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Cognitive deficits in bipolar disorders: Implications for emotion

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Abstract

Prominent cognitive deficits have been documented in bipolar disorder, and multiple studies suggest that these deficits can be observed among non-affected first-degree relatives of those with bipolar disorder. Although there is variability in the degree of cognitive deficits, these deficits are robustly relevant for functional outcomes. A separate literature documents clear difficulties in emotionality, emotion regulation, and emotion-relevant impulsivity within bipolar disorder, and demonstrates that these emotion-relevant variables are also central to outcome. Although cognitive and emotion domains are typically studied independently, basic research and emergent findings in bipolar disorder suggest that there are important ties between cognitive deficits and the emotion disturbances observed in bipolar disorder. Understanding these relationships has relevance for fostering more integrative research, for clarifying relevant aspects related to functionality and vulnerability within bipolar disorder, and for the development of novel treatment interventions.

Bipolar disorder (BD) is a severe psychiatric illness that has been ranked as one of the 20 leading medical causes of disability (WHO, 2011). BD has been shown to be the psychiatric disorder with the highest rates of completed suicide across two major cohort studies (Ilgen et al., 2010; Nordentoft, Mortensen, & Pedersen, 2011). In a cross-national representative sample, one in four persons diagnosed with bipolar I disorder reported a suicide attempt (Merikangas et al., 2011). Rates of relapse remain high despite available treatments (Gitlin, Swendsen, Heller, & Hammen, 1995), and in the year after hospitalization for manic episode, two-thirds of patients do not return to work (Strakowski et al., 1998). Poverty, homelessness, and incarceration are all too common (Copeland et al., 2009).

Despite the often poor outcomes, there is also evidence for outstanding accomplishments and creativity among those with milder forms of the disorder and their family members (Coryell et al., 1989; Jamison, 1993; Murray & Johnson, 2010). Some individuals appear to achieve more than the general population, suggesting the importance of understanding the variables that predict differential outcome within bipolar disorder.

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Contributors

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Conflict of interest

All authors declare that they have no conflicts of interest.

Within this paper, we focus on two key predictors of outcomes within bipolar disorder: cognition and emotionality. We review evidence that problems in cognition and emotionality are prominent among those diagnosed with the disorder, are not artifacts of symptom state, and relate substantively to poorer outcomes. Although traditionally studied separately, new work points toward the idea that cognition and emotionality are intricately linked within bipolar disorder. Drawing from research within bipolar disorder as well as outside of bipolar disorder, we build a model of how cognition and emotionality might be tied within bipolar disorder. We then provide suggestions for future research.

Before considering findings, it is worth noting that there are several forms of the disorder, defined by varying degrees and duration of manic symptoms (APA, 2013; WHO, 1993). Manic episodes are defined by abnormally elevated or irritable mood, accompanied by increased activity and at least three symptoms (four if mood is only irritable) such as decreased need for sleep, increased self-confidence, racing thoughts or flight of ideas, rapid speech, distractibility, goal-directed activity, and engagement in pleasurable activities without regard to potential negative consequences. To meet criteria for mania, these symptoms must persist for at least one week or require hospitalization, and must lead to difficulties with functioning. If functional impairment is not more than mild and duration is between 4 and 6 days, the episode is considered a hypomanic episode. Bipolar I disorder (BD I) is diagnosed on the basis of at least one lifetime manic episode within the DSM-5 and by at least two episodes within the ICD, whereas bipolar II disorder is diagnosed on the basis of at least one hypomanic episode (and no manic episodes) as well as major depressive episodes. Cyclothymic disorder is defined by chronic but milder fluctuations between manic and depressive symptoms. Most research focuses on BD I.

In addition to diagnosed samples, research has focused on those at high risk for bipolar disorder, including first-degree relatives of those with BD. This work draws on the evidence for extremely high heritability of BD I, with estimates from community-based twin studies of 0.85 (Kieseppä, Partonen, Haukka, Kaprio, & Lönnqvist, 2014). Other research has considered high risk for BD by virtue of lifetime subsyndromal symptoms, as measured by scales such as the Hypomanic Personality Scale (Eckblad & Chapman, 1986) or the General Behavior Inventory (Depue, Krauss, Spoont, & Arbisi, 1989). The study of high-risk individuals provides a way to decipher whether deficits are present before the onset of the disorder, of importance given models suggesting that episodes of the disorder may change brain function (Chang, Steiner, & Ketter, 2000; Strakowski, 2012) as well as individuals' perceptions of their emotion regulation.

Beyond defining BD, it is worth defining some of the many different neuropsychological tasks that have been widely studied in BD. Perhaps no area has received more attention than executive function. Executive function is related to three core functions: 1) inhibition, the ability to suppress irrelevant information in working memory in order to accomplish an established goal; 2) working memory, the ability to hold and manipulate information in mind; and 3) cognitive flexibility, the ability to shift strategies in response to feedback (Diamond, 2013; Miyake et al., 2000). Attention (defined as the process of selecting information reception from internal or external cues) is implicated in all three of these aspects of executive function. Much of the literature we will discuss focuses on response

inhibition, or the ability to suppress a prepotent response, which is considered a subtype of inhibition. Some tests measure multiple facets of executive function; for example the Trails B test likely requires working memory and cognitive flexibility (Sánchez-Cubillo et al., 2009).

Aside from executive function, multiple other facets of cognition have been widely studied in bipolar disorder. Verbal and non-verbal memory are related to the ability to register, store and retrieve verbal or visual information (Lezak, 1995). Verbal fluency is measured as the number of verbal responses a person can generate to a given target, such as a specific semantic category (e.g., animals, furniture) or phonetic category (e.g., words that begin with letter F) (Diamond, 2013). Although cognitive tasks have been designed to evaluate these specific functions, it is important to note that most measures are highly inter-correlated and may assess multiple overlapping functions to some extent (for example, the Trails B test is often described as an “executive function” task, although this task likely involves both working memory and cognitive flexibility. Not surprisingly, then, some authors label the function of certain tests differently, and this is particularly evident in meta-analyses of cognition. As we describe findings in this paper, we will use the terms proposed by the authors but will also identify key tests used to define a cognitive construct.

With this background in mind, we turn to a discussion of cognitive deficits, then of emotion-related traits. Our hope is that those concise summaries provide evidence for the importance of both domains, but also specificity regarding the facets of emotion and cognition that are most impaired in BD. This specificity then guides our consideration of models that integrate cognition and emotion.

1. Cognitive deficits in bipolar disorder

A large body of work identifies prominent cognitive deficits, as measured using neuropsychological tests, in BD (Bourne et al., 2013; Kurtz & Gerraty, 2009; Robinson et al., 2006). Although depressive and manic symptoms relate to lower performance on cognitive tests (Kurtz & Gerraty, 2009) and lower self-reported cognitive functioning (Peters et al., 2014), a more important question for mapping vulnerability is whether these deficits persist after remission. Findings of one meta-analysis suggest that the severity of impairment for those with remitted BD as compared to healthy control groups varies across domain and task, with higher effect sizes for verbal and nonverbal memory, attention, and the Trails B test, relevant to both attention and working memory (Kurtz & Gerraty, 2009). For verbal memory, the most frequently used task was the Rey and California Verbal Learning Test (deficits in the range of $d = 0.81$ across 18 studies). Parallel effect sizes were observed for a nonverbal memory task, the Delayed Recall Task of the Rey Complex Figure Test, ($d = 0.80$ across 3 studies). Executive function, as assessed using the Trails B measure, yielded an effect size of $d = 0.73$ across 18 studies. Comparable effect sizes were also observed for attention on the Trails A task ($d = 0.65$ across 17 studies) and Continuous Performance Task ($d = 0.69$ across 13 studies). Other domains appear to be less affected, with smaller between-group effect sizes, including Verbal Fluency ($d = 0.51$ across 15 studies, measured by FAS phonetic fluency and animal naming) and visuospatial abilities ($d = 0.55$ across five studies, assessed by Block Design). Findings of a different meta-analyses converge in suggesting

that remitted BD was related to a similar profile of larger deficits in verbal memory ($d > 0.8$, assessed by California Verbal Learning Test in 10 studies), executive function ($d > 0.8$, assessed by Trail B in 10 studies) and and smaller deficits in visuoperception ($d < 0.5$, assessed by the copy version of the Rey Complex Figure Test in 4 studies) (Arts, Jabben, Krabbendam, & Van Os, 2008). A more recent meta-analysis confirmed this pattern, with deficits also evident on verbal learning, trail-making, and verbal working memory tasks (Bourne et al., 2013). This literature provides substantial evidence that cognitive deficits persist after remission.

Congruent with findings of the correlational research showing cognitive deficits during remission, longitudinal research documents that symptom fluctuations do not seem to explain most cognitive deficits. In one longitudinal study of individuals who were re-tested as episode status varied, attention and processing speed deficits appeared stable as manic and depressive symptoms fluctuated, although verbal fluency deficits appeared more prominent when depressive symptoms were present (Chaves et al., 2011). Another longitudinal study found that cognitive deficits in BD were stable across five years, with the exception of worsening in verbal memory (Santos et al., 2014); in parallel, a review of meta-analyses concluded that cognitive deficits were generally stable (Szmulewicz, Samamé, Martino, & Strejilevich, 2015). Taken together, evidence from the large number of cross-sectional studies and available longitudinal research indicates on average, those with BD experience cognitive deficits after remission.

Although fewer researchers have compared bipolar subtypes, meta-analytic findings indicate that persons with BD II also show cognitive deficits of the same form but slightly less severe than those observed in BD I (Bora, Yücel, Pantelis, & Berk, 2011). More specifically, those with BD II show less severe deficits than those with BD I, with moderate effect sizes for the contrast of BD I versus II for verbal memory ($d > 0.5$, seven studies), and smaller effect sizes for visual memory ($d = 0.38$, six studies), processing speed ($d = 0.28$, six studies) and general cognition ($d = 0.26$, eight studies). Findings did not indicate that BD I and II differed significantly in attention, planning, working memory, shifting and inhibition. The relatively greater impairment in verbal memory among those with BD I could reflect severity of the illness vulnerability, or the effects of antipsychotic medications, which are more commonly prescribed for BD I than II, as iatrogenic effects of antipsychotic medications have been observed on verbal memory and processing speed (Balanzá-Martínez et al., 2010).

These findings of cognitive deficits do not appear to be strictly an after-effect of years of illness, in that youth with BD show cognitive deficits that appear parallel to those observed among adults. As with adults, treatment and concomitant symptom improvements can improve cognitive performance among those with pediatric BD (Lera-Miguel, Andres-Perpina, Fatjo-Vilas, Fananas, & Lazaro, 2015). Nonetheless, in a meta-analysis of pediatric BD, effect sizes indicated greater deficits among the BD group compared to controls. The effect sizes varied, with larger effect sizes for verbal learning and memory ($Z = 4.65$, nine studies), followed by processing speed ($Z = 3.61$, seven studies), working memory ($Z = 4.19$, seven studies), executive function ($Z = 4.07$, nine studies) and attention ($Z = 3.81$, eight studies). Smaller, but significant, group differences of those with BD group compared to controls were observed in verbal fluency, visual memory, visuospatial skills, and general

cognitive ability, with particularly small effects for motor skills ($Z = 1.76$), assessed in only one study (Nieto & Castellanos, 2011).

These results indicate that adults and children with BD demonstrate cognitive deficits, and that these deficits can be observed even during remission. The most prominent deficits involve executive function, attention, verbal memory and non-verbal memory, indicating that the profile of deficits is quite broad.

Although BD is clearly related to cognitive deficits, these deficits do not appear to be universal. It has been estimated that about 30% of patients with remitted BD will show cognitive performance levels within the normative range (Gualtieri & Morgan, 2008; Martino et al., 2014). This suggests the importance of considering how individual differences in cognition will relate to other domains, such as emotion and functioning.

1.1. Cognitive deficits before onset

Given the robust evidence that many adults and children with BD show cognitive deficits, a key question is whether cognitive deficits can be observed before onset. Much of this work has focused on IQ (Gale et al., 2013; Tiihonen et al., 2005). Here, though, we focus on executive function, as prospective research indicates that executive function more robustly predicts BD onset than does IQ (Meyer et al., 2004). In one study, offspring of mothers with BD, unipolar depression or no history of BD ($n = 74$) completed general intelligence tests between ages 8 and 15, executive function tests between ages 11 and 19, and diagnostic interviews (SCIDs) in early adulthood. Nine offspring were diagnosed with BD as young adults. Those 9 participants showed an average IQ score, but had lower performance on executive function tests ($ds = 0.58–1.34$)than did those who were not diagnosed with BD.These effects persisted when two offspring who had developed bipolar II disorder during adolescence were removed from analyses.

In a study of younger children at high risk for psychosis as indicated by symptom ratings on the Comprehensive Assessment of At-Risk Mental Status, the 16 children who developed BD during the eight-year follow-up period showed substantially lower performance on Trails A and B tests compared to those who did not develop BD (Ratheesh et al., 2013). In sum, two small studies suggest that lower executive function and visuospatial ability can predict the onset of BD.

1.2. Cognitive deficits in relatives of those with bipolar disorder

Two meta-analyses have been conducted with first-degree family members of those with BD as compared to healthy controls (Arts et al., 2008; Bora, Yucel, & Pantelis, 2009). Although the effect sizes observed in unaffected relatives suggest less profound disturbance than the deficits observed in those diagnosed with the disorder, cognitive deficits can be consistently observed in unaffected relatives as compared to controls. These studies provide a different window into the nature of deficits that might be most tied to genetic vulnerability. In one meta-analysis of 17 studies, the family members of those with BD showed the largest deficits in response inhibition (measured by the Stroop Task), ($d = 0.51$ in 6 studies), followed by other executive function measures ($d = 0.36–0.38$), with smaller effects for verbal learning and memory measures ($d \leq 0.33$)(Bora et al., 2009). A meta-analysis with

slightly different selection criteria replicated findings for response inhibition (Stroop d = 0.49, four studies) and other executive function deficits (Trails B, $d = 0.37$, seven studies), and also identified significant bipolar deficits in verbal memory (CVLT immediate and delayed recall $d = 0.42$ and 0.56, respectively assessed in four studies) (Arts et al., 2008). Effects for IQ, FAS, digit span backward, Wisconsin Card Sort Task perseverative errors (assessing shifting, Miyake et al., 2000) and Trails did not reach statistical significance. Despite the clear evidence that deficits are common among unaffected relatives, there is evidence of cognitive heterogeneity in relatives of those with BD. For example, one twin study found that healthy twins with a bipolar sibling showed enhanced verbal fluency and verbal learning relative to controls (Higier et al., 2014).

Findings of multiple studies indicate that these cognitive deficits can be observed early in life, among child or adolescent offspring of those with BD, although these deficits are not as severe as those observed among probands of those with schizophrenia (for review, see Olvet, Burdick, & Cornblatt, 2013). In one study, deficits in executive function, as measured by the Wisconsin Card Sort Task were more severe among adolescent offspring of mothers with BD ($n = 43$) than among the offspring of mothers with major depressive disorder ($n = 72$) (Klimes-Dougan, Ronsaville, Wiggs, & Martinez, 2006), and these effects remained significant when controlling for IQ and current symptoms. In a second study, researchers focused on 45 offspring at risk for schizophrenia or BD, defined as at least one first-degree family member with the disorder and at least four relatives with the disorder (first degree or more distant). The 23 offspring from bipolar families demonstrated impairment in executive function measures of problem solving ($d = 0.98$), initiation ($d = 0.72$), planning ($d = 0.58$), verbal memory ($d = 0.94$ for immediate and 1.03 for delayed recall) and visual memory ($d = 0.73$ for immediate and $d = 0.88$ for delayed recall) as compared to controls (Maziade et al., 2009). Deficits in response inhibition have also been identified in offspring of those with BD I (Frangou, Haldane, Roddy, & Kumari, 2005).

In sum, multiple studies indicate that a broad range of cognitive measures are impaired among unaffected family members of those with BD, even at an early age, including executive function, verbal learning, and visual learning. Of note though, a clearer profile emerges in these at-risk studies of response inhibition as a particularly robust indicator of risk.

1.3. Cognition as a predictor of functional impairment

Given the cognitive deficits observed in those diagnosed with and at risk for BD, a key question is whether these deficits can help explain functional impairment. In a qualitative review of 52 studies, cognitive deficits were consistently associated with lower functioning within BD, in both cross-sectional and longitudinal studies (Baune, Li, & Beblo, 2013). Findings of a quantitative meta-analysis also indicated that cognitive deficits were tied to worse functional impairment, and effects did not appear to be moderated by clinical state, age, or study design (Depp et al., 2012). Multiple cognitive domains were tied to functional impairment, with aggregate *rs* ranging from 0.21 to 0.29, but working memory was the domain most specifically related to functional outcome.

The effects of cognition on functional impairment are also observed in longitudinal studies that control for baseline symptom severity. For example, when cognitive deficits were measured just after a first manic episode among young adults, more severe cognitive deficits significantly predicted lower quality of life six months later when controlling for baseline symptom severity (Mackala, Torres, Kozicky, Michalak, & Yatham, 2014). Although cognitive deficits tend to be stable over time (Samamé, Martino, & Strejilevich, 2014; Santos et al., 2014), a subgroup of patients show declines in cognitive function over time, and faster decline predicted lower functioning (measured via the FAST) at six-year follow-up (Mora, Portella, Forcada, Vieta, & Mur, 2013).

The strength of relationships between cognition and functional outcome likely depend on the measures of functioning employed. Baune et al. (2013) noted that effects were smaller when researchers relied on the Global Assessment of Functioning (GAF), which is not surprising given the low inter-rater reliability of the GAF. In a meta-analysis of 22 studies of cognition and psychosocial function ($N = 1344$), stronger effects were observed for performance-based ($r = 0.32$) and milestone (e.g., achievements such as autonomy, marriage, employment, $r = 0.33$) measures of functioning as compared to clinician- or self-rated measures ($r = 0.23$ and $r = 0.20$ respectively). In this meta-analysis, an overall average correlation of 0.27 was observed between cognitive measures and everyday functioning (95% CI = 0.22–0.32, $p < 0.001$; (Depp et al., 2012).

Cognitive impairment can predict declines in functioning above and beyond the role of symptom status. Parallel with the profile of cognitive findings in BD, evidence implicates a broad range of cognitive variables tied to functional impairment but supports the key role of executive function measures such as working memory indices.

1.4. Summary of cognitive deficits

A large literature indicates that executive function and other facets of cognition are impaired in adults and youth with BD, even during remission. Such deficits can be observed in unaffected family members, with particularly robust evidence for deficits in response inhibition. Although not universally present, cognitive deficits can predict the onset of BD among those at-risk. Cognitive indices, including executive function measures, appear closely tied to functional impairment in cross-sectional and longitudinal studies, particularly when strong measures of function are used. Taken together, these findings suggest that cognition could serve as a vulnerability marker for BD.

2. Emotion in bipolar disorder

Euphoria and anger are defining features of mania, and accordingly, researchers have placed considerable emphasis on understanding emotionality in BD. Findings indicate that even after remission, those with BD display heightened or more frequent negative affectivity on self-report measures (Heerlein, Richter, Gonzalez, & Santander, 1998; Keitner et al., 1996), experience sampling (Knowles et al., 2007), and laboratory-based measures using standardized stimuli (Gruber, Harvey, & Johnson, 2009; Pavlova, Uher, Dennington, Wright, & Donaldson, 2011; Rich et al., 2010). This does not appear to be limited to negative valence, in that those with BD also display heightened or more frequent positive affectivity,

again across self-report measures (Gruber et al., 2009), experience sampling (Knowles et al., 2007), and laboratory-based measures (Gruber et al., 2009; Gruber, Dutra, Eidelman, Johnson, & Harvey, 2011; Gruber, Harvey, & Purcell, 2011). Given the heterogeneous nature of emotion problems that have been observed, one study considered a broad range of emotion-related difficulties, and findings indicated that BD was more strongly tied to elevations of negative emotion as compared to positive affectivity problems (Johnson, Tharp, Peckham, & McMaster, 2016).

Beyond reactivity, researchers have found that those with BD tend to feel less confident in their ability to use adaptive emotion regulation strategies (Gruber, Harvey, & Gross, 2012). Disentangling the emotion regulation literature is not straight forward. Perhaps in response to the frequent emotion states, those with BD often report using many different emotion regulation strategies more frequently than those with no mood disorder do (Gruber, Kogan, Mennin, & Murray, 2013). In addition to the more frequent use of adaptive strategies, they tend to endorse using more maladaptive forms of emotion regulation, such as rumination, more than those without mood disorders do (Gruber et al., 2011; Gruber, Eidelman, & Harvey, 2008; Johnson, McKenzie, & McMurrich, 2008; Rowland et al., 2013; Thomas, Knowles, Tai, & Bentall, 2007). Beyond the large literature on responses to negative emotions, those with severe forms of the disorder endorse frequent use of strategies to dampen positive emotions (Edge et al., 2013; Gruber et al., 2011).

Beyond these typical domains of emotion and emotion regulation, findings have indicated that in the face of a given emotion state, those with BD report more difficulty controlling their speech and behavior. This phenomenon has been referred to as emotion-related impulsivity, and those with BD endorse significantly more concern about this form of impulsivity than other forms of impulsivity, even after remission (Muhtadie, Johnson, Carver, Gotlib, & Ketter, 2014). This tendency toward emotion-triggered impulsivity does not appear to be just the aftermath of the episodes, as it has also been observed among samples at risk by virtue of subsyndromal manic symptoms (Giovanelli, Hoerger, Johnson, & Gruber, 2013; Johnson, Carver, Mulé, & Joormann, 2013). Intriguingly, effects of emotion-related impulsivity do not appear to be just an effect of a higher level of emotionality or arousal, but rather, a more specific problem with constraint in the face of an emotion state (Johnson, Tharp, Peckham, Sanchez, & Carver, 2016).

There is also some mixed evidence that difficulty accurately identifying facial displays of emotion can be observed in adults and children with BD. Because these difficulties are not observed in most studies of remitted samples of adults with BD (Samamé, Martino, & Strejilevich, 2012), we focus on other facets of emotion here.

Given the well-established emotionality among those with BD, a key question is whether emotion problems can be observed before onset. Several studies suggest that problems with emotionality are apparent in the offspring of those with BD, including prolonged duration of emotion responses (Chang, Blasey, Ketter, & Steiner, 2003), and greater lability of negative emotions and to a smaller extent, positive emotions (Birmaher et al., 2013). Indeed, by preschool, offspring of those with BD tend to show increased reactivity to negative emotions in others and difficulty regulating their own negative emotions (Zahn-Waxler, Cummings,

McKnew, & Radke-Yarrow, 1984; Zahn-Waxler, McKnew, Cummings, Davenport, & Radke-Yarrow, 1984). The TEMPS-A cyclothymic scale, comprised of items capturing the tendency to experience overly pronounced shifts in emotion states, has been shown to predict the onset of BD among youth (Kochman et al., 2005).

Given evidence that multiple emotion problems are observed in those diagnosed with and at risk for BD, researchers have considered how individual differences in emotion regulation relate to outcomes within BD. Within BD, more maladaptive approaches to regulating negative emotion relate to more severe depressive symptoms cross-sectionally (Gilbert, Nolen-Hoeksema, & Gruber, 2013; Green et al., 2011; Johnson et al., 2008; Rowland et al., 2013; Thomas et al., 2007) and longitudinally (Johnson et al., 2016; Van Rheenen & Rossell, 2014).

Beyond depression, emotion regulation also appears of import for functional outcomes. In one study, greater tendencies to engage in suppression were related to significantly lower function in BD, even after controlling for indices of negative and positive emotionality (Johnson et al., 2016). When those with BD I over-use dampening of positive mood states, lower functioning and quality of life are observed (Edge et al., 2013). Hence the ability to effectively regulate both positive and negative emotions appears important for outcomes in BD.

Higher emotion-related impulsivity has been found to relate to greater suicidality and aggression among those with remitted BD I. Given this, perhaps it is not surprising that emotion-related impulsivity is tied to substantially lower quality of life and functional outcomes for those with BD I (Johnson & Carver, 2016; Johnson, Carver, & Tharp, 2017; Muhtadie et al., 2014; Victor, Johnson, & Gotlib, 2011).

In sum, findings indicate that BD is related to increased negative emotion reactivity, and perhaps slightly less robustly degree, positive emotional reactivity. Those with BD may use emotion regulation strategies more often than others do, but they feel less confident about the effectiveness of these strategies. BD is also tied to emotion-related impulsivity. Many of these facets of emotionality can be observed before onset among those at risk for the disorder. Emotion variables also are important for outcomes. Those who use more rumination and suppression are prone to depression, and those who use more reappraisal may develop less depression over time. Negative emotionality, maladaptive strategies for regulating negative emotions, and dampening of positive emotion all relate to lower function. Emotion-related impulsivity relates to worse functional outcome, more aggression, and more suicidality in BD.

3. Links between cognition and emotion

Above, we have outlined evidence that both cognitive deficits and emotion disturbances are closely tied to BD, of central importance for functional outcomes in BD, observable before onset, and predictive of the onset of BD. Surprisingly, research in these two important domains has evolved in a largely separate manner within BD. That is, relatively little behavioral research has considered the interface of cognitive deficits and emotionality in

BD. Accordingly, we focus on the rich literature outside of BD on the interface of cognition and emotion regulation.

3.1. Cognition and emotion outside bipolar disorder

Nearly all theories of emotion and emotion regulation involve cognitive processes. According to several theories of emotion, cognition provides a framework for appraising internal and external stimuli (Oatley & Johnson-Laird, 2014). Many experimental approaches provide insight into the ways in which variations in the valence and arousal of specific affective states influences cognition. Certainly, considerable research suggests that high levels of emotion may interfere with executive function, in part because of the resources consumed by prioritizing attention to highly salient, emotion-relevant stimuli (Pessoa, 2009). This prioritization of cognitive attentional resources is frequently observed when stimuli have high “motivational intensity” or induce high arousal (Harmon-Jones & Gable, 2009; Harmon-Jones & Gable, 2010). In contrast, low-arousal positive emotions have been theoretically and empirically linked with greater cognitive flexibility and broader attention, yet more difficulty inhibiting attention to distractions (Ashby, Isen, & Turken, 1999; Dreisbach & Goschke, 2004; Fredrickson, 2003).

In addition to effects of emotion on cognitive processes, experimental and correlational research in healthy populations and in other clinical disorders has provided understanding of the opposite direction–ways in which cognitive processing ability can influence emotion regulation (for review, see Hofmann, Schmeichel, & Baddeley, 2012; Schmeichel & Tang, 2015). Better skill in regulating emotions, as re-flected in the tendency to use reappraisal more and to gain more benefit from reappraisal, has been tied to higher performance on measures of executive function (von Hippel & Gonsalkorale, 2005), and more specifically working memory (Hendricks & Buchanan, 2016; McRae, Jacobs, Ray, John, & Gross, 2012; Schmeichel, Volokhov, & Demaree, 2008), cognitive flexibility (Malooly, Genet, & Siemer, 2013), and cognitive inhibition (see Joormann & Vanderlind, 2014), with particularly strong effects when cognitive inhibition is tested under conditions of stress (Quinn & Joormann, 2015). Other findings show that higher verbal fluency scores are related to greater emotion regulation success, as reflected in physiological measures and changes in facial affect (Gyurak et al., 2009; Gyurak, Goodkind, Kramer, Miller, & Levenson, 2012).

Other research focuses on cognitive abilities related to maladaptive regulatory strategies. The cognitive underpinnings of rumination have been correlated with each of the major domains of executive function: cognitive flexibility, inhibition, and working memory. Perhaps not surprisingly then, cognitive inflexibility, which involves the inability to switch strategies after negative feedback and related tendencies to perseverate, has been correlated with ruminative tendencies (Davis & Nolen-Hoeksema, 2000). To assess other domains of executive function in relation to rumination, multiple studies have used tasks that contrast negatively valenced and neutral stimuli. Multiple studies suggest that rumination is related to poorer executive function—including inhibition, working memory updating, and switching—when valenced stimuli are used as distractors or when the stimuli to be processed are negatively valenced (Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014; Bernblum & Mor, 2010).

Finally, a large body of work has focused on executive function measures and emotion-related impulsivity. Across a set of studies, a recent meta-analysis indicated that emotion-related impulsivity was specifically tied to response inhibition, particularly when samples with more extreme deficits in this form of impulsivity were tested (Johnson et al., 2016).

Studies demonstrating a role of executive function in emotion regulation and emotion-related impulsivity dovetail with findings from functional MRI and electroencephalography (EEG) research. Although a full discussion of the rich literature on the neurophysiological basis of the emotion-cognition interactions is beyond the score of this review, recent approaches in this field increasingly highlight evidence showing substantial overlap in brain regions involved in both emotion and cognitive processes (e.g., Pessoa, 2009). Within this literature, much work has focused on the important role of prefrontal cortex (PFC) for emotion processing and emotion regulation(Banich et al., 2009; Beckwé et al., 2014; Bernblum & Mor, 2010; Davis & Nolen-hoeksema, 2000; Goodkind et al., 2015; Kahl, Winter, & Schweiger, 2012; Ochsner et al., 2004). The PFC is centrally implicated in executive function domains including cognitive control, cognitive flexibility and working memory (Diamond, 2013).

Some of this work has focused on brain regions that are activated when individuals are asked to inhibit attention to emotionally relevant content. These studies broadly show that activation of the right ventrolateral PFC is heavily involved in inhibition, both in the presence of affective and non-affective stimuli (Chiu, Holmes, & Pizzagalli, 2008; Dillon & Pizzagalli, 2007), while the rostral anterior cingulate cortex (rACC) may specifically support inhibition of emotional distractors (Chiu et al., 2008; Etkin, Büchel, & Gross, 2015). To test working memory in the face of emotion distractors, researchers assessed the ability to store goal-directed information after the presentation of an emotional interference (Banich et al., 2009). The results show heightened activation in PFC regions suggesting their engagement in down-regulating activity in emotion-related region, such as the amygdala.

Understanding neural underpinnings of effective emotion regulation rests on considering specific forms of emotion regulation (for review, see Ochsner & Gross, 2005). A large body of work has focused on the neural underpinnings of reappraisal, most commonly using experimental designs in which participants are asked to view emotionally-relevant stimuli and to engage in emotion regulation or as a control condition, to simply view the stimulus. In a meta-analysis of 48 studies, reappraisal was consistently associated the frontoparietal network, including activation of the lateral prefrontal cortex (LPFC), dorsal anterior cingulate cortex (dACC), and the intraparietal sulcus (IPS), and deactivation of the amygdala (Buhle et al., 2014; Messina, Bianco, Sambin, & Viviani, 2015). Emotion distancing, a form of reappraisal in which people try to distance themselves from negative emotions by thinking of the stimulus as far away, long ago, or less relevant to them, also has been found across seven studies to activate the frontoparietal network (Belden, Pagliaccio, Murphy, Luby, & Barch, 2015; Lewis, Todd, & Honsberger, 2007). Of interest, this same frontoparietal network, has been tied to response inhibition(Aron, 2011; Braver, Paxton, Locke, & Barch, 2009; Jamadar, Fielding, & Egan, 2013). Finally, although less is known, emotion-related impulsivity has been linked to diminished activation of some of these same regions, including dACC as well as bilateral inferior frontal gyrus/insula during response

inhibition (Wilbertz et al., 2014). Taken together, the findings highlight the range of neural regions that must be coordinated to produce effective regulation of emotion.

Recent meta-analysis findings suggest that rumination is also tied to regions of the PFC, although in a different manner. That is, rumination appears related to increased functional connectivity of the subgenual prefrontal cortex with the default-mode network, and to elevated activation of the medial PFC in particular (Disner, Beevers, Haigh, & Beck, 2011; Hamilton, Farmer, Fogelman, & Gotlib, 2015).

Much of the research on neural and cognitive mechanisms in emotion, though, is correlational. Certainly, deficits in executive function, such as the ability to inhibit interference from emotion stimuli on a working memory task, can prospectively predict rumination (Zetsche & Joormann, 2011). Moreover, addressing deficits in executive function via computerized training (involving working memory training, or the combination of working memory training with attentional control training) can lead to reductions in rumination in those diagnosed with clinical depression (Siegle et al., 2014; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007; Vanderhasselt et al., 2015), in people with remitted depression (Hoorelbeke, Koster, Demeyer, Loeys, & Vanderhasselt, 2016), and in student samples (Hoorelbeke, Koster, Vanderhasselt, Callewaert, & Demeyer, 2015). In addition, 20 sessions of training on a working memory task involving affective stimuli enhanced the efficiency of the frontoparietal network, and in turn, enhanced ability to down-regulate response to an emotion picture (Schweizer, Grahn, Hampshire, Mobbs, & Dalgleish, 2013). These findings indicate that deficits in executive functions and related neural activity appear to exert an important influence on emotion regulation effectiveness. With these models as backdrop, we turn to the smaller literature on cognition and emotion in BD.

3.2. Links between cognition and emotion in bipolar disorder

Considerable neurobiological evidence supports the premise that prefrontal control of affective states is impaired in BD (e.g., Green, Cahill, & Malhi, 2007; Strakowski, 2012). Reviewers consistently conclude that BD is tied to elevated neural activation in regions involved in responding to salient stimuli, such as the amygdala and ventral striatum compared to other, as well as deficient activation of regions involved in emotion regulation, such as prefrontal cortical areas (Chen, Suckling, Lennox, Ooi, & Bullmore, 2011; Green et al., 2007; Strakowski, Delbello, & Adler, 2012). Reviewers have also identified a consistent pattern of diminished connectivity of regions involved in top-down control with those involved in reactivity for those with BD (Phillips, Ladouceur, & Drevets, 2008). Not only are key emotion-relevant neural regions impaired in BD, but specific work confirms that activity in these regions is relevant to effective emotion regulation for those with BD. For example, frontoparietal activation has been shown to correlate negatively with amygdala activation levels during reappraisal in BD I (Kanske, Schönfelder, Forneck, & Wessa, 2015). The imaging findings fit with a profile of heightened emotionality and diminished emotion regulation in BD.

Although the biology of BD has been proposed to give rise to pervasive difficulties regulating emotion (Ghaznavi & Deckersbach, 2012), surprisingly little behavioral research

in BD has considered how specific cognitive deficits relate to emotion and emotion regulation strategies. One approach has been to consider whether the deficits in executive function observed among those with BD are heightened in the context of emotion stimuli as compared to non-emotion stimuli. A second approach has been to more directly consider the facets of cognition that relate to effective emotion regulation within BD. We consider both approaches here.

We begin by considering research on affective stimuli during executive function tasks. The BD studies on this front have focused on response inhibition tasks such as the go-no go or anti-saccade tasks (Van Rheenen & Rossell, 2013). Those with BD, even during remission, have been found to have difficulty on the affective go-no go task compared to control participants, while showing less impairment on non-affective trials (Bauer, Frazier, Meyer, Youngstrom, & Zunta–Soares, G. B., & Soares, J. C., 2015; Gopin, Burdick, Derosse,Goldberg, & Malhotra, 2011). Similar effects have been observed with a version of the antisaccade task. That is, persons with BD and those with ADHD showed comparable deficits in a standard version of the anti-saccade task; for those with BD, though, emotion faces interspersed between trials of the antisaccade task led to significant decay, such that those with BD performed significantly more poorly than those with ADHD when the emotion faces were presented between trials(Soncin, Brien, Coe, Marin, & Munoz, 2016). Beyond the use of distractors with negative valence, one team used a variant of the antisaccade task in which participants were asked to rapidly look away from positive stimuli on some trials, in contrast to neutral stimuli on other trials. Those with BD showed particular deficits compared to controls when asked to rapidly look away from emotion stimuli (Mueller et al., 2010).

Drawing on the behavioral evidence for difficulties with executive function when emotion stimuli are present, one neuroimaging study examined neural responses to an executive function task involving emotion stimuli among those with BD. More specifically, participants were asked to complete a working memory task (the N-back task) with and without emotional distractors (Mullin et al., 2012). Patients diagnosed with BD showed reduced activity in several areas implicated in executive function and regulation, such as the dorsolateral PFC, dACC and inferior parietal cortex, compared to healthy controls. When emotion distracters were present, however, the bipolar patients showed increased activity in these regions as well as in emotion processing regions such as the amygdala and striatum; the authors argued that this increased activity reflected compensatory responses due to difficulties in regulating in the face of emotion stimuli. Atypical connectivity was also observed between the dACC and the amygdala when emotion distractors were present. The findings support the idea that executive function may be particularly difficult for those with BD when emotion-relevant stimuli are present.

Researchers have extended this approach to consider whether deficits in response inhibition for emotion stimuli can be observed among unaffected family members of those with BD. In the first study, 20 unaffected siblings of those with BD showed a nonsignificant trend toward poor performance on the affective go-no go task compared to controls (Brand et al., 2012). In the second study, seven unaffected offspring did not display significant deficits on the affective go-no go task (Bauer et al., 2015). The null findings are difficult to interpret,

though, given the small sample size and meta-analytic evidence that emotion stimuli are not related to performance on the go/no-go task for those with emotion disorders (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). It will be important to consider response inhibition and emotion in at-risk populations using other tasks and larger samples.

Early findings on whether cognitive indices can predict emotion regulation among those with BD have been more mixed. In one study, a composite measure of executive function based on Trails, Stroop, and Matrics scores did not predict self-rated Difficulties with Emotion Regulation scores among 51 persons with BD (Van Rheenen & Rossell, 2014). Although that study relied on "cold cognition" measures that did not integrate emotion-relevant stimuli, a measure of cognitive flexibility (task-switching) in the context of affective faces was found to predict self-rated ability to use Reappraisal, as measured by the Emotion Regulation Questionnaire among persons with remitted BD (Gul & Khan, 2014). Although preliminary, these findings suggest that it will be important to consider the influence of affective stimuli and contexts in using neuropsychological indices to predict emotion regulation within BD.

4. Summary of cognition and emotion

A large body of basic behavioral and neural research suggests that effective emotion regulation rests on strong executive function, and particularly inhibition. Beyond the evidence that cognitive ability supports effective emotion regulation, the presence of heightened emotion appears to lead to decays in executive function ability. One might expect that persons with clinically relevant deficits in cognition, then, could be prone to heightened emotion states, which in turn, would contribute to decays in cognitive performance. BD, then, is a natural place to consider the interface between cognition and emotion.

Nonetheless, emotionality has often been considered separately from the cognitive deficits in BD. The few integrative findings within BD suggest a more nuanced profile of cognitive deficits than has been observed in the general neuropsychological literature. That is, those with BD show more pronounced difficulties on cognitive tasks that integrate emotion stimuli than tasks without such stimuli. Moreover, performance on tasks with emotion stimuli may be more powerfully predictive of emotion regulation capacity than tasks without such stimuli. This research highlights that in the study of cognition, researchers would do well to include measures that incorporate emotion stimuli, as those may be a more powerful window into BD vulnerability.

5. Future directions

The few available findings support the idea that cognitive deficits are centrally involved in the emotion difficulties observed in BD. This has some overlap with transdiagnostic models of the importance of cognitive deficits (Goodkind et al., 2015), and particularly executive function deficits, with emotion-related symptoms across syndromes (Snyder, Miyake, & Hankin, 2015). If the idea that cognition can explain the emotion problems is supported, this model provides a different way to view the source and treatment of emotion difficulties

within BD. Before considering those implications, we turn to several questions that have not been addressed in the bipolar literature.

To begin, it is often assumed that either cognition or emotion are central starting points for the eventual cascade into bipolar symptoms. Nonetheless, multiple variables may be involved in shaping both cognition and emotion. Of particular relevance, among those with BD, early trauma and adversity are tied to deficits in both cognition (Savitz, Van Der Merwe, Stein, Solms, & Ramesar, 2008) and emotion lability (Aas et al., 2014). Overall, it will be important to consider contextual factors that might contribute to the overlap between cognition and emotion before concluding that cognition is truly a central driver for other outcomes. Given that emotion and cognitive difficulties have been observed in unaffected family members, genetically-informed designs are likely to be of particular importance in understanding these links.

Beyond a better understanding of the context linking emotion and cognition, relatively little is known about whether cognition can explain the full range of emotion problems observed in BD. To date, researchers have been particularly focused on emotion regulation, and yet emotion experience and reactivity may be more closely tethered to disorder. Given how tightly correlated reactivity and regulation are, these facets of emotionality should be conjointly investigated in relation to cognition. Laboratory studies which examine responses to standardized emotion-relevant challenges can be particularly powerful ways to consider the time course of initial reactivity and recovery. Understanding this question could help disentangle whether it is appropriate to consider cognition as a driver of all facets of the emotionality profile in BD, or more strictly of relevance to emotion regulation, or even regulation only of negative stimuli.

Perhaps of most importance, there are key questions about which facets of cognition are most centrally involved in emotionality within BD. Although early research has focused on specific facets of cognitive inhibition or executive function, it remains possible that a much more general cognitive factor guides performance on specific cognitive tasks, as well as a range of emotional and functional outcomes. The facets of executive function are highly correlated, and that there may be general factors that explain much of the variance in specific facets of executive function and that explain emotion-relevant symptoms (Snyder et al., 2015). If true, then hunting for the singular cognitive process that explains these problems could be a flawed approach.

In addition to considering that there may be a general cognitive vulnerability to multiple facets of executive function deficits, the bipolar literature may be prematurely focused only on executive function. That is, multiple studies indicate that a range of cognitive functions beyond executive function are requisite to effective emotion regulation. For example, memory is required to consider alternative perspectives (a key aspect of reappraisal) (Schneider, Gur, Gur, & Shtasel, 1995). Similarly, processing speed is relevant for rapidly processing dynamic emotion stimuli, such as facial expressions as they occur in the real world (De Sonneville et al., 2002). Verbal fluency has been found to mediate the ability to down-regulate responses to expected, but not unexpected, laboratory stimuli (Gyurak et al., 2012; Schmeichel & Tang, 2014). Drawing on such findings, Van Rheenen and Rossell

(2013) argued that language processing, processing speed, memory, and attention might all be relevant for understanding emotion in BD. Disentangling the true nature of the cognitive deficits underpinning emotionality in BD will require work using a battery of cognitive indices, as well as more sophisticated modeling that considers latent variables constructed to consider the role to address measurement error.

Beyond the need for greater specificity of the models linking cognition and emotion, there is a need for understanding how cognitive deficits and emotionality conjointly relate to functional outcomes, given that emotion and cognitive deficits are both clearly linked to functional outcomes in BD when examined separately. At the current time, studies have not examined whether the effects of cognitive deficits fully explain the functional outcomes observed among those with emotion dysregulation. That is, it remains unclear whether emotion qualities will explain functional outcomes above and beyond the role of the cognitive deficits. Understanding the overlapping and unique contributions of cognition and emotion to functionality will be important in choosing the most important targets for intervention. Overall, given the complexity of ways in which cognition and emotion may interface, experimental designs would be particularly helpful to understand these relationships.

Notwithstanding the gaps in testing a fuller model of cognition and emotion in BD, findings of the current review have several implications. Most importantly, the idea that cognition might drive emotionality in this disorder suggests that treatment might focus on relieving cognitive deficits. This is a marked shift from current approaches to treatment, which employ many emotion regulation techniques drawn from cognitive behavioral therapy or Dialectical Behavior Therapy (Linehan, 1993). Although such work remains a vital part of the research agenda in BD, if cognitive deficits precede and drive the emotionality observed in BD, it may be more important to focus on relieving cognitive concerns and hope that this leads to emotion improvements.

Certain pharmacological treatments may help reduce cognitive deficits in BD. For example, cognition has been found to improve with treatment lamotrigine (Pavuluri, Passarotti, Mohammed, Carbray, & Sweeney, 2010) or mifepristone (Young et al., 2004) in BD. It would be helpful to map the time course of changes in cognition and whether this precedes improvements in emotion with receipt of these medications.

To the extent that emotion difficulties improve when cognition problems are treated, this would also provide a clearer test of directionality of links between these two domains. In this way, some of the recent cognitive remediation work is particularly relevant and may provide cleaner information about the direction of effects. To date, a 24-session cognitive remediation program targeting a broad array of 21 cognitive tasks was shown to lead to significant improvements in global executive function and depression, but did not significantly shift working memory in BD. In one open trial of a treatment for BD that included mood monitoring, as well as cognitive coaching on how to improve planning, attention, and memory, participants self-reported improved cognitive and occupational function (Deckersbach et al., 2010). While these studies indicate that cognitive training

could potentially address core cognitive concerns in BD, no research is available on whether this improves emotion regulation.

Again, the literature outside of BD provides helpful insights. That is, cognitive remediation has been shown to be helpful in addressing emotionality when applied with persons who were not diagnosed with BD. In part, these programs may have been successful because they targeted cognitive domains that seem particularly relevant for emotion outcomes. For example, several investigators have developed programs to improve working memory when viewing negative stimuli or completing a stressful task. These working memory training programs have been found to be efficacious in reducing rumination and depressive symptoms (Cohen, Mor, & Henik, 2015; Hoorelbeke et al., 2015; Porter et al., 2017). Taken together, the success of the more specific cognitive remediation programs outside of BD highlight how important it will be to understand which facets of emotionality are driven by cognition, and which facets of cognition make the most appropriate targets in BD. Studies applying cognitive remediation in BD would do well to consider whether improving “cold” cognitive processes alone are sufficient to improve emotion outcomes, as some evidence indicates is the case, or whether cognitive engagement with emotional stimuli amplifies improvement. This line of research may contribute to the development of optimized treatments, based on specific profiles of neurocognitive abilities or emotion dysregulation.

In sum, researchers have gained considerable traction in understanding both cognition and emotion in BD. Given the considerable support for both domains as key facets of vulnerability to the disorder, it is time for a more integrative perspective. We hope this review provides some backdrop that will foster this quest.

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HIGHLIGHTS

- People with bipolar disorder and their relatives often show neurocognitive deficits.
- Emotion concerns are observed in bipolar disorder and those vulnerable to the disorder.
- New models outside of bipolar disorder suggest these domains are linked.
- It is important to integrate neurocognition and emotion research within bipolar disorder.