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Highlights:

- On all levels of the emotion regulation model there are seemingly contradictory findings in BD, with evidence for a profile that is characterized by the tendency to upregulate positive affect, as well as a profile that tends to over-use downregulation strategies for both positive and negative affect.
- These profiles could be characterized by different emotion regulation mechanisms, personality profiles and biological and psychological vulnerability factors.
- Based on these findings we tentatively identify two emotion regulation profiles in BD (reflecting 'approach' and 'avoidant' behaviours respectively)

Understanding Bipolar Disorder Within a Biopsychosocial Emotion Dysregulation Framework

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Abstract:

Bipolar disorder is characterized by extreme mood fluctuations and ongoing affective instability. Mechanisms involved in emotion regulation (ER) seem to be a contributing factor, however the nature and extent of these are not clear yet. The aim of the current review is to contribute to a comprehensive model that covers the full scope of the emotion regulation processes in BD, in order to understand the psychological mechanisms that could contribute to the onset of both manic and depressive states. To this end we review each stage (attentional, behavioural and cognitive processes) of the Process Model of Emotion Regulation in relation to the extant literature on mood or emotion-linked responses in BD. Additionally, potential vulnerability factors (e.g. biological, genetic, personality) for dysfunctional emotion regulation patterns are described. We conclude that on all levels of the emotion regulation model there are seemingly contradictory findings in BD, with evidence for a profile that is characterized by the tendency to upregulate positive affect, as well as a profile that tends to over-use downregulation strategies for both positive and negative affect. These profiles could be characterized by different emotion regulation mechanisms, personality profiles and biological and psychological vulnerability factors. Based on these findings we tentatively identify two emotion regulation profiles in BD (reflecting 'approach' and 'avoidant' behaviours respectively) and

discuss clinical implications and different treatment approaches. To illustrate the latter, we present two clinical cases of both ER profiles and their different treatment approaches.

Introduction

Bipolar disorder (BD) is characterized by extreme fluctuations of mood, including depressed, hypomanic, manic and mixed mood states. The lifetime prevalence of BD is estimated around 1% for bipolar I disorder (BDI) and between 1 and 2% for bipolar II disorder (BDII) (Merikangas, Jin, He et al., 2011). One of the main goals in the treatment of bipolar disorder is to stabilize mood and diminish these extreme fluctuations. Despite evidence-based pharmacological and psychosocial interventions, complete stability is only reached by a very small proportion of people who have BD, with over 90% of BD patients relapsing into new mood episodes during their lifetime (Angst, Gamma, Sellaro et al., 2003). Furthermore, a substantial subgroup of people with BD report frequent switches between low and high mood states, outside of full episodes (Judd, Akiskal, Schettler et al., 2003; Judd, Akiskal, Schettler et al., 2002). Given the co-occurrence of these different patterns of affective instability within BD, a challenge for research is to characterise their distinctiveness, causation, inter-relatedness and implications.

The mechanisms involved in emotion regulation seem pertinent to this endeavour. In the field of major depressive disorder (MDD) it has already been proposed that people who are prone to becoming depressed do not necessarily experience more negative emotions, such as sadness, but instead face challenges in regulating these emotions, resulting in longer periods of negative emotion that contribute to depressed mood (Joormann & Stanton, 2016). It is likely that there are comparable difficulties experienced by people with BD, with the important difference that periods of both extreme negative and extreme positive mood states may be prolonged with dysregulation.

Currently, the idea that difficulty in regulating affective states represents an underlying mechanism of BD is captured in two distinct psychological models that are relatively well studied: The behavioural activation system (BAS) dysregulation theory (e.g. Depue & Iacono, 1989; Urosevic, Abramson, Harmon-Jones et al., 2008) and the Integrative Cognitive Model (ICM) (Mansell, Morrison, Reid et al., 2007). The BAS theory predominantly focuses on the (hypo-) manic episodes in patients with BD. The theory states that individuals with BD are overly sensitive and reactive to goal- and reward-relevant stimuli, which leads to excessive approach-related and reward motivation, which in turn precipitates manic symptoms. There is substantial evidence that, at least for a

subgroup of bipolar patients, such mechanisms play an important role in the development of manic symptoms (Ironside, Johnson, & Carver, 2020). However, the development of recurrent depressive episodes is less well explained by this theory, but could be explained by dysregulation of the behavioural inhibition system (BIS), which could be seen as the opposite of the BAS. The BIS drives inhibitory and avoidance behaviours and is associated with depression, avoidance, and heightened sensitivity to non-reward in the general population and populations with 'bipolar characteristics' (e.g. hypomanic personality, depressive symptoms) (Carver & Johnson, 2009; Dempsey, Gooding, & Jones, 2017; Jones & Day, 2008), but is less well studied in bipolar populations.

The ICM (Mansell et al., 2007) states that the underlying mechanism of both depressive and manic mood fluctuations could be explained by the extreme negative and positive appraisals of internal affective fluctuations. For instance, patients can have extreme positive appraisals about activated states ('when I feel energetic I am the best version of myself') or extreme negative appraisals ('when I feel energetic I lose all control'). Consequently, behaviour is guided by the content of the appraisal. For instance, engaging in stimulating activities to upregulate the energetic state (ascent behaviour) runs the risk of developing a (hypo-) manic state. Alternatively, social withdrawal to downregulate this state (descent behaviour) has the possible consequence of enhancing depressed mood. These different appraisal styles could also apply to negative affective states.

In summary, one model predominantly explains the onset of manic episodes on a behavioural level and the other explains both mood states on a predominantly cognitive level, where interpretations of current states have direct bearing on subsequent regulatory strategies that can be cognitive or behavioural. However, one of the most commonly used emotion regulation models in the literature – the Process Model of Emotion Regulation proposed by James Gross (1998) – posits that attentional processes are important to emotion regulation, alongside behavioural and cognitive processes. Hence, both the formerly discussed psychological models do not fully cover the complete emotion regulation process. The aim of the current review is to contribute towards a more comprehensive model that covers the full scope of the emotion regulation processes, in order to fully characterise the psychological mechanisms that could contribute to the onset of both manic and depressive states.

Aim of the paper

In the first part of this review we summarise each stage of the process model in relation to the extant literature on mood or emotion-linked responses in BD. To do so, we draw upon empirical studies of biases in cognition, behaviour and attention that have compared individuals with BD to those with other mental health conditions, or with no mental health condition. This includes studies

which have tested bipolar participants in the euthymic phase, as well as during depression and (hypo)mania. For the sake of brevity, we do not draw upon literature about broader cognitive functioning in individuals with BD (such as overall deficits in sustained attention, for example), unless this has been studied directly as part of the response to emotional stimuli or with respect to emotion regulation.

In the latter part of the review we consider potential vulnerability factors for the emotion regulation patterns described and briefly summarise the empirical literature on the relationship between emotion regulation difficulties and biological and genetic factors, personality style and life events. Finally, we synthesise the material discussed with insights from clinical practice to suggest a tentative classification of emotion regulation style in people with BD for future investigation.

Process Model of Emotion Regulation

As an organising framework, we refer to the Process Model of emotion regulation (Gross, 1998) (see Table 1). This model distinguishes between emotion regulation strategies according to the phase of emotional processing in which they occur. The model starts with antecedent-focused strategies that occur before full activation of emotion-response tendencies. Within this category, *situation selection* involves avoiding or approaching particular situations (e.g., things or people) to regulate emotion, and *situation modification* describes adapting the situation to change its emotional effects. Selecting particular aspects of the situation to focus upon is referred to as *attentional deployment*, whilst ascribing a particular meaning to these aspects to shape emotion response is termed *cognitive change*. Response-focused strategies, known as *response modulation,* represent attempts to change the course of the emotion response tendency once it has begun. In the following sections these different emotion regulation stages will be discussed in the light of BD. Findings are summarized in Table 1.

Method

Because we had to cover such a broad scope of the emotion regulation domain and its different stages we were not able to adopt a systematic review method/PRISMA guidelines (Moher, Liberati, Tetzlaff et al., 2009). However, we did perform a broad literature search using the followng search terms to identify relevant articles published until October 2019: bipolar (disorder/depression), mania/manic/hypomania, manic depress*, hypomanic AND emotion regulation, emotion, emotion dysregulation, emotional processing, emotion self-regulation, affective regulation, affective

dysregulation, OR dysfunctional beliefs, cognitive appraisal, response styles, cognitive response, cognitive style, coping OR behavioral approach/inhibition/BIS/BAS, goal regulation, reward.

The first search string (referring to bipolar) was limited to title and abstract. We used Web of Science and Pubmed. This led to 4159 papers that were selected for further screening. Based on title 2365 were excluded in the first screening round (for reasons such as not English, review papers, non-bipolar sample, case study etc.). The remaining 1394 papers were excluded (1098) or subdivided into the categories 'Cognitive Style and mood appraisal' (57), 'Structural/functional experimental' (120), 'emotion regulations problems (39) and 'behavioural inhibition/approach and reward' (80). These papers were used to inform the review and also to identify additional relevant literature.

The second part of the current review on potential vulnerability factors associated with emotion regulation processes in bipolar disorder was mainly based on meta-analyses and reviews on these specifics domains.

Stages of the Process Model of Emotion Regulation in Bipolar Disorder

Situation selection & modification

Situation selection, as well as the modification of situations, by definition involves behavioural choices on the part of the individual. These behaviours may reflect organised, goal-directed activity that brings one closer to sources of positive reinforcement which may be novel or inherently stimulating.

According to the BAS theory, individuals with BD are overly sensitive and reactive to goal- and reward-relevant stimuli, leading to excessive approach-related and reward motivation, and, in turn, manic symptoms. In theory, motivational systems such as the BAS are highly likely to be involved in situation selection (approach versus avoidance). Approach and avoidance behaviour can be thought of as operating at several different levels. Considering first the higher level cognitive task of goal-setting, several self-report studies have found people with BD to report stronger achievement and goal-attainment attitudes (Fletcher, Parker, & Manicavasagar, 2013a; Perich, Mitchell, Loo et al., 2014) and to endorse higher ambitions then unaffected controls (Johnson, Carver, & Gotlib, 2012). Two further studies (Carver et al., 2009; Tharp, Johnson, Sinclair et al., 2016) have found that BD patients with more ambitious goals have a higher risk of developing manic episodes prospectively. Additionally, individuals with BD who score high on goal-attainment were more likely to engage in

stimulating and activating behaviour that potentially induces a (hypo-) manic episode (Lee, Lam, Mansell et al., 2010), reported experiencing more BAS-relevant events such as goal-striving, opportunity to obtain goals or rewards, overcoming challenges (Boland, Stange, Labelle et al., 2016) and are more likely to engage in substance abuse behaviour (Abbasi, Sadeghi, Pirani et al., 2016; Alloy, Bender, Wagner et al., 2009).

Further, when individuals with BD made good progress towards a goal, they increased their effort towards this goal, whereas healthy controls (HC's) decreased effort in the same situation (Fulford, Johnson, Llabre et al., 2010). Conversely, when those with BD made lower than expected goal progress, they decreased their effort towards that goal significantly less than control participants did. Two other studies also indicate that individuals with BD put more effort in more rewarding and challenging tasks than do HC's (Harmon-Jones, Abramson, Nusslock et al., 2008; Hayden, Bodkins, Brenner et al., 2008).

At a more basic level of behavioural organisation, locomotion is a core output of BAS (Grillner, Georgopoulos, & Jordan, 1997). Indeed, findings suggest that both motor activity and the tendency to interact with novel objects is elevated in BD during manic and euthymic states (Henry, Van den Bulke, Bellivier et al., 2008; Minassian, Henry, Young et al., 2011; Perry, Minassian, Henry et al., 2010). This is in keeping with a number of studies demonstrating increased rates of both rapid-response and reward-delay impulsive behaviour (preference for a smaller immediate reward relative to a larger delayed reward) within manic episodes (Swann, 2010), whilst self-report measures of impulsivity, but less so behavioural indices, suggest this may remain elevated during euthymia (Newman & Meyer, 2014).

The above described mechanisms especially account for potential approach mechanisms but not avoidance mechanisms (as seen in the BIS theory). A small number of studies have examined avoidance tendencies in people with BD. There is some indication of greater self-reported behavioural inhibition in those with BD compared to HC's and MDD patients (Meyer, Johnson, & Winters, 2001; Weinstock, Chou, Celis-deHoyos et al., 2018; Yechiam, Hayden, Bodkins et al., 2008). These studies examined self-reported passive avoidance of threat. In one further study the majority of participants with BD reported avoiding at least one rewarding activity as a means of preventing mania, suggesting deliberate situation selection as a means of regulating affect (Edge, Miller, Muhtadie et al., 2013).

Besides selecting 'actual or real situations or events' one could also mentally go (back) to specific situations, which is known as 'mental imagery' or 'seeing with the mind's eye' (Kosslyn, Ganis, & Thompson, 2001). Holmes and colleague (Holmes, Geddes, Colom et al., 2008) proposed that mental

imagery might play a role as an amplifier of bipolar mood instability, and could specifically explain both the increased rate of anxiety in BD patients and the occurrence of elevated mood. It seems that bipolar patients report more vivid imagery of future events and higher levels of intrusive imagery compared to patients with stable mood, and comparable to subjects with depression and anxiety (Ivins, Di Simplicio, Close et al., 2014) (Di Simplicio, Renner, Blackwell et al., 2016; Holmes, Deeprose, Fairburn et al., 2011). Although this is a promising theory, evidence is limited and it is unclear which causal relation the heightened imagery in BD has with mood: is it caused by depressed or (hypo-) manic mood, or is it a trigger for mood disturbances?

In conclusion, with respect to situation selection, there is evidence that bipolar patients have an increased tendency to seek challenges/goals and novelty. Additionally they there is evidence for avoidance of threat, but it is unclear to what extent depressive mood contributes to this avoidance behaviour. Lastly, bipolar patients seem to have more vivid images of past and future situations and events. Again the role of mood is not fully clear in this increased imagery in BD.

Attentional bias

Attentional bias occurs when an individual preferentially attends to one class of stimuli in the environment relative to another. Heterogeneity between studies in methodology (e.g. emotional Stroop task, spatial cueing tasks using emotional words or faces and eye-tracking paradigms) and in the mood state of participants makes it difficult to draw strong conclusions about the nature of attentional biases in BD. Here we consider studies that attempted to measure bias in attention.

Two studies report a bias towards threatening stimuli in BD participants, regardless of whether currently euthymic or in an affective episode, relative to control participants (Garcia Blanco, Salmeron, Perea et al., 2014a; Garcia Blanco, Salmeron, & Perea, 2015), whilst another reports greater attentional interference in individuals with euthymic BD relative to control participants when exposed to negative or affectively neutral faces. However, in general, studies have tended not to report biases towards positive or more general negative stimuli in euthymic individuals using behavioural paradigms (Broch-Due, Kjaerstad, Kessing et al., 2018; Garcia Blanco et al., 2015; Garcia Blanco, Salmeron, Perea et al., 2014b; Peckham, Johnson, & Gotlib, 2016). Studying the event-related brain electrical activity to reward stimuli in a gambling task allows more fine-grained investigation of attentional biases during euthymia. In particular, one study found increased early attentional network activation around 100ms (as indicated by the N1 component) in euthymic individuals relative to a matched control group in response to gains and losses. This heightened attention to reward stimuli indicates a reward-related bias at an early, automatic stage which

interestingly predicted later aspects of reward processing such as risk monitoring (Mason, Trujillo-Barreto, Bentall et al., 2016).

A number of studies report differences in patterns of attention to emotional stimuli in bipolar depression relative to mania and euthymia. The direction of these effects tends to vary across studies, with some reporting a bias towards negative information relative to euthymia (Garcia Blanco, Perea, & Livianos, 2013) [emotional stroop task using words]; (Garcia Blanco et al., 2014b) [antisaccade task using faces]), away from negative information (Jongen, Smulders, Ranson et al., 2007) [dot-probe task using words] or away from positive information (Garcia Blanco et al., 2014b) [eyetracking using pictures] and Jabben et al. (2012) [dot probe task using words]. In addition, a further study making this comparison found reduced disengagement from positive and anger stimuli in the bipolar depressed group at short latencies (Leyman, De Raedt, & Koster, 2009). This indicates that disruption in the allocation of attention to emotional stimuli appears to be associated with bipolar depression.

Among those studies testing individuals during mania relative to bipolar depression or euthymia, there is some evidence favouring an attentional bias toward positive material (Garcia-Blanco, Perea, & Salmeron, 2013; Garcia Blanco et al., 2013; Garcia Blanco et al., 2015). At the same time, several studies have found increased attention to negative material and to threat amongst individuals experiencing mania (Garcia Blanco et al., 2013; Garcia Blanco et al., 2015; Lyon, Startup, & Bentall, 1999), although one small study found residual manic symptoms to be associated with gazing away from negative material (Broch-Due et al., 2018).

It is not straightforward to determine whether bias in allocation of attention represents an emotion-regulation strategy or an automatic process that feeds into an aberrant response tendency that requires effortful regulation. Indeed, it is possible that *both* early and late-stage attentional biases may be operating in people with BD. Measurement methods that allow exploration of effects at very short versus longer post-stimulus latencies can help to shed light on this issue. For example, the early-phase differences in brain activity reported by Mason and colleagues (2016) during outcome anticipation suggest that at least some bias in attention associated with BD may be automatic in nature. It is tempting to view findings of bias effects at longer latencies in behavioural tasks as evidence of differences in strategic regulation of attention; however, this requires other explanations to be ruled out (for a discussion of the role of non-attentional processes in delayed stimulus disengagement see Clarke, Macleod, & Guastella, 2013).

Regardless, the relatively small amount of research conducted on attentional bias suggests that compared to HC's, individuals with BD show greater attention to threat stimuli, with attention

towards general emotional stimuli more consistently found in mania than in euthymia. In bipolar depression, the pattern of attention may be characterised by bias both toward and away from emotional stimuli, possibly as a function of response latency and the type of emotion represented. Outside of manic episodes there is reason to suspect that if there is attentional bias towards positive or rewarding stimuli, this is most likely to be detected in the early, automatic stages of processing. Further comparisons with individuals with MDD are required to determine the extent to which patterns of bias may be specific to BD.

Cognitive change

The 'Cognitive change-domain' is the most well studied domain of Gross' Process model in BD. Cognitive change (as an aspect of emotion regulation) refers to modifying the meaning of situations or events. The meaning one ascribes to an experience in the first place will most likely reflect enduring cognitive styles, beliefs and attitudes, and appraisals of internal states. We will first describe these characteristics, before going into the strategies that may be mood dependent. Several studies investigated whether bipolar patients displayed a different cognitive profile (e.g. cognitive strategies that are specifically used by BD patients) compared to HC's and MDD patients. When taking current affective state into account, the majority of the studies found no differences in cognitive strategies between BD and HC's (Fletcher, Parker, & Manicavasagar, 2013b; Fuhr, Hautzinger, & Meyer, 2014; Jabben, Arts, Jongen et al., 2012; Jones, Scott, Haque et al., 2005; Lex, Hautzinger, & Meyer, 2011; Lex, Meyer, Marquart et al., 2008; Mansell, Paszek, Seal et al., 2011; Scott & Pope, 2003; Tzemou & Birchwood, 2007; Wright, Lam, & Newsom-Davis, 2005) or between those with BD and MDD (Fletcher et al., 2013b; Fuhr et al., 2014; Jabben et al., 2012; Jones et al., 2005; Lex et al., 2011; Lex et al., 2008; Mansell et al., 2011; Scott et al., 2003; Tzemou et al., 2007; Wright et al., 2005). Nevertheless, two studies report elevated levels of dysfunctional attitudes in those with BD relative to HC's (Fletcher et al., 2013b; Jones et al., 2005), and a small number have reported greater endorsement of specific types of cognitive style (goal attainment, need for approval in BD relative to those with MDD (Batmaz, Kaymak, Soygur et al., 2013; Fletcher et al., 2013b; Stange, Adams, O'Garro-Moore et al., 2015).

It also appears that people with BD who do have extreme negative attributions and attitudes are at greater risk of developing future depressed and (hypo-) manic episodes (Stange et al., 2015; Stange, Sylvia, Magalhaes et al., 2013a) and have longer time until recovery of mood episodes (Stange, Sylvia, Magalhaes et al., 2013b). In line with the ICM model, BD patients seem to have more extreme positive self-relevant appraisals of the feelings of activation than HC's and MDD patients (Jones,

Mansell, & Waller, 2006; Mansell & Jones, 2006; Mansell et al., 2011; Tosun, Mackali, Cagin Tosun et al., 2015) even after controlling for current mood. Kelly et al. (2011) showed that positive appraisal about activated states predicted BD (in a sample with BD, MDD and HC) only when in combination with high levels of negative appraisals of the same states. The authors suggested these contradicting appraisals might lead to conflicting attempts to up-regulate and down-regulate their mood states, leading to mood disturbances. Furthermore, the number of future relapses appeared to be associated with beliefs about lack of controllability of affective states, negative consequences and greater concern about mood swings (independent of current mood severity) (Lobban, Solis-Trapala, Tyler et al., 2013).

Besides these enduring cognitive strategies, there are also particular meaning-altering strategies that are brought to bear in the moment. Below we will describe whether BD appears to be associated with particular types of cognitive change strategies.

Use of reappraisal has been associated with better mental health outcomes in the general population (Hu, Zhang, Wang et al., 2014); hence, it has been of particular interest to those understanding emotion regulation in BD. Using self-report measures of reappraisal, some studies have found individuals with BD to report reduced levels of reappraisal (Kanske, Schonfelder, Forneck et al., 2015; Wolkenstein, Zwick, Hautzinger et al., 2014) whilst others have not (Green, Lino, Hwang et al., 2011). Self-reported reappraisal deficits in individuals with BD were found to disappear when negative mood was taken into account statistically (Johnson, Tharp, Peckham et al., 2016). Rather than measuring an enduring tendency (i.e., trait-like) use of reappraisal, Gruber, Harvey and Gross (2012) examined spontaneous reappraisal in people with euthymic BD whilst watching emotionally evocative film clips, in comparison to HC's. The group with BD reported engaging in a greater amount of reappraisal (trying harder) but felt that their emotion regulation attempts were less successful. A comparable study found similar rates of reappraisal in people with and without BD, and similar affective, facial and physiological reactivity (Gruber, Hay, & Gross, 2014). Interestingly, differences in neural activation patterns have been observed in people with BD during reappraisal, compared to those without (Kanske et al., 2015; Townsend, Torrisi, Lieberman et al., 2013). These studies showed that BD patients, when actively trying to regulate emotions, have significantly decreased activation of frontal structures and decreased connectivity between the amygdala, and specific prefrontal regions involved in emotion regulation (also during euthymia). Thus, it may be that individuals with BD can engage in reappraisal to the same extent as unaffected individuals, with similar emotional-expressive consequences, but that there are some differences in the neural networks that are recruited during this process, perhaps reflected in the felt sense of effort.

Alternatively, BD patients might overcompensate by trying harder because brain circuitries can't be recruited as effectively (Toma, MacIntosh, Swardfager et al., 2018). It is also possible that reappraisal is reduced in more negative mood states, which has been reported in MDD patients (Joormann & Gotlib, 2010).

Other potentially helpful cognitive change strategies that have been studied in BD include *Putting into Perspective, Refocus on Planning, Positive Refocusing* and *Acceptance*. Studies comparing people with BD to those without, and to other diagnostic groups, have tended not to find between-group differences on these subscales, with the exception of decreased use of *Putting into Perspective* (Green et al., 2011; Rowland, Hamilton, Lino et al., 2013; Wolkenstein et al., 2014), although not in Rowland et al. (2012).

Self-Blame and Catastrophising have been consistently found to be elevated in people with BD relative to those without (Green et al., 2011; Rowland et al., 2013; Rowland et al., 2012; Wolkenstein et al., 2014); however, it does not seem to be the case that elevated scores are associated with BD in particular (Bayes, McClure, Fletcher et al., 2016; Rowland et al., 2013; Rowland et al., 2012; Wolkenstein et al., 2014). There does not seem to be a consistent finding that *Other Blame* is elevated in those with BD relative to those without (Bayes et al., 2016; Green et al., 2011; Rowland et al., 2013; Wolkenstein et al., 2014).

In summary, there are some indications that BD patients use more dysfunctional strategies in the domains of approval, self-control and goal-attainment and that especially extreme positive and negative appraisal of positive and negative affect is characteristic of BD patients. However, it is difficult to separate this from mood dependent effects. Examination of cognitive change processes in real time experimental (for examples see Seeley, Garcia, & Mennin, 2015) settings might allow researchers to parse out these effects. At present though there does not seem to be evidence for a particular profile of cognitive change strategies that sets those with BD apart from those with other mental health conditions.

Emotion response tendencies

In this section we will summarize the neurocognitive and psychological evidence for emotion response tendencies in BD. This stage represents the 'output' of the emotion regulation process: the specific emotion that is felt and/or expressed, including its intensity and duration.

It has been hypothesized that heightened emotionality is an enduring characteristic of BD (Henry et al., 2008), meaning that people with BD experience more intense negative and positive emotions

(apart from their mood episodes) that fluctuate more frequently. One way to define emotional responses is by 'emotional reactivity', which is defined by the magnitude of change from an emotional baseline state in response to emotion-eliciting stimuli (Gross, 1998). The evidence on emotional reactivity in BD is deeply divided. There are indications that people with BD show stronger self-reported emotional reactivity compared to HC's (Aas, Aminoff, Lagerberg et al., 2014; Aas, Pedersen, Henry et al., 2015; Aminoff, Jensen, Lagerberg et al., 2012; Henry et al., 2008), or specifically report more anger and frustration during euthymic states (Dutra, Reeves, Mauss et al., 2014; Johnson & Carver, 2016), but again contradictory findings have been reported (Edge, Lwi, & Johnson, 2015).

Studies using frequent momentary assessments show that euthymic BD patients report more overall negative affect (Gruber, Kogan, Mennin et al., 2013; Havermans, Nicolson, Berkhof et al., 2010) and more fluctuations in both negative and positive emotionality compared to HC's (Knowles, Tai, Jones et al., 2007). However, the majority of laboratory studies that measure self-reported negative emotional intensity after mood induction or emotional stimuli fail to find differences in reactivity in euthymic BD compared to HC's (Aminoff, Jensen, Lagerberg et al., 2011; Edge et al., 2015; Gruber et al., 2014; Lemaire, El-Hage, & Frangou, 2015; M'Bailara, Demotes-Mainard, Swendsen et al., 2009; Mansell & Lam, 2006; Ruggero & Johnson, 2006; Wright et al., 2005) although some studies did find increased reactivity in BD patients (Gruber, Harvey, & Purcell, 2011b; Pavlova, Uher, Dennington et al., 2011). Results are also conflicting with respect to positive stimuli, with some laboratory studies finding increased self-reported reactivity (Gruber, Dutra, Eidelman et al., 2011a; Gruber, Harvey, & Johnson, 2009; Gruber et al., 2011b) whilst others do not (Farmer, Lam, Sahakian et al., 2006; Gruber et al., 2014). There is some evidence that BD may be associated with increased self-reported positive responding to neutral and negative stimuli in the form of film clips and pictures (Gruber et al., 2011b; M'Bailara et al., 2009); but not consistently (Edge et al., 2015; M'Bailara, Atzeni, Colom et al., 2012).

Emotional reactivity has also been measured on a physiological level in BD. For positive emotions, smaller decreases in respiratory sinus arrhythmia and greater heart rate variability (both indices of parasympathetic activity and effective emotion regulation (Thayer & Lane, 2000)) in response to emotional film clips and personal memories have been found in euthymic bipolar individuals compared to HC's (Gruber et al., 2011b; Gruber et al., 2014), although not in an earlier study (Gruber et al., 2009). More indirect evidence for heightened reactivity was found in one study showing excessive startle reflexes in reaction to neutral stimuli (M'Bailara et al., 2009), although this may reflect the way in which neutral stimuli are affectively evaluated rather than reactivity per se.

Arguably, some evidence for differences has been found at a neurological level. Neuroimaging research shows abnormally elevated activity in limbic structures (amygdala, hippocampus and insular cortex) in response to emotional stimuli (Morris, Sparks, Mitchell et al., 2012; Perry, Roberts, Mitchell et al., 2019). In summary, individuals with BD report experiencing elevated emotional responses during daily life, which might be illustrated by abnormal neurological emotional responses in BD patients, which are also subjectively experienced as such.

Response modulation

Response modulation represent attempts to change the course of the emotion response tendency once it has begun. In line with the ICM, appraisals of internal states are related to BD, risk of developing BD, and both clinical and subclinical symptoms of (hypo)mania and depression (Kelly, Dodd, & Mansell, 2017). Further, Dodd et al. (2019) systematic review found that, while there was some evidence for associations between specific facets of cognitive change and outcomes in BD (e.g., reappraisal), the bulk of the evidence was for specific response modulation strategies for upregulating and downregulating both positive and negative affect. Primarily, these were rumination (on both negative and positive affect), risk-taking and impulsive responding to positive affect, and dampening positive affect. Similar findings for relationships between these strategies and mania risk in non-clinical populations have been reviewed elsewhere (McGrogan, Dodd, & Smith, 2019).

Published research has continued to support the findings of Dodd et al.'s (2019) review, in showing that self-reported negative rumination is heightened in BD compared to non-clinical controls (e.g. Oh, Lee, Kim et al., 2019), and there are similarities between BD and MDD in tendencies to engage in negative rumination (Forgeard, 2018; Weinstock et al., 2018). Whereas positive rumination (ruminating on positive affect) may set BD apart from MDD as well as non-clinical groups (Hanssen, Regeer, Schut et al., 2018; Weinstock et al., 2018). However, more recently, and in contrast to much of the literature, brooding (a putatively maladaptive form of ruminating on negative affect) (Treynor, Gonzalez, & Nolen-Hoeksema, 2003) was unrelated to depressive symptoms in a recent study by Peckham et al. (2019).

While positive rumination is a response modulation strategy designed to enhance or maintain positive affect (if that is the desired internal state; Mansell et al., 2007), dampening positive affect is also theoretically and empirically linked to BD. With past experience of problematic high mood states, we would expect people with BD to try to downregulate positive affect when this is appraised as an undesirable state. The literature largely supports this, with more dampening in BD compared

to controls, and dampening being linked to depressive symptoms (Dodd et al., 2019). Further work has largely supported this (Weinstock et al., 2018), although interestingly a recent study (Hanssen et al., 2018) reported that those with MDD were more likely to dampen positive affect, whereas people with BD-II were more likely to dampen compared to those with BD-I. This seems counterintuitive given it is those with BD-I who have experienced the most functionally disruptive high mood states. However, the dampening of positive affect in BD II could also have a protective effect against developing more severe manic episodes. This is in line with a previous study, indicating

that some level of positive rumination is protective against depression, but at increasing levels it could put someone at risk of developing a severely elevated state (Kraiss, Ten Klooster, Chrispijn et al., 2019).

However, the literature does not suggest that people with BD are unable to engage in putatively adaptive emotion regulation strategies. A recent experience sampling study (Leung, So, Kwok et al., 2019) reported that positive affect predicted subsequent adaptive coping (problem-solving and distraction) and no differences were found between euthymic BD patients and HC's in the use of distraction and in neural-activation of involved brain regions (Kanske et al., 2015).

In summary, people with BD appear broadly similar to those with MDD in the use of rumination and dampening, but show greater levels of positive rumination. The available evidence suggests that people with BD do not show a deficit in their ability to engage in adaptive emotion regulation strategies. Furthermore, within BD, research focused on 'response styles' has overwhelmingly reported that poorer outcomes are linked to tendencies to engage in risky and impulsive behaviours, dampening as a *response* to positive or negative affect and in ruminative responses. It seems that people with BD may have tendencies to engage in a range of response-modulation strategies designed to approach or avoid that specific emotional experience, depending on their current context.

Potential vulnerability factors associated with emotion regulation processes in bipolar disorder

Neurobiological factors

There is a wealth of literature on biological vulnerabilities in BD, and it is beyond the scope of this paper to provide an exhaustive overview of the findings. Therefore we will only briefly summarize some important biological findings from neuroimaging, stress biology, and genetic studies.

Findings from neuroimaging studies show structural brain differences between BD patients and HC's in brain regions relevant to emotion regulation. A recent meta-analyses (Lu, Zhong, Ma et al., 2019) shows that BD is associated with significantly decreased volume of brain areas (e.g., PFC including the left DMPFC, left VLPFC, and right precentral) which are strongly implicated in emotion regulation processes. In earlier sections of this paper we reviewed evidence showing that decreased activation of frontal regions during specific emotion regulation processes in BD patients have been observed, together with decreased connectivity between the amygdala and frontal regions involved in downregulation of emotions (Kanske et al., 2015; Townsend et al., 2013)

One amongst many potential neurobiological factors contributing to aberrant emotion regulation in BD is HPA-axis dysfunction, which has been explored in a number of studies, as described in the meta-analyses by Murri et al. (2016). According to this meta-analyses (including studies using a variety of approaches to test HPA-axis activity such as basal cortisol levels, diurnal profile, ACTH test, CRH stimulation test or combined dexamethasone/CRH test (DEX/CRH)). HPA-axis hyperactivity seems to be more prominent among patients in the manic phase, but is also present in the euthymic phase. Genes that are related to HPA-axis activity do not seem to be specific risk factors for BD. However, specific polymorphisms of HPA-related genes have been associated with clinical features of BD, indicating that genetic variations seem to contribute to specific clinical representations of BD. The association between emotion regulation and HPA-axis functioning has not yet been thoroughly studied. However, some studies have shown that positive emotion regulation strategies like problem solving are associated with long term lower cortisol levels (Hoyt, Marin-Chollom, Bower et al., 2014; O'Donnell, Badrick, Kumari et al., 2008) and with increased momentary cortisol responses (Denson, Creswell, Terides et al., 2014; Gilbert, Mineka, Zinbarg et al., 2017; Lam, Dickerson, Zoccola et al., 2009). Whether dysfunctions of HPA-axis activity in BD are related to emotion regulation problems thus remains unclear. One of that factors that may affect both HPA-axis functioning and emotion regulation is childhood or later life trauma, as discussed in the next section.

In terms of genetic predisposition for emotion dysregulation, a review by Van Rheenen and Rossell (2013) describes the role of common variation in the Catechol-O-Methyltransferase (COMT) and Tryptophan Hydroxylase 2 (TPH2) genes in contributing to abnormal prefrontal neurocognitive functioning, which in turn might lead to less control over the regulation of emotions in BD patients. This thesis was also put forward in an earlier review by Green, Cahill and Malhi (2007) with respect to the role of neurocognitive and associated disturbances in brain function in BD that might conceivably contribute to emotion regulation difficulties. In line with these ideas, a recent systematic review by Miskowiak et al. (2019) shows that affective cognitive functioning is impaired in BD, at least with respect to emotional facial recognition, attentional interference by emotional

stimuli, and reward processing. In summary, evidence points towards the existence of biological abnormalities commonly reported in BD patients, which make them vulnerable to emotion regulation disturbances. However, because of the complexity of these individual systems, let alone in interaction with one another, no causal links have been convincingly identified yet.

Early life experiences

A number of studies have demonstrated an association between trauma exposure in childhood and alterations in the different phases of emotion regulation. This includes emotion-modulated attentional biases and disturbances in reward processing (Jaffee, 2017; Pechtel & Pizzagalli, 2011). Rates of childhood abuse are elevated amongst those with BD relative to the general population (Etain, Mathieu, Henry et al., 2010; Watson, Gallagher, Dougall et al., 2014), similar to patients with MDD. It follows that at least some patterns of emotion regulation observed amongst people with BD may not be specific to BD, but may reflect the impact of early life trauma. Furthermore, we would expect to see heterogeneity in emotion regulation patterns across the population of people with BD, partly as a consequence of variation in early life experiences. Indeed, among people with BD, experience of childhood trauma has been found to be associated with worse long term outcomes including earlier age of onset, greater number of bipolar episodes, increased risk of suicide and increased substance use difficulties (Etain, Aas, Andreassen et al., 2013). Several studies indicate that childhood trauma in BD (specifically emotional abuse) seems to be related to later affective lability and more dysfunctional emotion regulation strategies in BD (Aas et al., 2014; Etain, Henry, Bellivier et al., 2008; Marwaha, Gordon-Smith, Broome et al., 2016). Given the strong heritability of BD, the apparent relation between early life trauma and BD may however be contaminated by genetic factors, affecting both parental abusive or neglectful behaviour in parents of BD patients and symptoms of bipolar disorder in (parents and) the offspring.

In addition to experiences of trauma, other chronic stressors in childhood, for example stressors associated with family poverty, has been found to be associated with difficulties in self-regulation and adaptive coping (Evans & Fuller-Rowell, 2013), and alterations in brain areas associated with emotion regulation (Kim, Evans, Angstadt et al., 2013). Young people with a family history of BD have been found to experience higher levels of chronic stress than individuals without a family history (Ostiguy, Ellenbogen, Linnen et al., 2009). Thus, individuals with BD may be vulnerable to particular emotion regulation issues as a consequence of elevated levels of chronic stress in childhood (including as a result of parental mental health difficulties and their impact on parenting behaviours, e.g Clement, Berube, & Chamberland, 2016).

Both childhood trauma and chronic stress have been associated with dysregulations of the HPA axis (Nemeroff, 2016) a process described as allostatic load (Heim & Nemeroff, 2001, 2002): that is, a cumulative adverse effect on the body when an individual is exposed to repeated stressors and/or inadequate responses of physiological systems that are involved in the adaptation to environmental challenges (Danese & McEwen, 2012; McEwen, 1998). Dysregulation of the stress axis also has detrimental effects on function and structural integrity of brain circuitries involved in emotional perception, memory and regulation (Arnsten, 2009, 2015; McEwen & Gianaros, 2011; McEwen & Morrison, 2013; Roozendaal, McEwen, & Chattarji, 2009), thus explaining the above-reported abnormal cortisol and heart rate responses in BD to a psychosocial stressor (Wieck, Grassi-Oliveira, do Prado et al., 2013). Kapczinski et al. (2008) proposed a model in which the interplay between early life stress and the repeated episodes of mood swings after BD onset, and potentially maladaptive symptom management/emotion regulation approaches, lead to a perpetuating allostatic load process.

Future investigation of emotion regulation patterns that may be specific to BD should consider the potential influence of early life trauma and stress. Research should consider that this influence may be additive, but equally may be interactive, whereby experience of childhood trauma in combination with a pre-disposition to BD shapes emotion regulation in particular ways. In later sections of this manuscript we propose that the experience of childhood trauma in combination with BD vulnerability could either predispose for over-regulation of emotions (dampening) or upregulation of specifically positive emotions and/or manic states.

Personality style

There is increasing interest in identifying a personality profile of BD that might (partly) explain the vulnerability to ongoing affective dysregulation. In the general population neuroticism has been linked to negative affect and increased reactivity to negative events, and both extraversion and agreeableness are associated with positive affect, less mood variability and reduced reactivity to negative stimuli (e.g. Bolger & Schilling, 1991; Gomez, Cooper, & Gomez, 2000). We are not aware of BD-specific literature on specific personality traits and their link with emotion regulation in BD. Most literature focuses on distinguishing personality features of BD patients, HC's and other clinical groups. When personality traits of BD patients are compared to healthy populations the most consistent finding is that BD patients demonstrate higher neuroticism compared with HC's (Bagby, Bindseil, Schuller et al., 1997; Barnett, Huang, Perlis et al., 2011; Bauer, Wu, Meyer et al., 2016). However, neuroticism seem to reflect a predisposition to psychopathology in general and not bipolar

specifically. Additionally, research results on extraversion are divided, reporting both higher and lower extraversion scores in BD patients (Barnett et al., 2011; Jylha, Mantere, Melartin et al., 2010). Also other big five personality traits like openness (Bagby et al., 1997; Tackett, Quilty, Sellbom et al., 2008) and low agreeableness (Murray, Goldstone, & Cunningham, 2007; Quilty, Sellbom, Tackett et al., 2009) are associated with BD. However, a consistent BD profile cannot be identified based on these studies.

The divergent results might indicate specific subtypes amongst those with BD; for instance, there may be some with a more depressive-prone or a more manic-prone disease course. Along these lines, it seems that in BD higher neuroticism scores are especially associated with more depressive symptomatology (Lozano & Johnson, 2001; Quilty et al., 2009) and other traits such as high conscientiousness (particularly the achievement striving facet), extraversion and low agreeableness predict increases in manic symptoms across time (Barnett et al., 2011; Kim, Lim, Kim et al., 2012; Lozano et al., 2001; Quilty et al., 2009). However, these associations might be spurious, since they seem to be strongly related to baseline symptoms and may reflect individuals with BD incorporating the experience with their past illness course into their self-reported personality (Parker, Parker, Malhi et al., 2004; Sparding, Palsson, Joas et al., 2017).

When studies look beyond the Big Five personality traits, it seems that bipolar patients report more personality features related to emotional instability, hostility, novelty seeking. A meta-analyses by Zaninotto and colleagues shows that BD patients report higher scores on novelty seeking, self-transecendence and lower on harm avoidance compared to healthy controls and MDD patients, although the exact effects of (subclinical) mood symptoms are not clear. Levels of anxiety are also elevated in BD, even in euthymia, and both compared to HC's and MDD (Bagby et al., 1997; Loftus, Garno, Jaeger et al., 2008; Savitz, van der Merwe, & Ramesar, 2008; Young, Bagby, Cooke et al., 1995).

A study by Qiu et al. (2017) offers a possible explanation for the somewhat contradictory findings in personality profiles. With cluster analyses based on different personality questionnaires they identified three 'subtypes' of bipolar patients: the 'anxious/depressed' type characterized by an anxious/inhibited and dysthymic temperament; the 'relatively stable' type characterised by low anxiety, and intermediate levels of persistence/drive, humour and impulsivity (associated with higher overall functioning, lower rates of rapid cycling and a more stable course); and a 'manic type', which is high on anxiety, but also on insecurity, persistence/drive, spirituality, expressiveness and humour. The existence of possible different 'subtypes' of BD patients might account for the contradicting findings we have found throughout this review.

Discussion

In this narrative review of emotion regulation in BD we have used Gross' Process Model of emotion regulation as a framework within which to consider studies of momentary emergence and regulation of emotion in BD, guided by two contemporary theories of BD (BAS dysregulation model and the ICS). From the evidence reviewed it appears that people with BD differ from unaffected participants in some aspects of emotion regulation, but show substantial overlap with emotion regulation tendencies in MDD. Tendencies that may distinguish people with BD from other groups include greater approach to challenge and novelty, and greater levels of positive rumination. Patterns observed in those with BD appear inconsistent across studies. For example, there is evidence of increase in both approach and avoidant behaviour, attentional biases towards and away from *both* positive and negative stimuli, and a combination of positive and negative cognitive appraisal strategies. It may be important to recognise mood states as a potentially contributing to these inconsistent findings. Still, there seems to be evidence for a profile that is characterized by the tendency to upregulate positive affect, as well as a profile that tends to over-use downregulation strategies for both positive and negative affect. This kind of heterogeneity is also seen when patterns of symptom course are investigated in BD. Based on longitudinal studies, three rather clear subgroups have been identified: 1) a predominantly depressed group; 2) a cycling or episodic group with alternating manic and depressed episodes; 3) a minimally impaired or stable group (Kalbag, Miklowitz, & Richards, 1999; Koenders, de Kleijn, Giltay et al., 2015; Post, Denicoff, Leverich et al., 2003; Solomon, Leon, Endicott et al., 2009; Uher, Mantere, Suominen et al., 2013). One would expect that patients with a different longitudinal course have a different profile on other characteristics as well. Indeed, Qiu et al. (2017) found personality profiles that overlap with groups of patients characterised by differences in course of illness: that is, an anxious/depressed subtype characterized by an anxious and inhibited temperament; a manic subtype with characteristics of persistence, drive and expressiveness; and a stable subtype with more moderate traits.

We thus propose that two distinct emotion regulation profiles could apply to BD. In Figure 1. we illustrate these profiles in detail, combining both the ICM and BAS hypotheses and the evidence in the current review. The first could be seen as predominantly "approach" oriented, and would be expected to be associated with positive appraisals about the (hypo-) manic features of BD. Within this profile, the individual would appear more reward sensitive, goal directed, and likely to seek novelty. They are likely to use stimulating or risk-taking behaviours to elevate positive mood and escape negative mood; this profile would be expected to promote hypo (-manic) phases, as well as contribute to lows due to exhaustion, or extreme negative appraisal and self-blame in the absence of highly energetic states or rewarding situations. In contrast, those showing an "avoidance" profile

are more likely to appraise any mood fluctuations negatively and engage in avoidant behavior in order to prevent intense affective fluctuations; all emotional reactions are (extremely) negatively appraised and responded to with rumination or dampening. Those with this more withdrawn and emotionally suppressed profile would be likely to experience a more depressed mood course. These different profiles might be related to early life experience, neurobiological vulnerabilities and the personality of the patient. For instance, it is known that childhood trauma could lead in some to harm-avoidance and internalizing behavior but in others to more risk-taking and externalizing behavior (Jaffee, 2017). While these speculations are consistent with the different personality profiles reported by Qiu et al. (2017), it is also possible that the experience with BD itself might shape emotion regulation strategies. For instance, very negative experiences with (the consequences of) manic episodes might lead to a more cautious approach to stimulating activities or feelings, while a more positive experience with the manic elements of the disorder might lead to a profile in which the 'manic feelings' are actively approached. This is reflected in the ICM whereby Mansell and colleagues (2007) propose that the individual's affect regulation behaviours have consequences that serve to strengthen the beliefs that led to these behaviours in the first place. To our knowledge this particular bidirectional relationship proposed in the ICM has not yet been tested directly, nor has the possibility of a psychological staging process across the course of BD, whereby life experiences result in changes in emotion regulation style over time which is in line also with the allostatic load model of BD (Kapczinski et al., 2008). To allow for the possibility that different emotion regulation styles could occur in the same individual over time, we refer to these as "profiles" rather than fixed personality types.

The current distinction we make between potentially different emotion regulation profiles clearly requires more empirical investigation, and we do not expect it captures the regulatory styles of all people with BD. Studies testing the validity of this model should go beyond self-report measures and should contain real-life assessment of emotion regulation strategies, for instance in experimental settings (combining self-report with physiological or (f)MRI data) and experienced sampling methods to test ecological validity and interpersonal processes. These mechanisms should also be tested across different phases and mood states of the disease. For more detailed suggestions on how emotion regulation research should be designed to be more ecologically valid we refer to Aldao et al. (2013).

If meaningful subgroups, such as the currently proposed, can be identified, this could also guide psychological interventions. Currently, most empirical studies in BD have tested aspects of the 'approach profile', often linked to BAS dysregulation theory. Although not thoroughly investigated in BD, the 'avoidant profile' has important overlap with emotion regulation dysfunctions that are

identified in MDD (Visted, Vollestad, Nielsen et al., 2018) and in BD this profile would also be associated with a more depressive prone course. As anxiety disorders are highly prevalent in BD patients (Merikangas, Akiskal, Angst et al., 2007), one would also expect that this specific comorbidity is especially seen in patients with the avoidant profile.

Clinical Implications

In our experience, patients with BD who attend psychological therapy (in addition to pharmacotherapy and other guideline treatment approaches) are often seeking support with mood and emotion regulation. Our review would suggest that therapists and patients should not select emotion regulation targets and techniques based merely on diagnosis. It is likely more informative and effective to consider what particular emotion regulation difficulties the individual is experiencing. Our tentative suggestion of two emotion regulation profiles requires empirical testing. However, our clinical experience is that this distinction has been helpful in pointing towards sets of targets and techniques; for example, working on mood-related impulsivity where there is more of an "approach" pattern active, versus working on graded exposure to "dangerous" emotions where the "avoidance" pattern predominates. Crucially, we predict that these interventions will not be necessary for every patient with BD: guideline-based treatment should be executed first. When effects remain limited over time, therapist and patient could consider whether there are ongoing patterns as described above. Therapists should also bear in mind that some emotion regulation difficulties are likely to reflect transdiagnostic vulnerability factors for emotional dysregulation, such as personality style and childhood trauma. In response they may look towards conceptual frameworks and techniques developed for other client groups, such as dialectical behavioural therapy and schema-level work. How the different profiles in our model lead to different disease courses and treatment approaches is described in Figure 2. These descriptions are based on two cases of our own clinical practice.

Limitations

The current review has several limitations. First, because of the broad scope of emotion regulation processes covered it was not possible to execute a rigorous systematic literature review, and thus some relevant literature may not have been included. Second, our proposed emotion regulation profiles require a-priori investigation, systematically comparing individuals across each phase of BD to HC's and individuals with other mental health conditions in order to confirm the existence of

these profiles. The current state of evidence on the role of emotion dysregulation is currently too fragile to draw any firm conclusion on a specific BD profile. Especially the predominant use of self-report measures in most studies and the difficulty to control for the effects of mood state on such measures is a major concern. Further, since studies comparing those with BD to those with other diagnoses are relatively rare, we cannot conclude that the profiles observed are bipolar-specific rather than common to other groups who experience a mental health condition. Our contention however is that they need not be disorder-specific: emotion regulation profile arises as a consequence of the interaction of disorder-specific factors with individual differences.

Conclusion

The current review considered evidence for aberrant emotion regulation strategies in BD, concluding that evidence for a single characteristic pattern of emotion regulation in BD is not evident, but that a small number of specific emotion regulation strategies are consistently implicated in BD. Our review highlights that the field is hampered by absence of systematic comparisons between BD cases and other psychiatric comparison groups, as well as a lack of longitudinal research designs in well-powered cohorts. Nevertheless, the evidence to date supports a model of emotion dysregulation in BD that builds on existing theories (BAS and ICM); we articulate the potential for two distinct profiles of emotion regulation styles in BD in an attempt to propose a more comprehensive psychological model that incorporates all stages of the emotion regulation processes. We have also considered the role of childhood trauma, personality and neurobiological vulnerabilities in contributing to these processes. Although evidence is still limited, this model could have clinical relevance and help to identify problematic emotion regulation patterns in BD patients that suffer from ongoing mood symptoms despite treatment according to clinical guidelines.

Conflict of interest

All authors declare no conflict of interest

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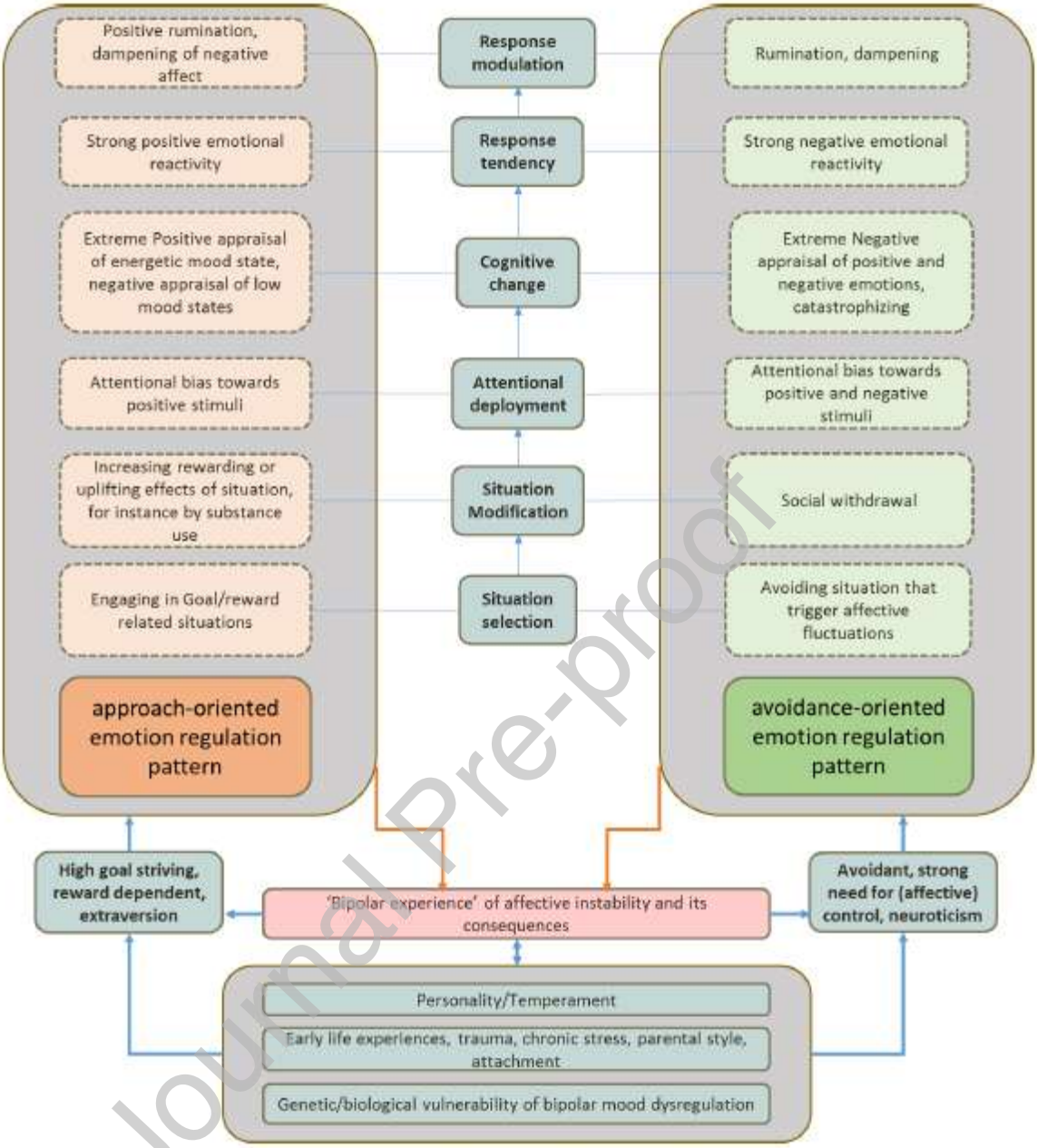

Figure 1. Emotion regulation processes in bipolar disorder for the 'avoidant' and 'approach' profiles

Figure 2. *Descriptions of two clinical cases based on emotion regulation profile*

CASE 1 – TREATMENT EXAMPLE OF THE 'APPROACH PROFILE'

A 48 year-old man, suffers since his thirties from recurring manic and depressive episodes. Despite guideline-treatment he keeps relapsing into a manic episodes, with a consecutive severe depressive episode. He is referred to the psychologist to assess whether psychological mechanisms contribute to the ongoing mood instability.

The patient is high functioning and also demands from himself to excel in what he does (high goal-setting). He appears to identify himself with what appears to be his 'hypo-manic side'. He positively appraises his highly energetic, fast, humorous, festive, creative and intelligent states. He detests his depressed phases. He is motivated to not becoming manic again because of the damage this has caused to his professional and private life. However, he does not want to give up the (subclinical) hypomanic states. He actively seeks for uplifting situations such as festivals, concerts, traveling and occasionally stimulates his energetic mood with substances. Through CBT techniques the patient is able to identify this pattern, in which he 'chases' an energetic mood state, which in turn puts him at great risk for developing a manic symptoms, which results in a subsequent long and severe depression. Through cognitive and behavioral interventions he is able to shape situations that are associated with an energetic mood in such a way that they are less risky for developing a manic state. For instance, going to festivals he does in 'senior' style now, which means that he sleeps in a rental house outside the festival terrain, does not use drugs and plans a couple of days rest after the festival. Cognitive interventions are focused on cognitions about being dull, slow and not 'himself' when in a normal state. Additionally, his extreme positive beliefs about his energetic mood states are challenged. The result is that the patient is able to be more satisfied with his stable state and self. He only experienced a brief hypomanic episode, in the following year and is now able and motivated to intervene in time when his mood is dysregulated instead of upregulating this mood state.

CASE 2 – TREATMENT EXAMPLE OF THE 'AVOIDANT PROFILE'

A 45 year-old women with a bipolar I disorder experienced several depressive episodes from her twenties on and had her first (and last) manic-psychotic episode at 30 years of age after an impactful life event. Despite guideline treatment she remained suffering from chronic depressive symptoms. She followed additional CBT for self-esteem and generalized anxiety disorder, which had very little effect on her depressed symptoms. After thorough psychological assessment it appears that she had a traumatic background, with a history of emotional and physical abuse. After her manic episode she became very afraid of getting ill again. She started living a very 'cautious' live. The combination of her traumatic background, the experience of extreme affective dysregulation during her mania and a personality characterized by neuroticism and harm avoidance, let her to dampen basically all her emotions by highly punitive cognitions and avoiding situations that could trigger any affective state. Through individual schema-therapy these dysfunctional patterns were identified and addressed through cognitive and exposure-based (trauma-focused) interventions. She was able to develop more neutral cognitions about emotional states, and she experienced that allowing herself to feel and express emotions did not lead to uncontrollable mood dysregulation. Consequently, she did not need to over-control emotions to remain stable. As a result, she was also far more able to experience positive emotions and situations. This led to full remission of her depressive symptoms and a stable mood course.

Table 1. Emotion regulation problems in BD according to different models

Gross model		BAS model of BD	ICM model of BD	Current review
Situation selection and modification	*Avoiding or approaching particular situations, things or people to regulate emotion and adapting the situation to change its emotional effects*	Increased approaching of goal- and reward related situations	Increased approach or avoidant behavior	• Increased approach-behaviour of goal- and reward related situations, substance (ab-) use*; increased interaction with novel environments** • Some evidence for increased inhibition and avoidance of reward related situations. **
Attentional deployment	*Selecting particular aspects of the situation to focus upon*	Increased focus on rewarding or goal directed stimuli	Increased focus on internal changes	• Overall no bias to both negative and positive stimuli in euthymia (only during mood states)*. • Some evidence for increased attention to emotional stimuli. *
Cognitive change	*Ascribing a particular meaning to the aspects of a situation to shape emotion response*	Increased positive appraisal of rewarding situations	Increased extreme positive or negative appraisal of internal state	Cognitive traits • No convincing evidence for overall increased dysfunctional attitudes. ** • More extreme positive and negative appraisals of internal feelings of activation. ** Cognitive strategies • Less effective use of positive strategies (reappraisal). * • Less use of positive strategies, but may be a function of depressive symptom level * • Elevated use of negative cognitive strategies (catastrophizing and self-blame), but may be a function of depressive symptom level. *

Response tendency	*The specific emotion that is felt and/or expressed, its intensity and duration*	Increased positive, energetic, active affect	Increased positive or negative affect	• Some evidence for increased emotional responses based on self-report* • No convincing evidence for increased emotional reactivity and peripheral psychophysiological responses* • Based on neuro-imaging studies abnormally elevated activity in limbic structures in response to emotional stimuli*
Response modulation	*Attempts to change the emotion response tendency once it has begun*	Upregulate positive mood by goal directed behaviour	Ascent or descent behaviours	• Heightened negative* and positive **. rumination • Increased dampening of positive and negative affect* • Risk-taking and emotion-based impulsivity (urgency) in response to positive affect*

Note. * From studies comparing individuals with Bipolar Disorder to unaffected control participants; **From studies comparing individuals with Bipolar Disorder to unaffected control participants, and to other patient group(s).