Emotion Regulation Difficulties and Social Cognitive Impairments in Bipolar Disorder

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Abstract

Bipolar disorder is characterized not only by mood dysregulation but also by marked difficulties in regulating emotions and interpreting social information. This paper examines research on the intersection of emotion regulation and social cognition in bipolar disorders, especially focusing on how regulatory processes such as cognitive reappraisal and suppression shape social cognitive functions, including theory of mind, emotion recognition, and interpersonal interpretation. Evidence consistently indicates individuals with bipolar disorder experience impairment in both domains, and that deflection in emotional regulation may affect their biases in social cognition, contributing to interpersonal difficulties and functional impairment. However, the literature remains fragmented. Most studies addressing these constructs separately rather than integratively. Overall, the synthesis suggests the emotion regulation difficulties are an important mechanism underlying social-cognitive impairments in bipolar disorder. Future research should employ integrative designs to clarify causal relationships and explore how targeting emotion regulation strategies may improve social functioning and treatment outcomes in bipolar disorder.

Keywords

Bipolar Disorder; Emotion Regulation; Social Cognitive; Emotion Recognition; Theory of Mind; Cognitive Impairment.

1. Introduction

1.1. A Severe Psychiatric Illness

Bipolar disorder is a severe psychiatric illness defined by recurrent episodes of depression and mania or hypomania, accompanied by disturbances in energy, activity, and cognition. Bipolar I disorder is characterized by at least one manic episode, often accompanied by depression, whereas bipolar II disorder involves recurrent depression with episodes of hypomania. Other subtypes include cyclothymia and bipolar disorder not otherwise specified. These mood fluctuations contribute to substantial impairments in daily functioning and present ongoing challenges for accurate diagnosis (Phillips & Kupfer, 2013).[15] Recent epidemiological data also highlight the growing burden of bipolar disorder. From 1990 to 2019, the prevalence among adolescents and young adults increased from 79.2 per 100,000 population to 84.98 per 100,000 population worldwide (Zhong et al., 2024).[20] This rising trend over the past three decades underscores the importance of identifying not only effective treatments but also the underlying psychological mechanisms that contribute to symptom persistence and functional impairment.

1.2. Difficulties in Emotion Regulation

Emotion regulation is the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions(Gross, 2008)[21] .

Building on this foundation, the process model distinguishes between strategies that intervene at different points in the emotion-generative process, ranging from situation selection and attentional deployment to cognitive reappraisal and expressive suppression. Effective use of these strategies is central to adaptive functioning in everyday life. It is supporting not only psychological well-being but also social relationships, occupational success, and physical health. Conversely, difficulties in emotion regulation have been linked to heightened vulnerability to a range of psychopathologies, including mood and anxiety disorders, which are considered a transdiagnostic risk factor across mental illness(Aldao et al., 2010).[1] Within this framework, bipolar disorder represents a particularly relevant condition in which emotion regulation difficulties have been consistently observed. This makes emotion regulation an important mechanism for understanding both symptom expression and long-term outcomes. Beyond intrapersonal regulation, difficulties in bipolar disorder also extend to social cognition, which is a domain that is closely intertwined with how individuals navigate emotional experiences within interpersonal contexts.

1.3. Deficits of Social Cognition

Social cognition is the formation of social perception and behavior shaped by attention, memory, and judgment (Higgins & Bargh, 1987).[10] Social cognition encompasses multiple processes, including theory of mind, emotion recognition, and attributional style. Those disabilities together enable individuals to interpret and respond appropriately to social information (Bora & Pantelis, 2016).[5] However, deficits of social cognition are found in bipolar disorder both biologically and cognitively. For example, individuals with bipolar disorder often struggle with accurately identifying facial expressions and show impairments in inferring the beliefs and intentions of others (Aydemir et al., 2013).[2] Such difficulties are clinically significant because they contribute to misunderstandings in daily interaction, reduced social support, and unnecessary interpersonal conflict which ultimately exacerbates functional impairment and risk of relapse. Importantly, these deficits are not limited to acute episodes but are also evident during periods of euthymia, suggesting that impaired social cognition represents a core feature of the disorder rather than a state-dependent phenomenon.

1.4. The Purpose of this Research

Although a substantial body of research has examined social cognition and emotion regulation in bipolar disorder, most studies have considered these domains separately rather than in an integrated framework. This separation overlooks important interconnections: bipolar disorder is associated with marked difficulties in emotion regulation, which in turn are closely linked to social cognitive processes, while bipolar disorder and social recognition are themselves inseparably related. Recognizing these overlaps is essential, as the three domains which are often treated as independent are in fact intertwined in ways that shape both symptom expression and functional outcomes. The purpose of this research is therefore to synthesize existing findings across these intersecting areas, clarify how emotion regulation and social cognition interact within the context of bipolar disorder, and identify current gaps in the literature in order to highlight directions for future research. To better organize the reviewed literature, the discussion is divided into three major categories: biological, cognitive, and clinical evidence.

2. Biological Evidence

A comprehensive understanding of emotion regulation and social cognition in bipolar disorder requires consideration of the neural mechanisms that support these processes.

2.1. Understanding Neurocognitive Differences in Bipolar

Clark and Sahakian (2008) provide an influential framework for understanding neurocognitive differences in bipolar disorder by distinguishing between state-related and trait-related changes.[6] State-related changes refer to neural alterations that emerge specifically during manic or depressive episodes. Using fMRI, the authors observed exaggerated limbic responses to emotional stimuli during these states, including hyperactivation of the amygdala during emotional processing and altered ventral striatal activation during reward tasks, suggesting an imbalance between heightened limbic reactivity and reduced prefrontal control. In contrast, trait-related changes represent stable deficits observable even during euthymia. Neuroimaging findings reveal persistent abnormalities in the dorsolateral, ventrolateral, orbitofrontal, and medial prefrontal regions, along with reduced activity in the anterior cingulate cortex. These alterations are associated with impairments in executive control, decision-making, affect regulation, cognitive control, and conflict monitoring. Overall, the interaction between stable trait-like deficits in control networks and state-dependent fluctuations in limbic activity reflects a reduction in top-down regulation and exaggerated bottom-up reactivity (Clark & Sahakian, 2008).[6]

2.2. How Brain Differences Manifest in Emotional Processes

While biological studies highlight the neural underpinnings of bipolar disorder, a complementary body of work has examined how these brain differences manifest in everyday emotional processes, particularly in emotion regulation. Berboth and Morawetz (2021) reported in an fMRI study that the amygdala and the left ventrolateral prefrontal cortex show significant functional connectivity during emotion regulation.[4] This finding suggests that these two regions cooperate to support the control of emotional responses, particularly when individuals engage in cognitive reappraisal as a regulation strategy. While Sun et al. (2023) extend this framework by showing the dynamic bottom-up and top-down interplay within the same circuitry during the processing of emotional ambiguity.[18] When individuals are confronted with ambiguous emotional stimuli, the amygdala is rapidly engaged in bottom-up processing, after which the dorsomedial and ventromedial prefrontal cortices become involved to initiate top-down regulation. This pattern highlights the directional and dynamic nature of emotion regulation mechanisms.

In bipolar disorder, disruptions in neural pathways help explain persistent difficulties with emotion regulation. Impaired top-down control from prefrontal regions over the amygdala contributes to mood instability and enduring regulatory deficits (Clark & Sahakian, 2008; Phillips & Kupfer, 2013). [16]Notably, the ventromedial prefrontal cortex typically inhibits amygdala responses (Sun et al., 2023).[18] In bipolar disorder, however, reduced gray matter volume in the ventromedial prefrontal cortex and weaker functional connectivity with the amygdala contribute to heightened mood swings and affective lability. Moreover, abnormal dorsomedial prefrontal cortex activation during social cognition and reappraisal tasks has been associated with difficulties in processing ambiguous social and emotional cues (Aydemir et al., 2013).[2] Take together, these findings suggest that impairments in prefrontal-amygdala circuits underlie core emotion regulation difficulties in bipolar disorder. Such impairments also extend to processes of social cognition, which represent another critical domain of dysfunction in the disorder.

2.3. Neuroimaging Studies

Neuroimaging studies have therefore turned to the neural correlates of social cognition, revealing consistent disruptions in prefrontal and temporo-parietal regions. In their fMRI review, Schurz et al. (2014) identified the medial prefrontal cortex and the bilateral temporoparietal junction as the core neural substrates of theory of mind.[17] Within this

network, the medial prefrontal cortex is primarily involved in mentalizing and in distinguishing between self and others, whereas the bilateral temporoparietal junction supports perspective-taking and facilitates the attribution of beliefs to others. In addition, the superior temporal sulcus is closely associated with the processing of socially relevant visual information, including facial expressions and biological motion. Finally, self-referential processing, scene construction, and episodic memory have been linked to activity in the precuneus and posterior cingulate cortex, further highlighting the distributed neural basis of social cognition. Additionally, the inferior parietal lobule, inferior frontal gyrus, and ventral premotor cortex have also been linked to social cognition. Together, these regions form a core network that plays a central role in action imitation, intention understanding, language processing, and facial expression simulation. Beyond this core network, involvement of limbic system regions further extends these functions by integrating emotional components, thereby transforming simple action imitation into affective empathy (Molenberghs et al., 2012). [12]

Importantly, many of these regions overlap with those implicated in emotion regulation and show functional impairments in bipolar disorder, suggesting that difficulties in social cognition may share common neural mechanisms with emotion dysregulation in the illness. Hypoactivation of the prefrontal cortex results in deficits in top-down control, which not only disrupt emotion regulation but also impair social cognitive processes. In bipolar disorder, hyperactivity of the amygdala during both acute episodes and euthymia further exacerbates emotional dysregulation and contributes to difficulties in decoding emotions within social cognition (Phillips & Swartz, 2014; Aydemir et al., 2013).[2]

3. Cognitive and Behavioral Evidence

Building on the biological evidence, which highlights disruptions in prefrontal–limbic circuits underlying both emotion regulation and social cognition in bipolar disorder, cognitive and behavioral studies provide complementary insights into how these neural abnormalities manifest at the level of observable functioning. Experimental tasks have shown that individuals with bipolar disorder struggle with implementing adaptive strategies such as reappraisal and with accurately interpreting social cues, reflecting the behavioral consequences of impaired top-down control and heightened limbic reactivity.

3.1. Emotion Regulation in Individuals with Bipolar Disorder

Gruber et al. (2012) conducted an experimental study to examine emotion regulation in individuals with bipolar disorder.[11] Participants included euthymic patients, confirmed with scores ≤ 7 on both the Hamilton Depression Rating Scale and the Young Mania Rating Scale, and matched healthy controls. Emotional stimuli consisted of short movie clips validated to elicit positive, negative, or neutral affect. Before each clip, participants were randomly assigned to one of three conditions: cognitive reappraisal, expressive suppression, or passive viewing as a control. After viewing, participants completed self-report measures of effort, success, and emotional intensity. Results showed that, even in euthymia, individuals with bipolar disorder demonstrated an "effort–success gap." They invested greater effort in regulating their emotions yet reported lower perceived success and more intense emotional experiences compared to controls. These findings suggest that patients are not unwilling to regulate emotion, but rather that their internal regulatory mechanisms are impaired, and heightened emotional intensity further compounds these difficulties (Gruber et al., 2012).[11]

Another experiment conducted shortly thereafter also supported the presence of an effort–success gap. Bebko et al. (2014) found that attentional deployment was not necessary for successful regulation via reappraisal or suppression.[3] Although attention may help reduce emotional intensity, it is not a prerequisite for regulation success. This further suggests that

the difficulties observed in bipolar disorder are not merely a matter of attention or motivation but rather reflect impairments in the underlying emotion regulation mechanisms.

3.2. Impairments in Emotion Regulation and Difficulties in Social Cognition

These impairments in emotion regulation are closely paralleled by difficulties in social cognition, another domain in which individuals with bipolar disorder consistently show deficits. In a theory of mind (ToM) task, Montag et al. (2010) found that euthymic patients with bipolar disorder showed significant impairments in cognitive ToM, whereas affective ToM appeared relatively preserved. Importantly, after controlling for age, the number of (hypo) manic episodes was positively correlated with deficits in cognitive ToM ($r = 0.527$, $p = .030$) and negatively correlated with affective ToM performance ($r = -0.546$, $p = .023$).[13] These findings suggest that different dimensions of ToM may be differentially affected as the course of bipolar disorder progresses. However, the study relied on a relatively small sample and behavioral measures alone, which is limiting the generalizability of its conclusions.

3.3. Facial Emotion Recognition (FER)

Findings in the domain of facial emotion recognition (FER) are more complex. David et al. (2014) and Robinson et al. (2015) each conducted experiments on bipolar disorder yet reported strikingly different results.[7][16] In David et al.'s (2014) study, 110 patients with bipolar disorder were compared with 96 healthy controls using a static FER task and executive function tests.[7] The patient group included individuals in depressive, manic, and euthymic states. Without strict control for executive functioning and psychomotor speed, results indicated that all patients with bipolar I disorder performed worse than controls on FER, particularly in recognizing fear, happiness, and surprise. Moreover, FER performance was significantly correlated with executive functioning among euthymic patients, leading the authors to conclude that FER deficits in bipolar disorder are trait-like and present across mood states. In contrast, Robinson et al. (2015) found no significant differences between 91 patients (in remission or depression) and 75 controls.[16] Their design expanded on David et al. by incorporating dynamic morphing facial recognition tasks, the Reading the Mind in the Eyes Test, and the Hexagon Test, while also applying stricter controls for executive functioning and psychomotor speed. Based on these results, Robinson et al. argued that FER deficits are not universally present in bipolar disorder and may instead reflect broader cognitive impairments rather than a specific deficit in social cognition. Taken together, inconsistencies between these studies make it difficult to determine whether FER deficits in bipolar disorder are trait-like or state-dependent. These inconsistencies stem from differences in sample composition, task paradigms, and statistical controls. On the one hand, David et al. (2014) emphasize the pervasiveness and stability of FER difficulties; on the other, Robinson et al. (2015) suggest that such difficulties may be secondary to general cognitive impairments. Future research will therefore require larger samples and more standardized emotion recognition paradigms to clarify these discrepancies.

4. Clinical and Interpersonal Evidence

Beyond laboratory-based cognitive tasks, clinical and interpersonal studies provide converging evidence that emotion regulation and social cognitive difficulties in bipolar disorder manifest in everyday functioning, influencing coping styles, relapse risk, and social relationships.

4.1. Study of Outpatients with Bipolar Disorder

In a study of 92 affectively stable outpatients with bipolar disorder, Nitzburg et al. (2016) found through self-report questionnaires that patients exhibited significant impairments in

overall functioning.[14] These impairments were strongly associated with maladaptive coping strategies, particularly behavioral disengagement and self-blame, which were especially common. In contrast, adaptive coping strategies were not significantly related to functional outcomes. Furthermore, Fletcher et al. (2013) assessed approximately 200 patients with bipolar I or II disorder, 109 patients with unipolar depression, and 100 healthy controls using several questionnaires while using multiple standardized questionnaires (e.g., the Responses to Positive Affect Questionnaire and the Cognitive Emotion Regulation Questionnaire) to assess emotional and coping processes.[9] Their findings indicated that rumination on positive affect was more prevalent among patients with bipolar I and II disorder, who tended to reflect excessively on the causes and meanings of positive emotions. When confronted with negative emotions, these patients were also more likely to engage in risk-taking behaviors.

4.2. Influence of Family Environment

Beyond individual factors, the influence of family environment on patients with bipolar disorder should not be overlooked. Ellis et al. (2014) conducted interviews and administered questionnaires assessing suicidal ideation in 95 adolescents (mean age = 15.54) diagnosed with bipolar I or II disorder, alongside the Five Minute Speech Sample with their parents. Results showed that high parental expressed emotion was significantly associated with greater suicidal ideation among adolescents.[8] Specifically, parental criticism, hostility, and emotional overinvolvement increased the likelihood that adolescents with bipolar disorder reported suicidal thoughts. These findings suggest that the emotional climate of the family constitutes an independent environmental risk factor for symptom severity in bipolar youth. In addition to coping and family environment, broader interpersonal functioning which includes occupational impairment, reduced social networks, and lower quality of life has been consistently documented in bipolar disorder (Sylvia et al., 2017).[19]

5. Conclusion and Future Directions

Overall, evidence from biological, cognitive, and clinical studies demonstrates that emotion regulation and social cognition are consistently impaired in bipolar disorder, and that difficulties in emotion regulation may exacerbate or even underlie deficits in social cognition. From a biological perspective, abnormalities in prefrontal–amygdala circuitry led to insufficient top-down control and exaggerated limbic reactivity. This pattern is mirrored in the cognitive domain, where the effort–success gap in emotion regulation and impairments in theory of mind tasks reflect the consequences of these neural dysfunctions. Importantly, such deficits also manifest in everyday life, contributing to maladaptive coping strategies, risk-taking behaviors, family conflict, and even suicidal ideation, highlighting the pervasive impact of these impairments across multiple levels of functioning.

5.1. Current Insufficiency

Across the reviewed studies, important methodological inconsistencies remain. Task paradigms, sample characteristics, and statistical controls often differ, contributing to heterogeneity in findings. Most studies also rely on relatively small samples and predominantly cross-sectional designs, which limit the generalizability of results and may account for contradictory outcomes, such as the divergent findings in the two FER studies discussed earlier. Moreover, in this intersecting field of emotion regulation, social cognition, and bipolar disorder, integrative designs remain scarce, with most research examining these domains in isolation rather than within a unified framework.

5.2. Future Direction

Building on the limitations outlined above, several directions for future research in this field are worth emphasizing. First, sample sizes need to be expanded. Many existing studies rely on small cohorts, which reduces statistical power. Therefore, larger, multi-center studies are required to improve the generalizability of findings. Second, research design should move beyond predominantly cross-sectional approaches, which capture only phenomena rather than causality. More longitudinal studies are needed to track changes across different clinical states of bipolar disorder and to clarify temporal relationships. In addition, multimodal approaches should be more widely adopted. Current work often relies on either fMRI or behavioral paradigms alone, providing only a partial view. By integrating neuroimaging, behavioral tasks, and ecological momentary assessment, future studies can simultaneously capture neural mechanisms, laboratory performance, and real-world functioning, thereby informing more effective interventions for social cognition in bipolar disorder. Finally, given the inconsistency in findings from facial emotion recognition studies, standardized and unified task paradigms will be essential to reduce methodological heterogeneity and increase comparability across studies.

In future clinical applications, several promising approaches may provide valuable benefits for patients with bipolar disorder. Because difficulties in emotion regulation may form the basis of social cognitive deficits, structured training in strategies such as cognitive reappraisal and suppression could help improve mood stability and potentially enhance social functioning. It is also essential to emphasize the importance of family interventions. High levels of expressed emotion within the family environment increase the risk of relapse in bipolar disorder, so future clinical efforts should aim to reduce criticism and hostility. In addition, since maladaptive coping strategies are strongly associated with functional impairment, psychological treatments should incorporate training to strengthen adaptive coping skills.

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