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psychosocial function in bipolar disorder: Is there evidence that social cognitive and emotion regulation abnormalities contribute?

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Phenomenological predictors of

Abstract

Objectives: Neurocognitive ability and mood have often been discussed as contributing mechanisms to the severe psychosocial dysfunction experienced in bipolar disorder (BD). In contrast, there has been little discussion on the contribution of social cognition or emotion regulation. This paper aims to assert a potential role for these constructs in psychosocial functioning in BD, with an overarching goal to highlight the necessary importance of considering them in future research examining psychosocial outcomes in the disorder.

Methods: This paper provides a theoretical synthesis of available and indirect evidence for an influence of (1) social cognition and (2) emotion regulation on psychosocial functioning; it acknowledges important clinical questions that need addressing, and discusses how current research might be translated to improve the treatment of psychosocial dysfunction in BD.

Results: Given their assumed roles in facilitating social interactions and modulating behaviours, it is certainly plausible that abnormalities in social cognition and emotion regulation are detrimental to psychosocial functioning. Currently, there is only minimal direct evidence examining their influence, although existing BD studies are preliminarily supportive of relationships between these constructs.

Conclusions: There are reasonable theoretical grounds, supported by indirect and preliminary evidence, to suggest that social cognition and emotion regulation may be important in the prediction of psychosocial outcome in BD. However, this proposition is limited by the paucity of empirical research directly examining this matter.

Keywords

Emotion regulation, functional outcome, neurocognition, quality of life, social cognition

Introduction

Bipolar disorder (BD) is a severe mood disorder characterised by problems in psychological, social and interpersonal functioning (Coryell et al., 1998; Godard et al., 2012; Judd et al., 2005; Keenan-Miller and Miklowitz, 2011; MacQueen et al., 2001; Rosa et al., 2009; Sánchez-Moreno et al., 2009; Serretti et al., 1999). For patients diagnosed with BD the capacity for effective employment, meaningful and long-term interpersonal relationships and good psychological adjustment is significantly reduced (Australian Bureau of Statistics, 2007). Such impairment can have detrimental effects, with a lifetime risk for suicide in people with BD estimated to be as high as 15% (Black Dog

Institute, 2012). Indeed, BD disrupts life to the same degree as chronic medical illnesses such as multiple sclerosis and

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rheumatoid arthritis, with psychological well-being, family and social relationships, and employment being the life domains most affected (Robb et al., 1997).

Many people with BD have difficulty in carrying out work functions (Judd et al., 2008) and in some cases are unable to work (Dion et al., 1988). They report difficulties in social activities (Morriss et al., 2007) and social skills performance (Goldstein et al., 2006), as well as maladjustment in marital or romantic relationships (Blairy et al., 2004; Tsai et al., 1999). People with BD also consistently demonstrate lower scores on subjective psychosocial functioning (i.e. quality of life) scales (Cramer et al., 2010; Freeman et al., 2009; Gutiérrez-Rojas et al., 2008; Saarni et al., 2010; Srivastava et al., 2010) and have reduced or labile self-esteem (Blairy et al., 2004; Knowles et al., 2007; Serretti et al., 1999).

Traditionally, clinical symptomatology has been implicated as a primary predictor of psychosocial outcome in BD; residual depressive symptoms occurring independently of mood episodes are often found in BD patients (Goossens et al., 2008; Keitner et al., 1996) and this depressive symptomatology appears to exacerbate psychosocial difficulties. However, even when it is attenuated, psychosocial impairment remains (Bauer et al., 2001; Bonnín et al., 2010; Godard et al., 2012; Simon et al., 2008; Tabarés-Seisdedos et al., 2008; Wingo et al., 2009).

At a phenomenological level, BD is characterised by a compromised neurocognitive profile (Arts et al., 2008; Balanzá-Martínez et al., 2005). Meta-analytic studies indicate large effect size deficits for patients with BD, even during remission (Bora et al., 2009a). It is now becoming increasingly clear that patients' ability to develop new skills, respond flexibly to a changing environment and create complex understandings of life are constrained. Consequently, neurocognitive ability has been associated with objective, and to a lesser degree, subjective psychosocial functioning in the disorder in multiple studies, including consistently those examining euthymic samples (Altshuler et al., 2008; Atre-Vaidya et al., 1998; Bonnín et al., 2010; Bowie et al., 2010; Brissos et al., 2008a, 2008b; Burdick et al., 2010; Dickerson et al., 2004, 2010; Dittmann et al., 2007; Fujii et al., 2004; Jabben et al., 2010; Jaeger et al., 2007; Laes and Sponheim, 2006; Lahera et al., 2009; Malhi et al., 2007; Martínez-Arán et al., 2002, 2004, 2007; Martino et al., 2009, 2011a; Mur et al., 2009; Sánchez-Morla et al., 2009; Simonsen et al., 2010; Solé et al., 2012; Tabarés-Seisdedos et al., 2008; Torres et al., 2011; Wingo et al., 2010; Yen et al., 2009). Given evidence that neurocognitive deficits perpetuate psychosocial dysfunction independently of affective symptomatology, neurocognition is considered to have a significant, yet clinically separate role in the prediction of functional outcome in BD (Bowie et al., 2010; Burdick et al., 2010; Jaeger et al., 2007; O'Shea et al., 2010; Tabarés-Seisdedos et al., 2008; Torres et al., 2011).

Nevertheless, as the variance that neurocognition explains in psychosocial outcomes differs across studies (Martínez-Arán et al., 2007; Martino et al., 2009), and as BD is a complex disorder likely to have several biological, psychological and environmental aetiologies, it is likely that other core features in its phenomenological profile also contribute to its psychosocial dysfunction. For example, documented BD patient difficulties in regulating emotion and in social-cognitive processes, including emotion perception and theory of mind, may be involved (Bora et al., 2005; Getz et al., 2003; Meyer et al., 2001; Rossell and Van Rheenen, 2013; Van Rheenen and Rossell, 2013b).

Although conceptually distinct, with neurocognition describing a range of mental functions innately linked to the brain, social cognition describing a branch of processing involved in perceiving, interpreting and responding to the social world, and emotion regulation describing a range of processes involved in the reactivity to, and evaluation and modulation of emotion, there is likely to be inherent overlap in these constructs (Frith and Frith, 2007; Gratz and Roemer, 2004; Gross, 2011; Larner, 2008). Indeed, converging evidence suggests that neurocognition is influential to social cognition and emotion regulation, with the potential for good neurocognition to be a necessary precursor to good social cognitive and emotion regulatory function (Fanning et al., 2012; Van Rheenen and Rossell, 2013a). It is theoretically possible then, that these processes contribute to psychosocial outcome in the disorder as well, particularly given their likely role in facilitating adaptive social interactions that are core to adaptive occupational and social behaviours.

Currently, there is only minimal research in the BD literature explicitly investigating whether social cognition and emotion regulation are related to psychosocial function (Inoue et al., 2004; Simonsen et al., 2010; Torres et al., 2011; Wingo et al., 2009, 2010). As social cognition and emotion regulation have the potential to represent more proximal and complex predictors of psychosocial outcome than neurocognition itself, a better understanding of how significant these constructs may be for predicting psychosocial outcome is important for enhancing the assessment and treatment for the disorder.

This paper draws on both direct and indirect available evidence to assert a potential role for social cognition and emotion regulation in psychosocial functioning in BD. Its overarching goal is to highlight the necessary importance of considering these factors in future research examining psychosocial outcomes in the disorder. To this end, the paper provides a theoretical synthesis of available evidence for an influence of (1) social cognition and (2) emotion regulation on psychosocial functioning, gives rise to important clinical questions that need addressing, and discusses what such research could offer to the treatment of psychosocial dysfunction in BD.

Social cognition as a potential predictor of psychosocial dysfunction in BD

The ability to recognise and theorise about other people's emotions are important factors in social and emotional competence, and are fundamental to interpersonal relationships (Wallace, 1984). Misunderstanding facial and prosodic emotional expressions reduces the accuracy of inferences made about the emotional state of a communicator, and limits the capacity to make social hypotheses, which can lead to a range of maladaptive psychosocial consequences (Batty and Taylor, 2003). Accurate emotion perception enables empathy and pro-social behaviours, whilst misperception of emotion reduces social communication, degrades appropriate emotional responses and increases the tendency to socially withdraw (Hooker and Park, 2002; Izard et al., 2001; Schultz et al., 2001).

Accurate emotional perception has been associated with social and intercultural adjustment in healthy individuals (Leppänen and and Hietanen, 2001; Yoo et al., 2006). In disorders characterised by poor emotion perception and theory of mind ability, there is also growing evidence to demonstrate that emotion perception is related to psychosocial functioning (Couture et al., 2006; Lee et al., 2005; Mueser et al., 1996; Persad and Polivy, 1993). For example, in schizophrenia populations, a greater ability to recognise emotions is related to better independent living and occupational skills (Kee et al., 2003), better social problem solving (Vaskinn et al., 2008), greater overall satisfaction (Sparks et al., 2010) and better social skills (Irani et al., 2012).

Schizophrenia is theoretically and phenomenologically similar to BD, and its associated deficits may also be well represented in BD samples (Bora et al., 2009b; Tabarés-Seisdedos et al., 2008). As in schizophrenia, patients with BD tend to have difficulty in accurately identifying or distinguishing emotional expressions and conceptualising other people's emotional and mental states (Addington and Addington, 1998; Bozikas et al., 2006, 2007; Getz et al., 2003; Hofer et al., 2010; Lembke and Ketter, 2002; Murphy and Cutting, 1990). These difficulties are likely to detrimentally influence the formation of social networks and social relationships (Inoue et al., 2004; Schenkel et al., 2008) that are often significantly impaired in the disorder (Bauwens et al., 1991; Blairy et al., 2004; Calabrese et al., 2003; Elgie and Morselli, 2007; Sánchez-Moreno et al., 2009; see Huxley and Baldessarini, 2007 for a review).

Indeed, there is some evidence to demonstrate that emotion perception deficits are involved in psychosocial functioning in BD. For example, in two separate studies examining samples of euthymic BD patients, facial emotion processing accuracy was found to be related to objective psychosocial functioning (Martino et al., 2011b), lower ratings of depression and greater subjective quality of life (Hoertnagl et al., 2011). There were also reports of a trendlevel relationship between emotion perception accuracy and higher rates of employment and general functioning in the latter investigation.

Our social cognitive ability to make inferences about the mental and emotional state of others, typically referred to as theory of mind, has also been strongly related to psychosocial outcome (Couture et al., 2006). Poor theory of mind ability has been associated with poor interpersonal skills (Pinkham and Penn, 2006), severe social behavioural problems (Brüne, 2005), worse overall community social functioning (Pollice et al., 2002) and poor pre-morbid functioning in schizophrenia (Schenkel et al., 2005). In BD too, theory of mind impairments have been preliminarily linked with poor functional outcome in two euthymic BD samples (Hajnal et al., 2010; Lahera et al., 2009). Such associations presumably occur as a result of the maladaptive emotional responses evoked on misinterpretation or misunderstanding of emotional information. Indeed, poorer ability to manage emotions is associated with poorer positive relationships and greater negative interactions with others (Lopes et al., 2003).

Emotion regulation as a potential predictor of psychosocial function in **BD**

The term 'emotion regulation' is a broad term describing both temperamental reactivity to emotional stimuli and abilities that involve inhibition, initiation and modulation of behaviours, acceptance, awareness, understanding and control of emotions, and the appropriate use of regulation strategies (Gratz and Roemer, 2004; Gross, 2011). Dysregulated emotional responses and trait emotional reactivity are common in BD (Johnson et al., 2009; Meyer et al., 2001); relationships between emotion regulation and psychosocial outcome have been demonstrated preliminarily in both the disorder itself and in other clinical samples that are phenotypically related. For example, Persad and Polivy (1993) found that depressed patients reacted with avoidance and heightened negative affect to emotional cues from others. A recent study by Matthews and Barch (2010) demonstrated a positive association between emotional reactivity to affective stimuli and functional outcome in schizophrenia. Similarly, Goldstein et al. (2006) reported that social skills performance in a group of euthymic adolescent BD patients was reduced in the absence of any observable deficit in social skills knowledge. The authors argued that emotional dysregulation interferes with the utilisation of appropriate social skills.

Indeed, the management of emotions has been found to be an important predictor of the ability to initiate relationships, manage conflict and provide emotional support for others (Yip and Martin, 2006). Those with a greater ability to emotionally manage are also less likely to have negative interactions with others and report greater psychological well-being (Lopes et al., 2003), whereas those with poorer emotion regulation have diminished self-esteem, reduced life satisfaction and depressive symptoms (Gross, 2003). Certainly, patients with BD often experience emotionally intense interpersonal situations characterised by anger or frustration, whereby emotional control is particularly dysregulated (Keenan-Miller and Miklowitz, 2011). Such explosive situations place strain on interpersonal relationships and can lead to the experience of depressive symptoms (Rowe and Morris, 2012) which impact subjective quality of life (Dias et al., 2008). Accordingly, early research indicates that euthymic BD patients' experiences of emotion are more intense than that of controls and correlate with subjective psychosocial function (Hoertnagl et al., 2011). Adolescent BD patients reporting a diminished ability to regulate emotion in anger-provoking situations have also been found to report lower self-esteem, greater feelings of hopelessness and poorer coping strategies than controls (Rucklidge, 2006).

Given the broad nature of the emotion regulation concept, one of several ways that poor emotion regulation may exert its potential influence on psychosocial functioning in BD is by influencing depressive symptoms that are strongly predictive of psychosocial impairment (for a review, see Sánchez-Moreno et al., 2009). Indeed, problematic emotion modulation strategies including rumination for both positive and negative emotion have been associated with depressive symptoms, including reduced or labile selfesteem in people with subclinical hypomania and fullblown BD (Bentall et al., 2011; Gruber et al., 2011). Depression occurring on the basis of disturbed self-esteem in the disorder has been reported to arise from patients' negative perceptions of other people's evaluations of themselves (Johnson et al., 2000). It is certainly plausible that characteristically dysregulated and variable emotional behaviour is at the basis of these negative self-evaluations.

Fluctuations in self-esteem have been specifically associated with the disorder's trait tendency to be reactive to salient emotional stimuli, such that even minor experiences of perceived threat or reward affect levels of self-esteem and confer vulnerability for the development of depressive or manic symptoms (Johnson et al., 2000; Klein, 1992; Shapira et al., 1999; Urošević et al., 2008). For example, heightened emotional reactivity is often accompanied by large changes in self-esteem in people with BD (Pavlova et al., 2011). These changes are reflective of ascent/descent behaviours that pre-empt the development of affective episodes; inflated self-esteem is a defining feature of mania and is prodromal to its onset. Likewise, low levels of self-esteem are prodromal to depression (Lam and Wong, 1997; Mansell and Pedley, 2008). Maladaptive emotional regulation strategies such as rumination and heightened threat reactivity (also called behavioural inhibition) have been found to load on the same factor as low self-esteem, suggesting that they are

highly related and can be considered to form a negative cognitive syndrome that predicts BD symptoms (Van der Gucht et al., 2009). Further evidence of this connection comes from Scott and Pope (2003), who found that hypomanic patients with negative self-esteem were significantly more likely to experience an affective relapse. Unfortunately, an increased history of mood episodes is predictive of worse psychosocial functioning (MacQueen et al., 2000; Sierra et al., 2005).

Trait emotional reactivity to threat or reward has been equated with neurotic temperament, which is increased in the disorder (Gray, 1981, 1987; Mitchell et al., 2007). That is, patients with BD tend to view the world as a threatening place and are highly self-conscious, insecure, low in selfesteem and tend toward worry and negative affect (Jylhä et al., 2010; Mitchell et al., 2004). Patients with BD also demonstrate heightened levels of impulsivity, a lower-order feature of neuroticism. This impulsivity is argued to arise when threat or reward inputs are made and arousal levels increase the speed of subsequently occurring responses (Wallace et al., 1991). There is a substantial literature that documents a relationship between facets of neuroticism and psychosocial function (Pope et al., 2007). For example, in BD, neuroticism has been linked to a lower subjective quality of life, increased symptom severity and frequency, and lower self-confidence (Carpenter et al., 1999; Heerlein et al., 1998; Jones et al., 2009; Lozano and Johnson, 2001; Quilty et al., 2009). Family-related neuroticism has also been associated with poor psychosocial outcome in children of mood-disordered parents (Ellenbogen and Hodgins, 2004) and impulsivity has been related to suicide attempts, increased aggression and poorer subjective quality of life in mood-disordered patients themselves (Ekinci et al., 2011; Perroud et al., 2011).

Neuroticism has also been found to predispose people to the experience of life events relevant to the onset or increase of BD symptoms. Heightened emotional reactivity to reward or threat-based environmental cues is likely to prompt these events (Magnus et al., 1993; Urošević et al., 2010). Indeed, research shows that neurotic people orient attention more readily and are attuned to or have difficulty shifting attention away from negative stimuli (Derryberry and Reed, 1994; Reed and Derryberry, 1995; Wallace and Newman, 1998). In fact, emotional reactivity is posited to be a fundamental variable in triggering BD symptoms by predisposing one to the experience of life events that are subsequently poorly controlled (Johnson, 2005). For example, mania is associated with unrealistically high confidence following an initial success (Johnson et al., 2005).

Discussion

Patients with BD are severely psychosocially impaired, an outcome that has been historically attributed to clinical symptomatology and, more recently, neurocognitive capacity (Bonnín et al., 2010; Brissos et al., 2008b; Zaretsky, 2003). Importantly, the growing body of research indicating a contribution of neurocognition to psychosocial outcome is evident consistently in euthymic samples, and often occurs independently of the influence of subclinical depression. This supports a growing consensus amongst researchers that clinical status and psychosocial status are separable constructs (Bowie et al., 2010; Tabarés-Seisdedos et al., 2008; Torres et al., 2011). Nevertheless, the variance explained by neurocognition varies across studies (Brissos et al., 2008b; Martínez-Arán et al., 2007; Martino et al., 2009) and it is likely that other key trait features of the disorder's phenomenological profile are also partially accountable.

In particular, it is plausible that the capacity for adaptive social cognition and emotion regulation contribute to psychosocial functioning in BD, given the roles of these processes in facilitating social interactions and modulating behaviours that are core to a healthy psychosocial outcome. Indeed, there is reasonable theoretical rationale for the proposition that social cognitive impairments, like neurocognitive impairments, directly influence adaptive psychosocial function in BD. Results from initial studies partly addressing social cognitive contributions in the disorder also suggest that its influence may occur in a manner that is independent from mood (Hajnal et al., 2010; Hoertnagl et al., 2011; Lahera et al., 2009; Martino et al., 2011b). Conversely, it is likely that abnormalities in emotion regulation perpetuate psychosocial dysfunction in BD by catalysing or exacerbating clinical symptoms.

Although there is only minimal direct evidence examining the influence of these functions on outcome, the existing BD studies are preliminarily supportive of relationships between these constructs (Hajnal et al., 2010; Lahera et al., 2009; Martino et al., 2011b). That there is reasonable theoretical justification for their association, formulated on the basis of indirect support for an association between these variables from studies of related disorders and healthy populations (Kee et al., 2003; Mathews and Barch, 2010; Sparks et al., 2010), suggests that the influence of social cognition and emotion regulation on psychosocial function in BD are areas worthy of future research.

Certainly, converging evidence suggesting that neurocognition is influential to social cognitive performance and emotion regulation is indirectly supportive of this proposition (Fanning et al., 2012; Van Rheenen and Rossell, 2013a). However, as there is little clarity with regards to how important the relative contributions of neurocognition, social cognition and emotion regulation are, or the mechanisms by which psychosocial difficulties in BD are maintained, a number of questions remain unanswered.

This is notable given that in overlapping clinical conditions such as schizophrenia, there are growing reports that the effects of neurocognition on psychosocial functioning are mediated by social cognitive processes; social cognition appears to be more proximal to psychosocial outcome and, thus, a potentially better treatment target for the remediation of psychosocial difficulties (Addington et al., 2006; Bora et al., 2006; Brekke et al., 2005; Couture et al., 2006; Green and Nuechterlein, 1999; Green et al., 2000; Harvey et al., 2010; Pinkham and Penn, 2006; Vaskinn et al., 2008; Vauth et al., 2004). Such findings are undoubtedly relevant to the study of psychosocial functioning in BD, and it is certainly possible, albeit speculative, that neurocognition affects psychosocial function indirectly via social cognition in BD as well.

Indeed, the relative influence and importance of these constructs on psychosocial outcome, and the potential mechanisms of mediation they may form for the maintenance of psychosocial difficulties in BD represent important clinical questions. Future studies of psychosocial outcome would therefore do well to investigate social cognition and emotion regulation more comprehensively and concurrently with neurocognition and subclinical symptomatology. Importantly, these studies would need experimental designs that consider the multiple interrelated variables appropriately, and thus employ robust statistical techniques including sophisticated forms of regression, path analysis or structural equation modelling to establish relationships and mechanisms of prediction. Given that it is also unclear as to whether objective psychosocial function parallels subjective psychosocial function (Goldberg and Harrow, 2005; MacQueen et al., 2000), future studies should also endeavour to investigate whether these constructs influence measures of patient-rated or administrator-rated psychosocial outcomes differently.

Certainly, these kinds of investigations would inform the development of psychological treatments that may be effective in improving psychosocial outcome in the disorder. Current psychological treatments for BD include cognitive behavioural therapy (CBT) and interpersonal social rhythm therapy (ISRT); these techniques show small effects on the reduction of symptoms and improvement in psychosocial functioning (Costa et al., 2011; Gregory, 2010; Hlastala et al., 2010; Hollon and Ponniah, 2010). However, these treatments provide intervention after neurocognitive, social cognitive or emotion regulation abnormalities are established. Treatments that target abnormalities such as those associated with neurocognition and those that aim to improve emotional processing, theory of mind and emotional regulation, prior to CBT and ISRT would provide a more solid foundation to build upon, which may in turn result in better psychosocial function for BD patients.

There is growing support for the development of novel treatment approaches to reduce psychosocial dysfunction in BD (Harvey et al., 2010; Martínez-Arán et al., 2011; Mennin and Fresco, 2009; Tufrey and Coulston, 2010). In schizophrenia and schizoaffective disorder, programs that remediate cognitive functions have been found to be efficacious in improving psychosocial function (Anaya et al., 2012; Lewandowski et al., 2011; McGurk et al., 2007;

Medalia and Choi, 2009). Improving cognitive function is also demonstrated to improve mood, possibly due to bolstering self-esteem and facilitating positive learning experiences (McGurk et al., 2005; Wykes et al., 1999). Preliminary support for cognitive remediation in BD has also been recently demonstrated by Deckersbach and colleagues (2010) who noted improvement in occupational functioning following the use of a new remediation treatment designed to improve cognition and depressive symptoms.

Improvements in the perception of facial emotions have also been demonstrated in schizophrenia with the use of a specialised intervention program aimed at remediating facial emotion perception difficulties (Frommann et al., 2003; Wölwer et al., 2005). Such remediation has also led to improvements in social relationships (Sachs et al., 2012). To our knowledge, studies examining the efficacy of such a program, or of recently developed emotion regulation therapies, are yet to appear in a BD sample.

Conclusions

There are reasonable theoretical grounds formulated from indirect and preliminary evidence to suggest that social cognition and emotion regulation may be important in the prediction of psychosocial outcome in BD. However, the paucity of direct and explicit research investigating these factors, and their relative contribution in the context of neurocognition and clinical symptomatology leave many questions unanswered. Clearly, future research acknowledging their potential contribution is necessary.

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Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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