber et al., 2013). In the laboratory, inter-episode BD adults self-report more trouble successfully regulating the intensity of the emotions they experienced in response to watching brief, standardized positive and negative emotionally evocative film clips (Gruber, Harvey, & Gross, 2012). Furthermore, when asked which emotion regulation strategies they tend to utilize in their daily lives, inter-episode BD adults also report using emotion regulation strategies (such as rumination) that amplify mood states and impede problem-solving (e.g., Dodd et al., 2019; Gruber et al., 2011, 2012). This suggests that although BSD individuals might employ effort in regulating emotions but may ultimately use strategies that are less successful in managing intense mood states.

Despite these clear difficulties regulating emotions in BD, when inter-episode BD adults are instructed to follow specific regulation strategies in the laboratory, they demonstrate an intact ability to successfully decrease emotion intensity across a variety of strategies, including cognitive reappraisal, mindfulness, and cognitive distancing techniques (e.g., Gilbert et al., 2014; Gruber et al., 2009, 2012). Inter-episode adults with BD also successfully select or choose regulation strategies that match the context in a manner like healthy controls (Hay et al., 2015). Taken together, this suggests that BD individuals nonetheless have an intact ability to regulate their emotions successfully despite experiencing difficulties on their own in the lab and in daily life. The latter finding suggests important avenues for psychological therapies focused on assisting BD individuals in implementing these strategies successfully on their own.

Finally, when examining emotional understanding (or perception) in BD, research indicates that those with a BD diagnosis or subclinical BD risk or symptoms may have an inaccurate perception of other people's feelings. This bias in perceiving others' emotions has been found among young adults at risk for BD who self-report overestimating another person's positive emotions when they are describing a personal and negative life event in a brief film (e.g., Devlin et al., 2016) or during a difficult conversation with a romantic partner (Dutra et al., 2014). Other research suggests that BD adults who are currently manic may also be less accurate in detecting negative facial expressions (e.g., Lembke & Ketter, 2002). Further work is needed to understand whether those with BD may exhibit a positive bias toward perceiving the social world and identify putative underlying neural and related mechanisms.

Neural Processes

BD is characterized by alterations in brain systems that help us process threatening and rewarding stimuli in our environment, regulate our emotions, and consolidate memories. For example, individuals with BD display structural and functional alterations in the amygdala, a brain region implicated in processing threatening stimuli in the environment (Foland-Ross et al., 2012; Lopez-Jaramillo et al., 2017). Studies of neural connectivity, which examine the relations between brain regions, indicate that BD also is associated with structural and functional alterations in the coupling between the prefrontal cortex and the amygdala (Manelis et al., 2021; Phillips et al., 2008). This suggests that individuals with BD may have difficulty attenuating or regulating amygdala reactivity in the face of stressors, which also is consistent with behavioral, clinical, and self-report data. Finally, BD is associated with deficits in portions of the prefrontal cortex and the hippocampus important for executive control (e.g., behavioral inhibition, goal-directed behaviors, attention) and working memory (Phillips & Vieta, 2007).

Intriguingly, other psychiatric disorders show similar alterations in these brain systems. A heightened sensitivity to threatening stimuli and deficits

in executive control and working memory are present in major depression (Hamilton et al., 2012) and anxiety disorders (Etkin & Wager, 2007; Shackman & Fox, 2016), and schizophrenia is associated with abnormalities in both the prefrontal cortex (Radhu, et al., 2015; Selemon & Zecevic, 2015) and the hippocampus (Kalmady, et al., 2017; Smeland, et al., 2018). Such findings are helpful in identifying risk factors that cut across or are common to multiple psychiatric disorders. This work also can help break down potentially arbitrary distinctions between categorically defined psychiatric disorders and account for comorbidity among *DSM-5* categories. In addition to identifying risk factors that are common across disorders, initiatives such as HiTOP and the Research Domain Criteria (RDoC) also aim to identify mechanisms that are unique to specific psychiatric disorders and symptoms and that reflect signatures of differential risk for these symptom profiles. This is important for understanding and unpacking the within-disorder heterogeneity discussed above and for identifying clinically meaningful subtypes. We suggest that brain systems that help identify, pursue, and process rewards are important.

Although many regions in the brain respond to rewards, a cortico-striatal neural circuit involving the ventral striatum and orbitofrontal cortex (OFC) are at the heart of the reward system (Berridge, 2019; Haber & Knutson, 2010). Activation of this circuit is typically associated with positive or rewarding emotions and a desire to pursue rewards or goals in the environment, whereas deactivation leads to lower motivation and emotions such as sadness and anhedonia. Individuals with high levels of activity and connectivity in this circuit tend to be highly responsive to rewards in their environment and have heightened motivation to pursue these rewards and goals (Hahn et al., 2009; Simon et al., 2010).

As mentioned earlier, there is growing evidence that individuals with and at risk for BD display heightened sensitivity to rewarding stimuli and an increased motivation toward rewards and goals (e.g., Johnson et al., 2005, 2015; Nusslock & Alloy, 2017). This hypersensitivity to reward can lead to an excessive increase in motivation (e.g., working excessively long hours) during life events involving the pursuit or attainment of rewards, which, in the extreme, is reflected in hypomanic or manic symptoms or episodes. It also can lead to an excessive decrease in such motivation and reductions in goal-directed behaviors in response to losses or failures to attain a desired reward, which is reflected in bipolar depression. In line with this model, individuals with and at risk for BD display a heightened sensitivity to rewards on self-report, behavioral, and neurophysiological measures (Alloy, Bender et al., 2012; Gruber & Johnson, 2009; Harmon-Jones, et al., 2008; Johnson et al., 2017). A heightened sensitivity to rewards also predicts first onset and recurrences of BSDs (Alloy, Bender et al., 2012) and progression to more severe BD diagnoses (e.g., BD I) among individuals with milder variants of the disorder (e.g., BD II, cyclothymia) (Alloy, Urosevic et al., 2012; Nusslock, Harmon-Jones et al., 2012).

Functional magnetic resonance imaging (fMRI) studies provide partial support for elevated reward-related brain function in manic and euthymic (i.e., not currently manic, depressed or mixed) individuals with BD. These individuals display elevated activation in the ventral striatum (Hassel et al., 2008; Lawrence et al., 2004), OFC (Elliott et al., 2004), and amygdala (Bermpohl et al., 2009) to pictures of happy faces and pleasant stimuli compared to healthy controls (although see Liu et al., 2012, for evidence of decreased striatal and OFC activation in bipolar individuals to happy vs. neutral faces). A number of studies also report that individuals with BD display elevated OFC and ventral striatal activation to both monetary and social reward cues during manic and euthymic episodes (e.g., Abler et al., 2008; Bermpohl et al., 2010; Dutra et al., 2015; Nusslock, Almeida et al., 2012), although other studies have not observed this effect (Johnson et al., 2019; Trost et al., 2014; Yip et al., 2015). Finally, individuals with a diagnosis of BD II and individuals at elevated risk for BD who have not yet developed the disorder (i.e., hypomanic temperament) display elevated OFC and ventral striatal activation to reward cues (Caseras et al., 2013; Harada et al., 2019). This suggests that heightened reward-related brain function may reflect a preexisting risk factor for BD, as opposed to a consequence of the illness.

In contrast, a blunted sensitivity to rewarding stimuli, reduced motivation toward rewards and goals, and low positive affect have long been considered hallmarks of risk for unipolar depression (without a history of hypomania or mania) (see Nusslock & Alloy, 2017, for review). Individuals with major depressive disorder and at-risk offspring of depressed parents report a lower sensitivity to rewards and greater anhedonia (Kasch et al., 2002; Kazdin, 1989; Luby et al., 2004) and are less responsive to both the anticipation and receipt of rewards on neural and behavioral indices (Forbes et al., 2009; Olino et al., 2010; Pizzagalli et al., 2008; Ng et al.,

2019). In prospective studies, a low sensitivity to reward assessed with behavioral tasks, neurophysiology, and brain imaging predicts later depressive symptoms and episodes (Bress et al., 2013; Forbes et al., 2007; Morgan et al., 2013; Nelson et al., 2016; Nusslock et al., 2011). Furthermore, other conditions such as ADHD (Volkow et al., 2009) and schizophrenia (Smucny et al., 2021) are associated with a blunted neuronal response to rewards, which, in the case of schizophrenia, is likely implicated in the negative symptoms or motivational deficits of the illness (Whitton et al., 2015). Taken together, this suggests that BD is characterized by a profile of heightened reward-related brain function that distinguishes it from other psychiatric disorders including depression, schizophrenia, and ADHD. We propose that what differentiates BD from other psychiatric illnesses is mania, and one of the primary risk factors for mania involves a propensity to experience abnormally elevated energy and motivation. Thus, reward-related brain systems are clearly important for understanding what distinguishes BD from other disorders, whereas threat, executive control, and working memory processes may be more relevant for understanding what is common or transdiagnostic across these illnesses.

Circadian Rhythms and Sleep Processes

There is growing interest in sleep and circadian pathways in the pathogenesis of BD, with numerous reviews drawing together heterogeneous circumstantial evidence for cross-sectional, prospective, and causal links (e.g., Logan & McClung, 2019; McCarthy, 2016; Murray 2019; Takaesu, 2018). This brief section will outline evidence for circadian and sleep parameters as elements in the web of mechanistic pathways underpinning BD. Following some background about the structure, function, and measurement of circadian and sleep-wake systems, evidence for circadian mechanisms is critically introduced (focusing on genetic and behavioral levels of analysis).

STRUCTURE AND FUNCTION OF THE CIRCADIAN SYSTEM

Human biology is rhythmic. A complex network of biological clocks—the circadian system—coordinates this "predictive homeostasis," adapted to optimize fitness in the context of Earth's 24-hour light–dark cycle. The molecular basis of the circadian system is well characterized: intrinsic 24-hour rhythmicity is coordinated through clock genes that are responsible for generating circadian rhythms in physiology, behavior, and cognition (Mohawk et al., 2012). Example 24-hour rhythms with a known circadian component include core body temperature (nadir around 4:00 AM), melatonin secretion (commences around 9:00 PM), and alertness (peaking around 10:00 AM) (Refinetti, 2006). The master oscillator of the human circadian system is in the suprachiasmatic nucleus (SCN), a small structure with cells operating autonomously and as part of a network. A critical feature of the circadian system is its sensitivity to environmental cues, which adapts the system to be entrained daily to shifting times of sunrise and sunset (Reppert & Weaver, 2002). The sleep-wake cycle is sometimes described as the most obvious circadian rhythm in humans, but circadian function has a complex relationship with the sleep-wake cycle. Borbély's two-process model of sleep regulation (Borbély, 1980; Borbély et al., 2016) proposes that the circadian system regulates sleep timing and architecture in a bidirectional interaction with sleep homeostasis. Sleep homeostasis increases with wake time and dissipates with sleep, while the circadian system sends alerting signals during the day which decrease at night. Optimally, these two processes work together to promote wakefulness during the day and sleep at night (Fuller et al., 2006).

Importantly, growing evidence links circadian function to BD. At the genetic level, animal studies have been informative because core circadian genes (i.e., CLOCK genes) are strongly conserved across evolution. The *Clock*Δ19 mutant mouse, for example, provides a well-characterized animal model of mania, exhibiting increased dopamine transmission, hyperactivity, increased reward-seeking and impulsivity-like behaviors, and reduced depressive-like behavior. Intriguingly, these abnormalities are partly reversible by lithium (Dzirasa et al., 2010; Mukherjee et al., 2010; Roybal et al., 2007). *Clock*Δ19 mice encode a dominant-negative CLOCK protein, causing arrhythmic behavior under constant darkness and reduced amplitude, long period, and delayed phase under a light–dark cycle (Vitaterna et al., 1994). In human candidate gene studies, common polymorphisms in CLOCK have generally, but not universally, been found to associate with BD and related phenotypes (Benedetti et al., 2003, 2007; Lee et al., 2010; Soria et al., 2010).

Given the broad acceptance of chronobiologically informed behavioral interventions for BD (Gottlieb et al., 2019), it is not surprising that numerous studies have used behavioral methods to explore the possibility of chronobiological correlates of observable behavior. The most-studied behavioral

variables are those derived from actigraphy-based measurement of locomotor activity and self-reported chronotype (Murray et al., 2020). Lower 24-hour activity compared to healthy populations has been found in people with BD (De Crescenzo et al., 2017; Scott et al., 2017; Shou et al., 2017), and people with BD also appear to have less robust (Jones et al., 2005; Rock et al., 2014), more unstable (Krane-Gartiser et al., 2016), and phase-advanced 24-hour activity rhythms (Salvatore et al., 2008). In people with BD, differences across the phases of the illness can also be observed. The manic phase of BD appears to be characterized by more disorganized and complex patterns of activity, particularly in the morning, whereas the depressive phase of BD appears to be characterized by higher minute-to-minute variability (Krane-Gartiser et al., 2014). On average, euthymic BD individuals report being of evening chronotype and exhibit delayed physiological measures of circadian phase (Kanagarajan et al., 2018; Melo et al., 2017; Nurnberger et al., 2000). For example, a review by Melo et al. (2017) identified 15 studies using the Composite Scale of Morningness, most of which found BD to be associated with eveningness. This association with self-reported eveningness chronotype has been corroborated in actigraphically measured chronotype (Gershon et al., 2018).

Consistent with emerging dimensional approaches to psychopathology (e.g., HiTOP, see above), circadian vulnerability is probably not specific to BD. A recent international task force review highlights that circadian abnormalities are present in the phenotypes of various psychiatric disorders, with evidence strongest for BD, major depression, and schizophrenia (McCarthy et al., 2021). For example, polygenic scores in genome-wide association studies (GWAS) for morningness (the trait of preferring activities to commence relatively earlier in the day) are negatively associated with presence of all three of these disorders (Hu et al., 2016; Jones et al., 2019; Lane et al., 2016), while polygenic scores for low amplitude are associated with BD as well as the general vulnerability trait of neuroticism (Ferguson et al., 2018; Lyall et al., 2018). In cell lines and postmortem brain, miRNAs-29a/c and 106b have been linked to the circadian clock and have been independently linked to BD as well as to major depression and schizophrenia (Geaghan & Cairns, 2015). In sum, there is great interest in the chronobiology of BD, and there is a broad range of evidence pointing to an array of circadian abnormalities that may be important in the pathogenesis of BD.

Context and Social Environment Influences

In recent years researchers have begun to better understand how and why the social environment is important in BSDs. We review the literature on social functioning and life events in BD to better appreciate the real-world and social contexts individuals with BD face and its influence on symptom presentation and course.

Social Context and Functioning

A large body of work has documented associations between BSDs and lower social functioning and social support (Johnson et al., 1999; Castanho de Almeida Rocca et al., 2008) as well as increased social conflicts (Romans & McPhearson, 1992). People with a diagnosis of BD self-report having less contact with friends compared to both people with major depressive disorder and nonpsychiatric controls (Johnson et al., 1999). In one qualitative study, Michalak and colleagues (2006) reported that many individuals with a diagnosis of BD noted that they had lost friendships and social connections due to their illness, especially during manic or hypomanic episodes. Furthermore, lower social support was a significant risk factor for lifetime recurrence of mood episodes in BD (Cohen et al., 2004). At the same time, early clinical observations among individuals with BD suggests increased social interest and motivation among adults with BD (Goodwin & Jamison, 2007). Furthermore, Ong, Zaki, and Gruber (2017) found individuals with BD who were currently interepisode also were more cooperative on a standardized computer task compared to those with a history of major depression as well as those without a psychiatric history. Taken together, this suggests that, despite some work highlighting increased social interest and cooperative behavior, social functioning and quality of social relationships is frequently impaired among adults diagnosed with BD.

Life Events and Context

Both positive and negative life events have been found to predict the course of BD, generally with negative, stressful, and traumatic life events predicting a more severe and impairing course of the disorder broadly (Agnew-Blais & Danese, 2016; Daruy-Filhod et al., 2011; Koenders et al., 2014; Lex et al., 2017) and with positive and goal attainment life events predicting manic episodes (Alloy et al., 2008; 2009; Johnson et al., 2000, 2008).

Negative and stressful life events play an important role in the onset and course of BD. Research

in this area has examined adverse events that occur early in life prior to the onset of the disorder, and those that occur after onset in relation to the timing of subsequent mood episodes. Many empirical studies and reviews and meta-analyses of these investigations have consistently found that childhood maltreatment is associated with a more severe course of illness for those with BD (Agnew-Blais & Danese, 2016; Daruy-Filhod et al., 2011; Garno et al., 2005; Leverich & Post, 2006; Sala et al., 2014). For example, Daruy-Filho and colleagues (2011) found that childhood maltreatment was strongly associated with early onset, substance abuse, and suicidality among those with BD. A more recent meta-analysis of 30 studies compared individuals with BD and childhood maltreatment (abuse, neglect, or family conflict) to those with BD without childhood maltreatment (Agnew-Blais & Danese, 2016). Results indicated that those with histories of childhood maltreatment had more severe and more frequent episodes of mania and depression, more severe psychosis, higher risk of suicide attempts, and higher risk of comorbid diagnoses including PTSD, anxiety disorders, and substance use disorders.

Stressful life events occurring after disorder onset have also been associated with more frequent and severe mood episodes. Lex et al. (2017) reported that stressful life events occurred more frequently preceding acute mood episodes compared to euthymic periods. A prospective study that followed hospitalized individuals with BD prospectively for at least 1 year found that those who experienced severe negative life events took more than three times as long to achieve recovery (defined as minimal or absent symptoms for 2 consecutive months) compared to those without severe life events (Johnson & Miller, 1997). Similarly, in a study of BD outpatients assessed quarterly for 2 years, negative life events were associated with the subsequent severity of both mania and depression, as well as with higher levels of functional impairment (Koenders et al., 2014). These data suggest that stressful life events are associated with a more severe course of BSDs.

Positive life events have also been consistently found to predict subsequent increases in mania symptoms, though the boundary conditions around which types of events are most associated with mania onset and how these should best be conceptualized are slightly less clear. One line of work in BD has focused on life events involving goal striving or attainment, based on prior evidence discussed above that those with BD self-report greater levels of ambitious goal setting and striving (Alloy, Bender et al., 2012; Gruber & Johnson, 2009; Johnson, 2005; Johnson, Eisner, & Carver, 2010). For example, Johnson and colleagues (2000) followed individuals with BD I monthly over a 2-year period and found that mania symptoms increased in the 2 months following events involving goal attainment. Notably, there was no relationship between goal attainment events and subsequent symptoms of depression. In a larger prospective study, Johnson and colleagues (2008) found that goal attainment life events predicted increases in mania symptoms. More recently, Tharp et al. (2016) found that when BD I individuals were asked to describe their goals in a laboratory session, the goals described by the BD group were objectively rated as more difficult to achieve compared to healthy control participants, and these ambitious goals among the BD participants were found to predict increased mania over time. These findings are consistent with work described earlier in this chapter suggesting that BSDs involve greater sensitivity to reward and goal striving and attainment events, which may predispose BSD individuals toward increased mania and hypomania symptoms (Alloy et al., 2009; Bender et al., 2010; Urošević et al., 2010).

Taken together, this research demonstrates a clear relationship between life events and the subsequent course and severity of BD. Negative and potentially traumatic life events appear to portend shorter time to onset of new symptoms, more severe symptoms, and higher rates of comorbidity broadly. Positive events, particularly those related to goal attainment and/or engagement of the BAS system, are associated with increases in mania symptoms prospectively. With these patterns established, researchers have largely turned toward better understanding the neuroanatomical and neurochemical correlates of these relations (e.g., Hanford et al., 2019; Sato et al., 2018) and to identifying risk and protective factors that may serve to moderate the relations between life events and illness course (e.g., Chan & Tse, 2018; Ng et al., 2016; Stange et al., 2012, 2013).

Psychotherapy and Prevention

Compared to other mental health problems such as anxiety or unipolar depression, research on the effectiveness of psychosocial treatments in the context of BD is still young and only recently was expanded upon in the 1990s. The need for psychological treatments has been made apparent by findings that BD patients, even when treated pharmacologically, report subsyndromal symptoms,

functional impairments in everyday life, and recurrent mood episodes (e.g., Goldberg & Harrow, 2011; Treuer & Tohen, 2010). Psychological approaches evaluated in sufficiently statistically powered randomized controlled trials (RCTs) can be grouped into four classes: psychoeducation, interpersonal and social rhythm therapy (IPSRT), cognitive-behavioral therapy (CBT), and family and conjoint interventions.

Psychoeducation refers to an interactive therapeutic situation in which patients and/or relatives discuss with a mental health professional the disorder, its causes and course, and treatment options that integrate and build on the individual experiences of the patient (and sometimes their loved ones). Psychoeducation has also been widely evaluated as a stand-alone intervention, within both individual and group settings (e.g., Candini et al., 2013; Colom et al., 2003; Perry et al., 1999). Colom and colleagues (2003, 2009) were able to demonstrate that their 21-session program had protective effects against recurrence of mood episodes for as long as 5 years. Additionally, the feasibility of effectively delivering psychoeducation within community mental health teams was assessed. It was evidenced that, while there was a nonsignificant advantage of psychoeducation with regards to recurrence risk, psychoeducation significantly increased social and occupational functioning (i.e., Lobban et al., 2010). At this point, psychoeducation has shown to be beneficial as a stand-alone intervention; however, all the previously described approaches consider psychoeducation as an integral part of psychological therapy for BD.

IPSRT (Frank, 2005) integrates interpersonal therapy (Klerman et al., 1984) with therapeutic strategies aimed at stabilizing circadian and social rhythms (Frank, 2005); it is rooted in the social *zeitgeber* model (Ehlers et al., 1988). IPSRT postulates three core mechanisms to reduce symptoms: medication adherence, stable social rhythms and daily routines, and improved interpersonal functioning. Frank and colleagues (1999, 2005) were able to illustrate that acutely symptomatic patients with BD who had received IPSRT in the acute phase had less recurrences over time than those who had received an intense clinical management, regardless of what treatment they were assigned to during the maintenance phase of their treatment. Additionally, IPSRT was considered for use as a stand-alone therapy under specific circumstances (Swartz et al., 2018). For depressed patients with BD II, they were able to provide equivalent effectiveness of IPSRT to quetiapine with regards to the outcome, and differences were only noted in the time to response and side-effect profile.

CBT, originally developed for unipolar depression (Beck, 1991), was also adapted for BD, and several manuals have been published on this treatment option (e.g., Lam et al., 2010; Meyer & Hautzinger, 2013; Newman et al., 2002). A case formulation from a CBT perspective interprets manic and depressive symptoms reflecting dysfunctional changes in behavior, thoughts, and emotions. The likelihood of recurrent mood episodes is therefore hypothesized to be achieved by targeting underlying dysfunctional attitudes, beliefs, behavioral habits, and cognitive errors (e.g., all-or-nothing thinking, overgeneralizations) in therapy. While research has shown that patients with BD show similar cognitions as depressed patients, a few are more specifically related to (hypo)mania (e.g., Johnson, Carver, & Gotlib, 2012; Lex et al., 2011; Shapero, et al., 2015; van der Gucht et al., 2009). Traditionally, CBT works toward a shared case formulation of individual problem areas and treatment goals based on psychoeducation and the derived individual relapse signature, which includes, for example, prior course of the disorder, early warning symptoms of mood episodes, and identified triggers (e.g., life events, interpersonal conflicts, changes in daily routines). The case conceptualization will inform what behavioral and cognitive strategies might be most helpful to cope with future emerging mood symptoms. Tailored to the individual, modules focus on problem-solving, communication skills, or stress reduction strategies, if there is indication that such additional skills will be beneficial to prevent the development or maintenance of mood symptoms. More recently, the focus of CBT has shifted from focusing primarily on reducing symptoms and preventing recurrence to highlighting individual recovery (e.g., Jones et al. 2015; Murray et al., 2017) and incorporating techniques related to mindfulness and acceptance (e.g., Williams et al., 2008).

Several studies have compared outcomes between individuals receiving CBT and individuals in no therapy (waiting list), treatment-as-usual, and supportive therapy groups (e.g., Lam et al., 2003; Meyer & Hautzinger, 2012; Parikh et al., 2012; Scott et al., 2001; Zaretsky et al., 2008). A review of these studies shows the evidence for CBT to be mixed, which is not surprising considering the variety of control conditions, primary outcome variables, and sample composition. For example, Lam and colleagues (2003) demonstrated that CBT reduced risk for relapse in a sample of euthymic BD patients (i.e.,

not currently manic, depressed, or mixed); however, the effects diminished during the second year of follow-up (see Lam et al., 2005). Conversely, a study involving remitted and acutely ill patients found that, while CBT did not affect recurrence rates overall, when the number of episodes were taken into account, those with fewer prior mood episodes did benefit from CBT while those with a longer illness history did not (Scott et al., 2006). While the latter two studies used treatment-as-usual as the control condition, Meyer and Hautzinger (2012) compared CBT with supportive therapy matched in frequency and intensity of sessions, and no differential effects were found aside from a trend of CBT outperforming supportive therapy during active treatment but not during follow-up. Considering effect sizes, most studies overall revealed positive effects on subsyndromal symptoms and indicators of improved psychosocial functioning (e.g., Szentagotai & David, 2010).

Family and conjoint interventions rely on diverse approaches; what they have in common is the setting in which they occur (i.e., not *individual* therapy). The approach that has received the most attention and empirical support is family-focused treatment (FFT; Miklowitz, 2010). Miklowitz and colleagues developed this treatment from research demonstrating that family communication styles and family functioning are associated with the course of BD (e.g., Miklowitz et al., 1988; Perlick et al., 2004); thus, targeting potential family processes directly or indirectly related to BD is a promising avenue for patients with BD, their significant others, and families. Several RCTs showed the efficacy of FFT in reducing risk of recurrent mood episodes (e.g., Miklowitz et al., 2003; Rea et al., 2003). Adaptations to specific groups such as adolescents (e.g., Miklowitz et al., 2008) or caregivers (e.g., Perlick et al., 2010) and to different healthcare contexts (e.g., Sharma et al., 2020) have been developed as well.

When acknowledging the whole range of adjunctive psychological interventions for BD, most systematic reviews and meta-analyses conclude that they reduce recurrence of mood episodes as well as improve symptoms and/or psychosocial impairment (e.g., Chatteron et al., 2017; Macheiner et al., 2017; Miklowitz & Scott, 2009; Schöttle et al., 2011). Some reviews note that the field is still in its early stages and that the evidence base is only low to moderate (e.g., Lynch et al., 2010; Oud et al., 2016; Szentagotai-Tătar & David, 2018). However, all these meta-analyses have never compared the relative efficacy of those psychosocial treatments with each other and only considered the active treatment relative to its control condition. This gap in the literature was addressed by a network meta-analysis recently published by Miklowitz, Efthimiou, and colleagues in 2020. These authors identified a total of 39 RCTs, including 3,863 participants, and their network analyses unveiled that empirically supported treatments were associated with lower recurrence rates than control treatments. Additionally, CBT provided the strongest evidence for reducing bipolar depressed symptoms when compared to treatment as usual, and to some extent this was also true for FFT and IPSRT (similar effects were found for manic symptoms and CBT but with lower certainty). The authors even identified that certain treatment elements were differentially related to outcome; for example, psychoeducation in a group format or involving family members, as well as use of mood monitoring, were more effective than individual psychoeducation in preventing recurrence. Contrarily, cognitive restructuring and regulating daily activities were the most potent strategies to reduce depressive symptoms (Miklowitz, Efthimiou et al., 2020).

There are still major gaps in our knowledge about mechanisms, mediators, and moderators of outcome, and our understanding is only further complicated in recognizing that most studies focus on symptoms, relapse, and recurrence only in patients after discharge from a hospital or when already stable. Fewer studies have looked at remission from acute symptoms (see the STEP-BD study; Miklowitz et al., 2006) or specifically targeted comorbidities such as substance use disorders (e.g., Crowe et al., 2020). It is also unclear how to best address coexisting mental health conditions in BD. Should they be treated sequentially or simultaneously as part of an integrative case formulation, or would a transdiagnostic approach be most appropriate? In addition, the typical primary outcomes chosen in RCTs, such as symptomatic remission, are often for patients themselves less important than functional or personal recovery (e.g., Jones et al., 2015; Murray et al., 2017).

Looking Ahead: Advancing Work on Diversity and Anti-Stigma Approaches

In this final section we review research highlighting critical work needed in BSDs that includes expanding our understanding in diverse and underrepresented populations and addressing the stigma that still shrouds this disorder.

Most of the current research discussed in this chapter has been conducted in Westernized countries. Even within the United States, research is limited by homogeneous samples consisting of primarily White, wealthy, and highly educated individuals. Research with the largest percentages of Black, Indigenous, people of color (BIPOC) individuals with BD tends to be hospital and prison studies, whereas undergraduate and community samples are composed of limited non-White individuals. These differences in participant characteristics and clinical severity by recruitment methods influence research questions and conclusions. For example, Johnson and Johnson (2014a) found that the prevalence of BD was correlated with reward-relevant cultural dimensions such as power distance and individualism in a cross-national study of 17 countries. Furthermore, insufficient recruitment of BIPOC individuals precludes analyses of potential racial and ethnic differences. Instead, race and ethnicity are typically "controlled for" and included as covariates in analyses—or worse, non-White participants are excluded to keep samples homogeneous, particularly in terms of genetic studies (Akinhami et al., 2017). These suboptimal research practices result in typifying White Western adults with BD as the framework for universal presentations of bipolar phenomenon. Thus, we have limited knowledge of the symptomatology, etiology, and treatment of BD in other, non-Western cultures and within minoritized racial and ethnic groups in the United States.

Research on serious mental illness (e.g., schizophrenia spectrum disorders, BSDs) suggests there may be critical differences across cultures and racial/ethnic groups in terms of symptom presentations, diagnostic considerations, and treatment efficacy that could inform more tailored and effective interventions for diverse populations. In a 2011 review of more than 50 articles in the United States and the United Kingdom, Haeri and colleagues concluded that there are racial disparities in the diagnosis of BD. Across multiple studies, current literature indicates that Black individuals are less likely to be diagnosed with an affective disorder (including depression and BD) and more likely to be diagnosed with schizophrenia spectrum disorders compared to White individuals. Furthermore, Kilbourne and colleagues (2005) found that African American veterans with BD were less likely to receive adequate follow-up outpatient care compared to White veterans. In a representative US sample drawn from the National Comorbidity Survey Replication (NCS-R) data, Johnson and Johnson (2014b) found that no African Americans (0%) received minimally adequate treatment (defined as use of a mood stabilizer alone or in combination with an antipsychotic) in the past year, compared to 17% of Caucasian Americans who did. A similar trend was found for Latinx Americans compared to non-Hispanic whites (0% vs. 21%; Salcedo et al., 2017). In addition, previous studies have demonstrated that Black individuals are more likely than White individuals to be prescribed antipsychotics with more severe side effects, irrespective of the presence of psychotic symptoms (e.g., Kilbourne & Pincus, 2006; Szarek & Goethe, 2003). Overall, mounting evidence has indicated racial disparities in the diagnosis and treatment of BD.

Haeri et al. (2011) identified potential factors that may contribute to observed racial disparities in diagnostic rates in BD. For example, there is some evidence for differences in BD symptom presentation, with Black individuals experiencing higher rates of hallucinations and delusions compared to Whites (e.g., Gonzalez et al., 2007; Kirov & Murray, 1999; Strakowski et al., 1996). Some studies have also found higher rates of exclusively or mainly manic presentations (without a prior history of depression) in Black BD patients including British outpatients of African origin (Kirov & Murray, 1999) and Yoruba Nigerian patients (Makanjuola, 1985). These findings contrast with the much lower rates of mania-only BD presentations reported in primarily White Western samples (Boyd & Weissman, 1981). At the same time, differences in symptom presentation of psychosis may be due to differences in treatment seeking and poorer access to healthcare among Black individuals, including an overreliance on emergency room services (Snowden, 2001) and a tendency to seek treatment at later, more severe stages of mania when psychosis is more likely to occur (Mukherjee et al., 1983). There is also some evidence for differences in how clinicians weigh the same symptoms in Black versus White individuals. In a study of US adult psychiatric inpatients, the presence of negative symptoms (e.g., blunted affect, monotonous speech) in African American patients—but not in White patients—was found to contribute more toward a diagnosis of schizophrenia and away from a diagnosis of BD (Neighbors et al., 2003; Trierweiler et al., 2000). The authors suggested these findings may indicate a tendency for clinicians to overpathologize a reluctance to disclose personal information and a general mistrust of the healthcare system that is common in Black communities. In addition to potential characteristics of

the individual with BSDs, other researchers have also proposed clinician biases as a contributor to the racial disparities in diagnosis and treatment for BSDs. While racial biases on the part of healthcare workers have been demonstrated in the medical field (e.g., Chapman et al., 2013), little research in mental health providers—or in clinicians diagnosing BD, in particular—has been conducted, and this remains a critical focus for future work (McMaster, 2016).

In sum, research is limited in terms of its ability to characterize BS cross-culturally and in racially and ethnically diverse communities. As discussed in previous sections of this chapter, accurate diagnosis of BD is critical to improving prognosis and treatment responsivity. Thus, an important avenue for future research is to examine BD in multiple sociocultural contexts and across more racially, ethnically, and socioeconomically diverse populations to promote equity of BD treatment and ensure that research findings are applicable to various individuals living with BD. Furthermore, the current literature highlights the importance of bolstering clinician experience with various sociocultural contexts pertinent to the patients they serve. It is likely that such endeavors will require greater community engagement to build partnerships and trust within BIPOC communities traditionally excluded from and exploited by clinical science (Akinhanmi et al., 2017).

Mental Illness Stigma

Here we focus on the stigma incurred by people with BD. The term "stigma" has a long (and distressing) history in cultural studies, human evolution, social and clinical psychology, and psychiatry (for a crucial review, see Kurzban & Leary, 2001). It literally refers to the sharp instruments used, in Greek and Roman times, to cut or burn visible marks into the skin of members of devalued societal subgroups—with the goal of making visible the denigrated status of traitors, former slaves, or diseased individuals. Ancient texts reveal that individuals displaying features of mental illness have received stigma, been shunned or banished, and even murdered throughout recorded history (Hinshaw, 2007). Although visible marking still occurs (e.g., concentration camp inmates with numbers tattooed on their wrists; the marking of HIV-positive individuals in several nations during the 1980s), "stigma" today is inferred mainly from one's group status, thus comprising a social-psychological "brand." Our postindustrial world includes far greater factual knowledge of mental illness than a generation or two ago, yet those experiencing mental disorders still receive and experience large amounts of stigmatization (Pescosolido et al. 2010).

Heuristic conceptual models of stigma as related to mental disorders include Link and Phelan (2001) and Pescosolido, Martin, Lang, and Olafsdottier (2008); also see the review by Martinez and Hinshaw (2016). In brief, the naturally selected propensity to define others based on key observable or inferred characteristics, called *stereotyping*, may lead to prejudice when the "other" is deemed as threatening. Discrimination, the abridgment of rights devalued individuals, is a common result, even comprising extermination. Stigma incorporates all three processes, also signaling the loss of individuality (or even fundamental humanity) of the derogated person.

Most stigmatized individuals know of the stereotypes that exist about them. As a result, they may internalize the stigma (Corrigan & Watson, 2004). For mental health conditions, such *internalized stigma* (self-stigma) is a major barrier to help-seeking, above and beyond symptoms such as anxiety or depression. Moreover, as defined by Goffman (1963), *courtesy stigma* signifies the tendency of the public to stigmatize anyone even associated with the stigmatized individual. Parents thus experience a double dose of stigma, also having been believed to directly cause their offspring's mental illness for most of the twentieth century. Clinicians and researchers also receive courtesy stigma, because they, by definition, treat or investigate members of devalued groups.

In general, the most severe forms of mental illness—particularly when psychotic features are involved—receive especially high levels of stigma (Jones et al., 1984). Extreme states of mania and/or depression are clearly involved. Even more, BD is susceptible to societal stereotypes (Jekyll vs. Hyde; "You're just acting bipolar" [i.e., in inconsistent fashion]), often fueled by pervasive and biased media depictions.

In an essential review, Hawke, Parikh, and Michalak (2013) found that stigma related to BD is strong, in terms of self-stigma and public stigma, within school and employment-related settings and across healthcare systems in general. Consequences (reduced quality of life and increased functional impairment) are pervasive. On the other hand, Ellison, Mason, and Scior (2015) surveyed more than 700 UK residents regarding attitudes toward

an individual depicted as having BD. Explicit attitudes were generally positive, especially if respondents believed that the conditions in question were biogenetic in origin and if they had experienced prior contact with someone dealing with BD. Moreover, beliefs that BD can signal creativity and positive attributes were linked to positive attitudes, potentially fueled by greater levels of disclosure by influential individuals. Yet it is not clear whether such explicitly stated attitudes reflect a deeper and less conscious set of reactions to people with BD (i.e., implicit attitudes). As well, a genetic or biochemical ascription for serious mental illness typically reduces blame on the part of the observer but at the same time increases beliefs about permanence, hopelessness, and propensities toward aggression or violence (Kvaale et al., 2013). Thus, the key solution for stigma is not simply to promote a reductionistic disease model; rather, humanization is part of the overall strategy (see Hinshaw, 2017).

The sheer inconsistency of performance in individuals with BD may be crucial. Although severe and chronic medical or psychiatric conditions receive high stigma (e.g., HIV is more stigmatized than the flu), when the public encounters someone with highly inconsistent performance—for example, a child or adult with ADHD (Nguyen & Hinshaw, 2020) or an individual fluctuating between phases of manic and depressive episodes—the perception may well be of a lack of effort or will. Such ascriptions of controllability, potentially linked to weak character or low moral fiber, are highly likely to fuel stigmatizing responses.

In terms of additional findings, Gilkes, Perish, and Meade (2019) examined self-stigma in 275 adults with BD, discovering that high self-stigma was associated with unmarried status (potentially indicating low social support) and severity of symptoms. Budenz et al. (2020) explored the nature of more than 1 million Twitter posts (and retweets) in 2016–2017. Although many general mental illness–related tweets were positive in nature, those related to BD were far less so. Also, in a provocative qualitative investigation, Richard-Lepouriel, Favre, Jermann, and Aubry (2020) analyzed complex self-reported themes among individuals with BD regarding phases of intensive self-stigmatization that eventually led to more self-accepting/destigmatizing stages. Despite the small sample size, it is inspiring to examine how individuals with BD can progress from self-negation to acceptance as a function of social support and self-enhancing attitudes.

Conclusion

In all, as emphasized throughout this chapter, BSDs incur high levels of impairment and features a tragic level of suicidal behavior. Although significant advances have been made towards understanding emotional, cognitive, and neural processes as well as empirically supported treatments for BSDs, positive features of the disorder clearly exist, and public stigma may be improving, public acceptance does not appear to be at optimal levels, and levels of self-stigma remain far too high. Future work is needed to expand our scientific understanding, treatment advancements and dissemination, and compassion towards those with lived experience of BSDs.

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