Published in final edited form as:

Clin Psychol (New York). 2009 December 1; 16(4): 470–475. doi:10.1111/j.1468-2850.2009.01185.x.

On the Role of Goal Dysregulation in the Treatment of Bipolar Disorder

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Abstract

Nusslock, Abramson, Harmon-Jones, Alloy, and Coan (this issue) propose that current psychosocial treatments for bipolar disorder be supplemented with interventions focused on altering goal dysregulation pathways. While innovations to existing treatment manuals are always welcome, there are several reasons why this suggestion may require further consideration. We highlight issues pertaining to the status of cognitive-behavioral therapy for bipolar disorder, the distinction between education and psychoeducation, the nature of familial expressed emotion, differences between clinical and analog samples, and the larger question of how to assess mechanisms in psychosocial treatment studies. We also raise the question of whether an optimistic goal orientation can be a protective factor in patients' long-term coping with bipolar disorder.

Keywords

Goal attainment; expressed emotion; family-focused therapy; psychoeducation; cognitive-behavioral therapy; life events

Research has documented that goal dysregulation is a pathway in the genesis of manic episodes among many bipolar disorder (BD) patients. Within samples defined by clinical diagnoses, elevated reward sensitivity (Meyer, Johnson, & Winters, 2001; Johnson, Eisner, & Fulford, in press) and heightened ambitions (Johnson, Eisner, & Carver, 2009) have been observed among those with remitted bipolar disorder. Similarly, life events involving goal attainment (Johnson et al., 2000; Johnson et al., 2008), overly confident views of self (Lam, Wright, & Sham, 2005), and excessive goal engagement (Lozano & Johnson, 2001) predict the course of mania within bipolar I disorder. For those of us who do treatment research in BD, the suggestions of Nusslock, Abramson, Harmon-Jones, Alloy, and Coan (this issue) on ways to improve our treatment protocols to recognize goal dysregulation are welcome. Indeed, current treatment protocols for BD show only moderate effect sizes overall, and these effects are often limited to depressive symptoms, with little comparable impact on mania symptoms. Our goal is to bring patients to recovery and keep them there, rather than asking them to accept the recurrence/remission course of BD, with its lengthy intermorbid periods of residual symptoms and functional disabilities.

The Nusslock et al. article raises important questions about the nature of treatment development. One common focus in treatment trials is to develop manuals that address as many

risk factors as possible. This broad band treatment approach has been a focus of considerable debate, as several authors have suggested that the field consider more conceptually driven treatment development approaches, in which we target specific treatment mechanisms in specific populations (Persons, 2005; Westen, Novotny, & Thompson-Brenner, 2004). Although it might seem intuitive that a manual that covers more ground is preferable, changing treatments that have already been found to be effective to incorporate new strategies must be weighed against factors such as: lengthening the treatment, decreasing therapist adherence, limiting the applicability of the treatment across settings or populations, or potentially, conflicting with other goals of the treatment. As an example, encouraging patients to limit their exposure to goal attainment events may be incompatible with the goal of exposing highly withdrawn patients to novel and rewarding social situations to increase their social and occupational engagement (e.g., Dimidjian et al., 2006). In other words, in continually adding targeted risk factors to existing manuals, we run the risk of developing the proverbial “horse designed by committee.”

There are other assumptions of the suggested approach that require careful examination. One is the conclusion that cognitive-behavioral therapies are highly effective in the treatment of bipolar disorder, and simply need to be “bulked up” with a greater focus on goal attainment cognitions. In fact, the literature on the effectiveness of CBT for bipolar disorder is more inconclusive than as summarized by Nusslock et al. When CBT is effective in bipolar patients, it appears to be with less recurrent patients, and mainly for the outcome variable of depression (Miklowitz & Scott, in press). The five-site U. K. study of 253 highly recurrent bipolar I and II patients, which compared CBT to treatment-as-usual in community centers (Scott et al., 2006), reported a primarily negative result: over 18 months, patients in CBT did not differ from those in TAU on time to recurrence, duration of illness episodes, or mean symptom severity scores. As Nusslock et al. report, a post-hoc analysis revealed that CBT was effective in delaying recurrences among patients with fewer than 12 prior episodes. The flip side of this finding was that TAU was more effective than CBT among patients with 12 or more episodes.

A randomized trial in Canada compared CBT plus individual psychoeducation to individual psychoeducation alone for minimally symptomatic bipolar I and II patients (Zaretsky, Lancee, Miller, Harris, & Parikh, 2008). There were no differences in relapse or rehospitalization rates or mania symptoms between the two study arms. The patients in CBT had 50% fewer days of depressed mood over the study year. Parikh, Zaretsky, and Beaulieu (2007) conducted an effectiveness trial of CBT versus psychoeducation across four Canadian sites with 204 bipolar I and II patients in full or partial remission. There were no differences in outcomes between patients who received pharmacotherapy and 20 weeks of individual CBT versus pharmacotherapy and 6 sessions of group psychoeducation.

The well-designed trial by Lam, Hayward, Watkins, Wright, and Sham (2005) deserves special consideration because of its focus on goal attainment cognition as triggers for mania. Although the study found that CBT was more effective than TAU in preventing recurrences over one year, a 30-month follow-up revealed that the effects of CBT were limited to depressive recurrences and symptoms. Moreover, Lam and colleagues found that CBT was ineffective among patients who showed a sense of “hyperpositive self,” marked by dynamism, persuasiveness, and productiveness. Given this pattern of findings, it seems unlikely that the observed clinical effects of CBT were due to its ability to modify hyperpositive cognitions that occur in the prodrome of mania. The Lam et al. findings, then, provide an example of why a small amount of focus on a treatment target might not achieve changes in the proposed target.

Let us now examine the recommendation that family or group psychoeducational approaches would benefit from informing patients that excessive goal striving is a risk factor for mania. Would providing patients with information about this link be enough? Psychoeducation is often

mistaken for *education*: the provision of information much like one might receive in a class. In our experience, patients and family members want much more than this. Psychoeducation requires at least three components: *exposure* to didactic information about an illness, *personalizing* that information (in this case, acknowledging that one has bipolar illness and that past episodes may have been triggered by surges in confidence secondary to goal attainment), and *strategic planning* (putting the information to use; for example, developing and practicing emotion regulation strategies when faced with goal attainment events). We grappled with these issues when designing family-focused treatment (FFT): caregivers did not change their behavior when told that their high EE behaviors could trigger the patients' recurrences. Instead, they wanted to learn behavioral strategies (for example, negotiation and problem-solving skills such as active listening or making specific requests for changes in another's behavior; self-talk to regulate one's emotions) to respond to the behaviors of the patient that would ordinarily trigger criticisms. They also want to see a concerted effort on the patient's part to be more sensitive to the needs of family members.

Frank et al. (2005) dealt with similar issues when designing the interpersonal and social rhythm therapy for BD. They concluded that it is not enough to teach patients that their episodes can be triggered by sleep dysregulation. Instead, patients must engage in an ongoing process of self-monitoring of daily routines, sleep, moods, and interpersonal events, and develop behavioral plans for regulating daily and nightly rhythms when a disruptive life event occurs. Hence across many different treatments, it is clear that interventions require not just brief mentions of risk factors, but personalization, along with many opportunities to develop and practice new strategies to reduce the impact of the risk factor.

It is worth noting the complexity of the targets that are already embedded within extant treatments. In linking the expressed emotion (EE) research with the literature on BAS-relevant goal dysregulation, Nusslock et al. propose that caregiving relatives who express high levels of EE criticism are often criticizing the patient's recent achievement failures. In our view, this is the nature of criticism in only a small proportion of cases. There are many other issues that drive EE and that must be addressed in treatment. Parents or spouses often use criticisms to hold the patient back from overactivity, overextension, and exhaustion, rather than to push him or her to do more. Relatives are more prone to criticize patients for negative behaviors that they view as being under the patient's control (Wendel, Miklowitz, Richards, & George, 2000; Hooley & Gotlib, 2000), regardless of whether these behaviors have anything to do with goal pursuit. An example would be, "You have trouble listening to me because you let so many other things go on in your head." The family dynamics associated with emotional overinvolvement, the other major component of EE, are even more complex. Some forms of overinvolvement may facilitate the patient's goal striving and developmental quest for independence, whereas other forms interfere with it (Fredman, Miklowitz, & Stanton, 2008). Hence, understanding how to intervene with EE requires attention to many different issues, from symptom identification to patterns of attribution to long-standing bidirectional styles of family communication.

Nusslock et al. recommend that family interventions strive to reduce EE as a means of reducing manic or depressive episodes associated with dysfunctional goal pursuit. In our experience, treatments addressing EE are most effective when therapists work toward building protective factors rather than decreasing risk factors in the family. In a randomized treatment trial of bipolar adults, Simoneau, Miklowitz, Richards, Saleem, and George (1999) found that despite its effects on recurrences and symptom severity, FFT did not bring about greater changes in familial EE relative to brief psychoeducation – levels of EE dropped on their own as patients stabilized. However, patients and relatives in FFT did show greater increases from pre-treatment to post-treatment in positive communication behaviors – such as eye contact, problem solving statements, validation, and affiliative gesturing – relative to patients and relatives in

brief psychoeducation. When families received only brief treatment, use of these skills tended to stay the same or drop in frequency over time. Moreover, when patients showed increases in positive engagement with their relatives, they showed corresponding reductions in their mood symptoms (notably, depression) over one year. Thus, one mechanism by which FFT brings about symptom improvements may be through increasing familial engagement and support, which may buffer the impact of high-EE attitudes.

Generally, there are three approaches to testing mechanisms of change in treatment research. The first is to gather measures on proposed mechanisms to determine if changes in those mechanisms correlate with outcomes (Kraemer, Wilson, Fairburn, & Agras, 2002). Because treatment manuals typically cover a range of highly correlated treatment targets, statistical clarity on such mechanisms is a difficult challenge. A second approach has been to dismantle well-working manuals to offer specific components, often with surprising results (see, for example, Jacobson et al., 1996; Dimidjian et al., 2006). Third, one can develop specific building blocks that target specific mechanisms, test whether they are clinically effective, and then work towards optimal combinations. This building block approach allows clinicians more flexibility in assessing the presence of key risk factors and choosing targeted interventions (Persons, 2005; Westen et al., 2004). Thus, one might implement the Nusslock et al approach by developing a treatment whose primary purpose is to address deficits in goal regulation, show that it works on the hypothesized target, and then show that modifying the target leads to better symptomatic outcomes.

In a recent study, Johnson and Fulford (2008) reported on the goals manual, an individual treatment designed to address four components of goal dysregulation as a mania prevention intervention: emotion dysregulation, overly ambitious goal setting, bursts of confidence and excessive goal engagement. In a 13-week open trial ($n = 10$), the intervention successfully produced decrements in mania and overly ambitious goal setting. This work dovetails well with many of the conceptual statements in the Nusslock et al. article. However, a controlled trial with a longer follow-up will be necessary to determine whether interventions focused on goal dysregulation are more effective than other treatments (for example, psychoeducation) which have been shown to stabilize depression and mania symptoms.

In identifying treatment targets for BD, we need to be attuned to issues of sampling. As noted above, many facets of the goal regulation model have been supported in bipolar I samples (for review, see Johnson, 2005). Other studies cited by Nusslock et al. concern college students who score high on one or more measures of mood lability, hypomanic personality, or cyclothymia. The majority of these subjects have never been treated for bipolar disorder. When they experience their first onset, it is often a hypomanic episode or a very short manic episode, after which they receive diagnoses such as “bipolar disorder not otherwise specified.” Can we generalize from college student samples to clinical populations of bipolar I patients? College students may be more achievement oriented, goal-driven, and prone to chaotic sleep/wake cycles than patient samples. College student studies of BD certainly have their place, but we have to consider that the triggers for hypomanic behavior, hypomanic episodes, manic symptoms, and full manic episodes may be quite different in clinical versus analog samples.

In applying the goal dysregulation model to more severe bipolar I or II patients, we must determine how well the findings generalize across variable courses of illness. Consider the case description provided by Nusslock et al: “The individual begins making risky financial decisions, takes on more work than she can handle, and engages in excessive pleasurable activities...because she is so overextended, she fails to meet professional/familial obligations; she becomes aware of the financial and/or personal implications of her impulsive decisions, and, as a result, experiences significant failure in the achievement domain (page #).” It is less clear how these processes change with multiple manic or depressive episodes, losses,

hospitalizations, or changes in social networks and occupational roles. Research has established that many of the facets of reward sensitivity diminish when a person experiences even mild depressive symptoms (Meyer et al., 2001). What role, then, should goal dysregulation interventions play in the treatment of a single, chronically depressed woman with bipolar II disorder for whom hypomania, when it is occurs, is a brief and enjoyable state that interrupts the “slow burn” of her ongoing depression? Or a service-connected, socially isolated veteran with bipolar I disorder who struggles with frequent depressive recurrences, interepisode dysthymia, comorbid substance abuse, and neuropsychological difficulties?

To gain an appreciation of the significance of this issue, consider the results of the National Institute of Mental Health Collaborative Depression Study, a 12.8 year follow-up of 146 bipolar patients. In that study, the ratio of weeks spent in depressed versus hypomanic states was 3:1 among bipolar I patients and 37:1 among bipolar II patients (Judd et al., 2002, 2003). In the Systematic Treatment Enhancement Program for Bipolar Disorder, a naturalistic follow-up of 1,469 symptomatic bipolar I and II patients, 416 (48.5%) experienced recurrences over the next 2 years, with more than twice as many developing depressive episodes (34.7%) as manic, hypomanic, or mixed episodes (13.8%) (Perlis et al., 2006). Thus, before recommending “one size fits all” approaches to treatment, we need to determine which subpopulations are most likely to benefit from which treatment strategies. Such subpopulations are often obscured in omnibus comparisons of treatment and control groups.

Finally, Nusslock et al. note that CBT and other psychosocial interventions should highlight “the potential value of targeting achievement-oriented cognitive profiles in managing both the depressive and manic phases of BD (page #).” Jamison (1993, 1995, 2005) has highlighted the importance of creative pursuits, exuberance, and productivity as central to many patients’ (including her own) eventual recovery from BD. Can a short-term risk factor such as excessive goal pursuit operate as a protective factor over the long-term course of the illness? Do these same temperamental attributes help patients get through the illness and recover? Cicchetti and Rogosch (1996) point out that risk factors at one point in the life cycle can be protective factors at another. We need to be sure that in protecting the patient from immediate recurrences, we do not dissuade her from using her natural talents and abilities in achieving goals that are realistic in the context of a chronic illness.

We commend Nusslock et al for their helpful integrative review. It is exciting to see this group of authors and others take their laboratory observations into the clinical context, and determine when they can and cannot be translated into effective treatment interventions.

Acknowledgments

Preparation of this article was supported in part by National Institute of Mental Health grants MH073871 and MH077856 to Dr. Miklowitz.

References

- Cicchetti D, Rogosch FA. Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology* 1996;8:597–600.
- Dimidjian S, Hollon SD, Dobson KS, Schmaling KB, Kohlenberg RJ, Addis ME, et al. Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression. *Journal of Consulting and Clinical Psychology* 2006;74:658–670. [PubMed: 16881773]
- Frank E, Kupfer DJ, Thase ME, Mallinger AG, Swartz HA, Fagiolini AM, et al. Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar I disorder. *Archives of General Psychiatry* 2005;62:996–1004. [PubMed: 16143731]

- Fredman S, Baucom DH, Miklowitz DJ, Stanton S. Observed emotional involvement and overinvolvement in families of bipolar patients. *Journal of Family Psychology* 2008;22:71–79. [PubMed: 18266534]
- Hooley JM, Gotlib IH. A diathesis-stress conceptualization of expressed emotion and clinical outcome. *Applied and Preventive Psychology* 2000;9:131–151.
- Jacobson NS, Dobson KS, Truax PA, Addis ME, Koerner K, Gollan JK, et al. A component analysis of cognitive-behavioral treatment for depression. *Journal of Consulting and Clinical Psychology* 1996;64:295–304. [PubMed: 8871414]
- Jamison, KR. *Touched with fire: manic-depressive illness and the artistic temperament*. New York: Maxwell Macmillan International; 1993.
- Jamison, KR. *An Unquiet Mind*. New York: Alfred A. Knopf, Inc.; 1995.
- Jamison, KR. *Exuberance: the passion for life*. Vintage publications; 2005.
- Johnson SL. Mania and dysregulation in goal pursuit. *Clinical Psychology Review* 2005;25:241–262. [PubMed: 15642648]
- Johnson SL, Cuellar A, Ruggero C, Perlman C, Goodnick P, White R, et al. Life events as predictors of mania and depression in bipolar I disorder. *Journal of Abnormal Psychology* 2008;117:268–277. [PubMed: 18489203]
- Johnson SL, Eisner L, Carver CS. Elevated expectancies among persons diagnosed with bipolar disorders. *British Journal of Clinical Psychology* 2009;48:217–222. [PubMed: 19254445]
- Johnson, SL.; Eisner, L.; Fulford, D. Psychological mechanisms in bipolar disorder. In: Salzinger, K.; Serper, MR., editors. *Behavioral Mechanisms and Psychopathology: advancing the explanation of its nature, cause, and treatment*. Washington, D.C.: American Psychological Association Press; 2009. p. 77-106.
- Johnson SL, Fulford D. Preventing mania: A preliminary examination of the GOALS program. *Behavior Therapy*. 2008 10.1016/j.beth.2008.03.002.
- Johnson SL, Sandrow D, Meyer B, Winters R, Miller I, Solomon D, et al. Increases in manic symptoms following life events involving goal-attainment. *Journal of Abnormal Psychology* 2000;109:721–727. [PubMed: 11195996]
- Judd LL, Akiskal HS, Schettler PJ, Coryell W, Endicott J, Maser JD, et al. A prospective investigation of the natural history of the long-term weekly symptomatic status of bipolar II disorder. *Archives of General Psychiatry* 2003;60:261–269. [PubMed: 12622659]
- Judd LL, Akiskal HS, Schettler PJ, Endicott J, Maser J, Solomon DA, et al. The long-term natural history of the weekly symptomatic status of bipolar I disorder. *Archives of General Psychiatry* 2002;59:530–537. [PubMed: 12044195]
- Kraemer HC, Wilson T, Fairburn CG, Agras WS. Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry* 2002;59:877–883. [PubMed: 12365874]
- Lam D, Wright K, Sham P. Sense of hyper-positive self and response to cognitive therapy in bipolar disorder. *Psychological Medicine* 2005;35:69–77. [PubMed: 15842030]
- Lam DH, Hayward P, Watkins ER, Wright K, Sham P. Relapse prevention in patients with bipolar disorder: cognitive therapy outcome after 2 years. *American Journal of Psychiatry* 2005;162:324–329. [PubMed: 15677598]
- Lozano BL, Johnson SL. Can personality traits predict increases in manic and depressive symptoms? *Journal of Affective Disorders* 2001;63:103–111. [PubMed: 11246086]
- Meyer B, Johnson SL, Winters R. Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of Psychopathology and Behavioral Assessment* 2001;23:133–143.
- Miklowitz DJ, Scott J. Psychosocial treatments for bipolar disorder: Cost-effectiveness, mediating mechanisms, and future directions. *Bipolar Disorders*. (in press).
- Parikh, S.; Zaretsky, A.; Beaulieu, S. Psycho-education versus cognitive behavioural therapy for bipolar disorder: A multi-site national RCT (Abstract NR322). Paper presented at the Annual Meeting of the American Psychiatric Association; Washington, D.C.; 2007.
- Perlis RH, Ostacher MJ, Patel J, Marangell LB, Zhang H, Wisniewski SR, et al. Predictors of recurrence in bipolar disorder: primary outcomes from the Systematic Treatment Enhancement Program for

- Bipolar Disorder (STEP-BD). *American Journal of Psychiatry* 2006;163:217–224. [PubMed: 16449474]
- Persons JB. Empiricism, mechanism, and the practice of cognitive-behavior therapy. *Behavior Therapy* 2005;36:107–118.
- Scott J, Paykel E, Morriss R, Bentall R, Kinderman P, Johnson T, et al. Cognitive behaviour therapy for severe and recurrent bipolar disorders: a randomised controlled trial. *British Journal of Psychiatry* 2006;188:313–320. [PubMed: 16582056]
- Simoneau TL, Miklowitz DJ, Richards JA, Saleem R, George EL. Bipolar disorder and family communication: Effects of a psychoeducational treatment program. *Journal of Abnormal Psychology* 1999;108:588–597. [PubMed: 10609423]
- Wendel JS, Miklowitz DJ, Richards JA, George EL. Expressed emotion and attributions in the relatives of bipolar patients: An analysis of problem-solving interactions. *Journal of Abnormal Psychology* 2000;109:792–796. [PubMed: 11196006]
- Westen D, Novotny CM, Thompson-Brenner H. The empirical status of empirically supported psychotherapies: assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin* 2004;130:631–663. [PubMed: 15250817]
- Zaretsky A, Lancee W, Miller C, Harris A, Parikh SV. Is cognitive-behavioural therapy more effective than psychoeducation in bipolar disorder? *Canadian Journal of Psychiatry* 2008;53:441–448.