### Title

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# Working Memory Interacts with Emotion Regulation to Predict Symptoms of Mania

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## Abstract

Although neurocognitive deficits and emotion regulation are closely linked within unipolar depression, little research has tested links between these two variables within bipolar disorder. The present study tested whether working memory is related to individual differences in emotion regulation strategies in bipolar disorder and whether working memory and emotion regulation can explain variability in symptoms over time. Fifty-nine euthymic adults with bipolar I disorder completed a working memory span task, symptom interviews assessing depression and mania, and questionnaires assessing brooding rumination, reappraisal, and suppression. At baseline, working memory was unrelated to emotion regulation. Symptom interviews were repeated at six months ($n$ = 41) and 12 months ($n$ = 36) follow-up. At 12 months, baseline working memory significantly interacted with baseline suppression to predict higher mania. Tests of simple slopes showed that at lower working memory levels, low use of suppression was associated with significantly greater mania symptoms. These results help to clarify previous inconsistent findings regarding cognitive functioning and emotion regulation strategies in bipolar disorder, suggesting that deficits in both domains combine to predict outcomes.

### Keywords

bipolar disorder; suppression; reappraisal; brooding

## 1. Introduction

Bipolar I disorder, defined by at least one lifetime episode of mania, has profound consequences for psychosocial functioning. In addition to episodes of mania, many individuals with bipolar I disorder also experience significant symptoms of depression (Cuellar, Johnson, & Winters, 2005). Given that mood episodes are highly recurrent in bipolar disorder (Perlis et al., 2006), there is a significant need to understand inter-episode features that predict symptomatic increases in this population. In the present study, we tested

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the influence of two such features—emotion regulation and working memory—on subsequent symptoms of mania and depression.

Extensive research has identified and differentiated various emotion regulation strategies. Considerable evidence supports the notion that some of these strategies are generally adaptive (such as cognitive reappraisal, or changing emotions by changing how one thinks about a situation) and others are more generally maladaptive (such as expressive suppression, or attempting to suppress visible signs of emotion) (John & Gross, 2004). Theories of emotion regulation posit that both the selection and the effectiveness of such strategies are influenced by contextual factors and by personal resources (Urry & Gross, 2010). One such resource thought to influence emotion regulation is working memory capacity (Opitz et al., 2012). Below, we review research supporting the importance of emotion regulation in bipolar disorder and the premise that working memory capacity could be an important influence on emotion regulation strategy use in bipolar disorder.

### 1.1 Emotion Regulation in Bipolar Disorder

Emotion regulation difficulties are widely documented among people with bipolar disorder (for review, see Dodd et al., 2019). There is some evidence those with bipolar disorder use reappraisal less than healthy participants do (Johnson et al., 2016; Gul & Khan, 2014; Rowland et al., 2013a; Rowland et al., 2013b; Wolkenstein et al., 2014; although see Green et al., 2011). Findings are more consistent regarding maladaptive strategies. Compared to those without the disorder, those with bipolar disorder tend to more frequently use suppression (Gruber, et al., 2012; Gul & Khan, 2014), rumination (Green et al., 2011; Gruber et al., 2008; Gruber et al., 2011; Rowland et al., 2013a; Thomas et al., 2007), and dampening of positive affect (Edge et al., 2013; Gruber et al., 2011; Johnson et al., 2008).

People with bipolar disorder vary in their emotion regulation profile; however, one relatively consistent finding is that greater use of maladaptive emotion regulation strategies is linked to problematic outcomes, including frequency and severity of depression (Green et al., 2011; Gruber et al., 2011; Rowland et al., 2013a; Van Rheenen et al., 2015), severity of mania (Van Rheenen et al., 2014; Van Rheenen et al., 2015), and lower quality of life (Edge et al., 2013). Of import, use of maladaptive emotion regulation strategies during remission also predicts increases in depressive and manic symptoms over time (Gilbert et al., 2013). Therefore, a better understanding of variables that influence adaptive and maladaptive emotion regulation during remission may help understand risk for future mood symptoms.

### 1.2 Cognitive Influences on Emotion Regulation

In normative populations, there is evidence for links of working memory with emotion regulation strategies (Buhle et al., 2014; Schmeichel & Tang, 2015). Findings link working memory capacity with reappraisal ability (McRae et al., 2012; Hendricks & Buchanan, 2016; Schmeichel et al., 2008), use of spontaneous emotion regulation (Schmeichel & Demaree, 2010) and more effective use of suppression (Hendricks & Buchanan, 2016; Schmeichel et al., 2008). Other evidence shows that in people with depression, working memory deficits are related to rumination (for review, see Joormann & Vanderlind, 2014).

Thus, working memory appears to contribute to use and effectiveness of several different emotion regulation strategies.

### 1.3 Cognition and Emotion Regulation in Bipolar Disorder

Given the evidence for emotion regulation difficulties within bipolar disorder and the evidence of executive functioning as a key determinant of emotion regulation outcomes, it has been posited that neurocognitive deficits may contribute to problems with emotion regulation in bipolar disorder (Green et al., 2007; Lima et al., 2018). This idea draws support from empirical findings showing that neurocognitive measures (including working memory) and emotion dysregulation conjointly predict psychosocial functioning in bipolar disorder (Van Rheenen & Rossell, 2014)—indicating the importance of understanding the unique and conjoint influences of these two domains on outcomes. Other evidence illustrates the pervasiveness of a broad range of cognitive deficits among people with bipolar disorder and their family members, including working memory, verbal memory, processing speed, and sustained attention (Bourne et al., 2013; Lee et al., 2013). Neuroimaging studies also provide consistent evidence for alterations in the underlying cognitive control networks thought to support emotion regulation among those with bipolar disorder (for review, see Green et al., 2007). While many facets of cognition worsen during mood episodes, deficits remain clearly present during remission (Martínez-Arán et al., 2004a; Martínez-Arán et al., 2004b). These cognitive deficits are associated with a range of poor outcomes, including impairments in life functioning (Bowie et al., 2010; Martínez-Arán et al., 2004b) and quality of life (Brissos et al., 2008).

Despite the frequently assumed connection between neurocognitive deficits and emotion regulation and the neurobiological evidence supporting such models (e.g., Green et al., 2007), relatively little research has considered links of neurocognitive deficits with emotion regulation strategies in bipolar disorder (Lima, et al., 2018). That is, despite theory, few studies have tested direct relationships of neurocognitive measures with specific emotion regulation strategies. Findings have not been consistent in the few studies in this domain. In one study, neurocognitive deficits (inclusive of working memory) did *not* significantly predict emotion dysregulation in euthymic bipolar patients (Van Rheenen et al., 2014); in contrast, task-switching capacity related to significantly more trait-like tendencies to use reappraisal among euthymic bipolar patients in another study (Gul & Khan, 2014).

### 1.4 Aims and Hypotheses

The present study was designed to test links between working memory and emotion regulation in remitted bipolar I disorder and to test how difficulties in those two domains uniquely and conjointly predict symptoms of depression and mania longitudinally. To accomplish this aim, we recruited adults with bipolar I disorder and followed them over time until they achieved symptomatic remission, at which point we assessed working memory, reappraisal, and brooding. Participants completed a well-validated measure of working memory, symptom interviews evaluating depression and mania (at baseline, six - and twelve-month follow-up), and a battery of emotion regulation measures encompassing putatively adaptive (reappraisal) and maladaptive (brooding rumination, suppression) strategies that have each been empirically linked with working memory. In selecting these measures, we

considered research showing that successful use of suppression is related to stronger performance on working memory tasks (Hendricks & Buchanan, 2016; Schmeichel et al., 2008). Although we evaluated use of suppression rather than its effectiveness, we predicted that working memory and suppression use would be linked given that strategy use is informed by the perceived effectiveness of that strategy (cf. Scheppes et al., 2014). Thus, we hypothesized that: (1) higher working memory capacity would correlate cross-sectionally with less brooding rumination and more reappraisal and suppression, and (2) greater working memory capacity, greater reappraisal, and less rumination and suppression would predict fewer mood symptoms among people with bipolar disorder over time.

## 2. Methods

### 2.1 Participants

Fifty-nine individuals with bipolar I disorder were recruited from a community sample in the San Francisco Bay Area using online advertisement, flyers, and clinical referrals. Inclusion criteria included age between 18 and 60 and English language fluency. Exclusion criteria included daily substance use or diagnosis of abuse or dependence in the past six months, brain injury or disease, medical conditions impairing the central nervous system, impaired mental status, developmental disability, or electroconvulsive treatment in the past 18 months. Individuals with a primary psychotic disorder were excluded from participation; however, psychotic symptoms were not an exclusion criterion if these symptoms occurred solely within a mood episode. Participants also reported their current medications and dosages (there were no exclusions for medication classes). Participants were recruited as part of a larger study. In one previous manuscript, we reported symptom and emotion regulation measures, but did not consider the working memory task (Johnson et al., 2016). All participants provided written informed consent, and all study procedures were approved by the university's Institutional Review Board.

### 2.2 Procedures

Potential subjects completed a telephone screening to determine likely eligibility status based on inclusion and exclusion criteria. After the initial screening, potential participants were scheduled for an in-person appointment to complete written informed consent procedures and the Structured Clinical Interview for DSM-IV (First et al., 1996). Medication status was also assessed at this meeting (for more detail, see Table 1). Then, monthly telephone interviews consisting of the Modified Hamilton Rating Scale for Depression (MHRSD) and the Young Mania Rating Scale (YMRS) were conducted to cover past month symptoms until symptoms remitted. Telephone interviews have previously been shown to be a reliable and valid method for evaluating symptom severity (Simon et al., 1993). Participants were considered remitted if symptoms were less than or equal to 6 on both the MHRSD and the YMRS for at least one month (Chengappa et al., 2003). Participants' remitted status was confirmed two days before returning to the university for a second in-person assessment, in which they completed working memory and self-report measures described below, administered by a trained graduate student or research staff member.

Participants completed 6- and 12-month follow-up telephone interviews of the MHRSD and the YMRS. Of the original cohort of 59 participants, 41 participants completed the HRSD at the 6-month symptom follow-up (37 completed the YMRS), and 36 completed the HRSD at 12 months (32 completed the YMRS). Follow-up data were unavailable for various reasons, including that participants were no longer available at their original telephone number or were no longer interested in participation. Four participants were not able to provide detailed information for follow-up YMRS interviews, and their data was coded as missing. Participants who did and did not complete the follow-up interviews did not differ from one another in terms of age, gender, years of education, or baseline symptom severity ($t$s < 1.5, $\chi^2$s < 1.0, $p$s > .1). Only three participants at 6-months and two participants at 12-months exhibited MHRSD symptom scores of 17 or greater, the traditional cut-off for clinical severity (Miller et al., 1985). Likewise, only one participant exhibited a score that was within the manic range (≥ 21; Suppes et al., 2005) at either the 6- or 12-month follow-ups.

## 2.3 Measures

### 2.3.1 Structured Clinical Interview for DSM-IV (SCID-IV).—

The SCID-IV is a widely used and well-validated semi-structured interview for assessing psychiatric diagnoses (First et al., 1996). Interviews were administered by clinical psychology graduate students and postdoctoral fellows who completed thorough didactic and role-play training before administering study SCIDs. Inter-rater agreement was established using a random selection of ten audio recorded interviews and was found to be excellent (intraclass $r$s = .88-.89 for current mania, lifetime mania, lifetime major depressive episode, .99 for current major depressive episode, and 1.00 for mania and depression symptoms). Interviewers also completed the SCID modules for alcohol and substance use disorder and psychotic disorder to assess exclusion criteria.

### 2.3.2 Young Mania Rating Scale (YMRS).—

We used the YMRS, a widely used clinician-rated semi-structured interview, to assess past-month manic symptom severity (Young et al., 1978). The YMRS consists of 11 items with scores ranging from 0-60. The YMRS has previously been shown to correlate with other indices of mania and with clinician ratings and with other clinically relevant outcomes (Young et al., 1978). Based on four randomly selected audio recordings, inter-rater agreement within our team for the YMRS was excellent (intraclass $r$ > .99).

### 2.3.3 Modified Hamilton Rating Scale for Depression (MHRSD).—

We used the MHRS, one of the most frequently used semi-structured depression severity interviews, to assess past-month depression symptom severity (Miller et al., 1985). The MHRSD scores ranging from 0-52. The MHRSD has been validated against other indices of depression, including the SCID, and has been specifically validated in bipolar samples (Johnson et al., 2008). The MHRSD has previously been shown to have good sensitivity for changes in depression symptoms over time (Keitner et al., 1992). Inter-rater agreement based on a random selection of four tapes was excellent (intraclass $r$ > .99).

### 2.3.4 Emotion Regulation Questionnaire (ERQ).—

The ERQ is the most widely used measure of trait-like tendencies to use cognitive reappraisal and suppression (Gross &

John, 2003). Participants are asked to respond to each of 10 items on a 7-point scale ranging from 1 (strongly disagree) to 7 (strongly agree). The reappraisal subscale reflects tendencies to change cognitions about an emotion-eliciting situation (6 items, e.g., “I control my emotions by changing the way I think about the situation I’m in”). The suppression subscale reflects the tendency to inhibit emotional expressivity (4 items, “I control my emotions by not expressing them”). The ERQ has been validated in BD samples (Gruber et al., 2012).

#### 2.3.5 Ruminative Responses Scale-Brooding Subscale (RRS).—

The RRS is a widely used measure of rumination on negative affect developed to disentangle rumination from symptoms of depression (Treynor et al., 2003). We used the Brooding subscale, which includes five items (e.g., do you typically ‘think about “What am I doing to deserve this?”’ when you experience negative affect?) that are rated on 4-point scale (1 = almost never; 4 = almost always). The brooding component of rumination has been identified as a maladaptive component of rumination across multiple psychopathologies (Aldao et al., 2010).

#### 2.3.6 WAIS-IV Backwards Digit Span Task (Wechsler, 2008).—

The Backwards Digit Span task was administered as an assessment of verbal working memory capacity. The task evaluates ability to recall numbers in reverse order of presentation. The Backwards Digit Span Task has been shown to be sensitive to working memory impairment in bipolar disorder and was identified in a meta-analysis as the executive function measure that most strongly differentiated bipolar and control samples (Bourne et al., 2013). Each block of the digit span task included two strings of digits of the same length (beginning at two digits); the task was discontinued when participants were unable to correctly recall both trials of a given block. We administered up to 16 trials (eight sets ranging from 2 to 8 digits). The score was calculated as the number of trials successfully completed (range: 0 to 16).

### 2.4 Analysis Plan

Data were analyzed using SPSS version 24.0. Distributions were graphed and checked for normality before conducting analyses. Paired t-tests with Cohen’s *d* effect size estimates were used to evaluate change in symptoms over time. For tests of hypotheses, Pearson correlations were used to test cross-sectional relationships of working memory with emotion regulation strategy use. Hierarchical linear regression was used to test the hypothesis that working memory, emotion regulation, and their combination would predict increased symptoms of mania and depression at follow-up. These analyses comprised three parallel models at each timepoint (6- and 12-month) for the DV of depression, and three at each timepoint for the DV of mania to examine the effects of Brooding, Reappraisal, and Suppression, each in conjunction with working memory. Centered interaction terms were calculated for working memory with each of the three emotion regulation scores, and these interaction terms were tested using forward selection in the final step. Simple slopes analysis, using procedures outlined by Aiken et al. (1991), was used to decompose significant interactions. To adjust for multiple comparisons, a Bonferroni correction was applied to all analyses. For cross-sectional analyses, individual tests were set to alpha = .003. Corrected error rates for regression analyses (based on the number of predictors in each model) ranged from alpha = .0125 to alpha = .0167.

## 3. Results

Demographic and clinical information for the full sample ($N = 59$) are presented in Table 1. Participants reported taking between 0 and 4 medications. Before testing hypotheses, potential demographic (age, gender, education level) and clinical (number of psychiatric medications, number of depressive episodes, number of manic episodes) confounds were examined as correlates of WM and emotion regulation. These effects were not significant ($p$s > .17), with the following exceptions. Higher brooding rumination scores correlated negatively with age, $r = -.50$, $p < .001$, and higher reappraisal correlated negatively with number of past manic episodes, $r = -.33$, $p = .015$. Women reported significantly higher levels of brooding than did men, $t(56) = -2.196$, $p = .03$; gender was therefore entered as a covariate in relevant models below.

### 3.1 Cross-Sectional Correlations

Table 2 shows Pearson correlations of baseline WM with emotion regulation variables. Contrary to hypotheses, working memory was not significantly related to any of the emotion regulation measures when controlling for multiple comparisons. (Depression symptoms were non-significantly related to higher brooding and suppression scores). To assess potential effects of medication, analyses in Table 2 were repeated as partial correlations controlling for the number of psychiatric medications participants reported taking. Results were parallel to Table 2.

### 3.2 Six- and Twelve-Month Analyses

As would be expected given that participants were required to be asymptomatic at baseline, both depression and mania symptoms significantly increased from the baseline to the 6-month follow-up assessment ($p$s < .001); symptom scores remained significantly elevated, compared to baseline, at the 12-month follow-up ($p$s < .001). Results of the regression analyses of 6- and 12-month assessments are presented in Table 3.[1]

**3.2.1 Depression.—**Controlling for baseline depression, emotion regulation (brooding, reappraisal, or suppression), WM, and the interaction of emotion regulation x WM did not account for a significant proportion of variance in depression measured at 6-month follow-up (all $p$s > .065). However, each of the three models predicting depression at 12-month follow-up were significant, accounting for 29 to 40% of the variance in symptoms ($p < .01$). In each model, higher baseline depression predicted significantly higher follow-up depression scores. For the brooding model, female gender predicted higher follow-up depression scores ($p = .01$). In the model of brooding, controlling for multiple comparisons, better WM performance predicted higher depression symptoms ($p < .01$). No emotion regulation strategy interacted significantly with WM.

[1]To assess potential medication effects, all regression analyses were also conducted with number of medications entered as a predictor in the first block. Sample size was reduced by 2-3 participants due to missing medication data. The significance and direction of all effects in all models remained the same when this correction was made. Given that the sample was remitted, we also conducted all regression analyses without baseline MHRSD or YMRS included, and the significance and direction of effects were similarly unchanged.

**3.2.2 Mania.—**Parallel with findings for depression, the overall models predicting mania symptoms at 6 months were not significant for reappraisal or suppression. As with the models predicting depression, female gender predicted higher follow-up mania scores in the brooding model ($p < .01$). In the prediction of mania at 12 months, controlling for baseline mania (and gender within the brooding model), only one of the emotion regulation and cognition variables was significant: the interaction between working memory performance and suppression. Analysis of simple slopes tested the effect of mean working memory levels, as well as working memory performance 1 standard deviation above and below the mean. These analyses showed that lower suppression predicted greater symptoms of mania only when working memory performance was low, $b = -4.52$, $t = -3.855$, $p = .001$, not when working memory performance was moderate ($p = .15$) or high ($p = .10$).[2] Figure 1 shows the effects of low (−1 SD) and high (+1 SD) working memory levels on suppression scores predicting follow-up YMRS scores.

## 4. Discussion

People with bipolar disorder often show cognitive and emotion regulation deficits, and both domains are important correlates of outcomes. Surprisingly, however, these two domains are rarely studied conjointly. We examined (1) whether performance on a well-validated working memory task related to concurrent emotion regulation strategies during remission and (2) the main and interactive effects of emotion regulation and WM on changes in symptoms of depression and mania at follow-up. Methodological strengths of this study include the use of a carefully diagnosed sample of adults followed until remission, use of well-validated working memory and emotion regulation measures, and longitudinal data collection for 12 months.

Despite multiple papers arguing for the link between these domains, we found little support for our hypotheses: working memory performance did not relate to frequency of use of emotion regulation tendencies. With the exception of suppression, low working memory performance did not amplify the effects of emotion regulation problems on symptoms. Lower working memory performance in the context of low use of suppression did predict greater increase in mania symptoms.

In one of the models (brooding rumination and depression), the main effect of working memory performance at baseline paradoxically related to greater increase in depression symptoms at the 12-month follow-up. The direction of effect was unexpected given the robust literature on working memory deficits within bipolar disorder (e.g., Lee et al., 2013) and within depression more broadly (Snyder, 2013). The effect we observed is potentially consistent with prior work showing that higher levels of rumination may correlate with better performance on certain cognitive control tasks (Altamirano et al., 2010). However, given the modest sample size, the unexpected nature of the finding, and the lack of consistency across models, replication of this effect is needed before further mechanistic explanations should be considered.

[2]We also tested 4 regression models (predicting mania and depression at 6 and 12 months) with all three emotion regulation strategies included in each model. Results of these analyses were parallel with primary analyses, and the interaction between suppression and working memory remained significant when brooding, reappraisal, and medications were controlled for, $\beta = .61$, $t = 3.76$, $p < .001$.

Our findings that working memory performance was not significantly related to any of the three emotion regulation strategies we assessed is consistent with findings reported by Van Rheenen and colleagues (2014), who found that an aggregated neurocognition score was unrelated to a composite emotion regulation measure in a bipolar sample. This absence of a correlation between working memory and brooding rumination is consistent with recent findings suggesting that links between working memory and rumination may only reliably emerge under conditions of stress (Quinn & Joormann, 2015). We may have missed important patterns with our focus on working memory during remission: emotion regulation strategies may be used more frequently during symptomatic periods, and neurocognition appears to become more impaired during mood episodes (Martínez-Arán et al., 2004). Future research could test the hypothesis that stress-induced or mood-related changes in cognitive performance contribute to dynamic difficulties in emotion dysregulation in bipolar disorder. This research would also benefit from inclusion of measures of life events to better understand contextual factors that may give rise to use of certain emotion regulatory strategies (Johnson & Roberts, 1995).

In contrast to these null findings at baseline, working memory performance and suppression significantly interacted in the prediction of symptoms over time. Specifically, a combination of lower use of suppression and lower working memory capacity predicted increases in mania symptoms. This finding is potentially consistent with previous research suggesting that working memory capacity is needed for successful use of suppression (e.g., Schmeichel et al. 2008; Hendricks & Buchanan, 2016). Suppression used by those without adequate working memory may be more prone to failure, which may be conducive to the emergence of mania symptoms. Of note, this relationship between suppression and working memory emerged only in the prediction of mania at 12-month follow-up, with no significant effect at 6 months or for depression.

Although unexpected, the finding that suppression use may be tied to lower mania has some grounding in the literature. Despite the frequent finding of suppression relating to maladaptive outcomes (Gross & John, 2003; John & Gross, 2004; Srivastava et al., 2009), the consequences of suppression are likely contingent on contextual factors (Opitz et al., 2012; Troy et al., 2013) and may not be tied to maladaptive outcomes in some contexts (e.g., Soto et al., 2011). Expressive suppression is frequently used in a social context (Gross & John, 2003), and the ability to manage emotions in social settings appears to be a particular challenge for those with bipolar disorder, with evidence for difficulties in perceiving emotions in social contexts (Donohoe et al., 2012), higher levels of inappropriate emotion expression (e.g., aggression; Ballester et al., 2014), and overall deficits in social support (Romans & McPherson, 1992). Although speculative, the combination of highly intense emotion experiences (Johnson et al., 2016) coupled with social cognition problems (Donohoe et al., 2012) may make subtle modulation of emotion expression more complicated for those with bipolar disorder. Those who are able to use even this blunt approach to emotion expression may be better poised to cope with their emotions in complex settings, helping to explain their decreased risk of manic symptoms over time. That this effect was not observed at 6-month follow-up suggests that other unmeasured variables influencing mania at the follow-up assessments (e.g., motivation to manage symptoms,

changes in relationship challenges) likely moderate the degree to which the influence of working memory and suppression are observed.

In contrast to hypotheses and to previous findings regarding the benefits of reappraisal in the general population (e.g., John & Gross, 2004) and in bipolar disorder (e.g., Gruber et al., 2014), reappraisal was not related to lower depression or mania symptoms over time, regardless of working memory levels. Future research would benefit from inclusion of emotion regulation effectiveness measures in addition to the frequency measures we used. In nonbipolar samples, cognitive resources are important contributors to the effectiveness of suppression (Schmeichel & Tang, 2015), and mood symptoms are more tied to the efficacy than the frequency of emotion regulation strategies (Ford et al., 2017). Understanding these ties in bipolar disorder may be especially important, given recent findings that those with bipolar disorder may particularly struggle with lowered perceived efficacy of emotion regulation (Gruber et al., 2012).

It is important to acknowledge several other limitations. First and most significantly, power was limited for analyses of symptoms over time and particularly for tests of interaction effects. Given the modest sample size, it will be important to replicate the significant interaction of working memory and expressive suppression. Second, although well-validated, the Digit Span task does not capture all components of working memory (e.g., updating), nor did we assess processes such as inhibition and shifting. Working memory measures that are more complex (such as the N-back task) than simple span tasks and require additional cognitive resources may help to understand which components of working memory are specifically related to emotion regulation use in bipolar disorder. Third, we did not assess emotion regulation and working memory measures at the follow-up assessments and so could not consider the influence of dynamic changes in these domains on symptoms. Fourth, the absence of a comparison psychiatric group limits our ability to determine how unique the effects of suppression and working memory may be compared to those observed in other disorders.

Taken together with several previous studies showing no evidence of cross-sectional correlations between working memory and emotion regulation, the present finding suggests the need for more sophisticated theories of how variability in cognitive functioning relates to emotion regulation strategies in bipolar disorder. We find little evidence for the idea that emotion regulation is simply a proxy for poor working memory. Given the significant heterogeneity in cognition, symptoms, and illness course in individuals with bipolar disorder, it may be difficult to formulate hypotheses derived from either the unipolar depression literature or findings in individuals without mood disorders. Future research would do well to consider how core processes implicated in bipolar disorder—such as reward sensitivity, circadian rhythm dysregulation, or emotion-relevant impulsivity—influence and are influenced by variability in cognitive functioning and emotion regulation. A better understand of such processes could lead to more efficient treatments for emotion dysregulation in this disorder.

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## Highlights

- Performance on a working memory task was not correlated with use of emotion regulation strategies in euthymic bipolar disorder.
- Working memory and expressive suppression interacted to predict higher mania symptoms at 12-month follow-up.
- At lower levels of working memory, low use of suppression was associated with greater symptoms of mania at follow-up.

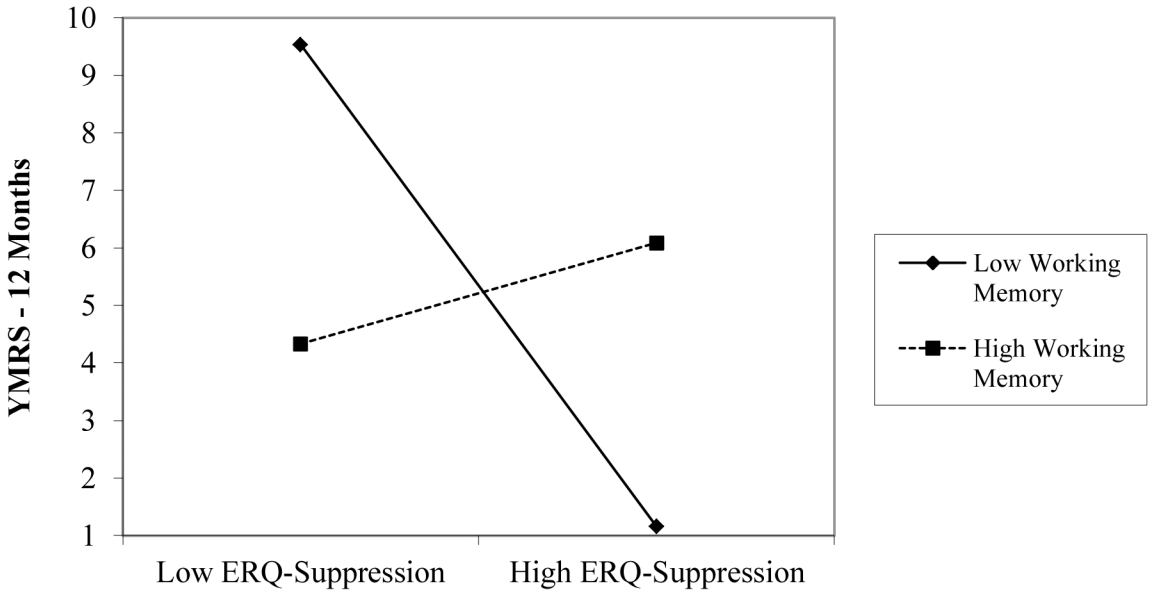


**Figure 1. Interaction Between Working Memory and ERQ-Suppression in Predicting 12-month YMRS, controlling for baseline YMRS (n = 32)**

*Note*. YMRS = Young Mania Rating Scale; ERQ = Emotion Regulation Questionnaire. "Low" Working Memory refers to scores 1 SD below the mean; "High" Working Memory refers to 1 SD above the mean.

**Table 1.**

Demographic and Clinical Characteristics of Sample

| **Demographic and Clinical Characteristics** | | ***n*** | **(%)** |
|---|---|---|---|
| Female | | 31 | (52.5%) |
| Male | | 28 | (47.5%) |
| Age (M, (SD) | | 35.24 | (11.56) |
| Race and Ethnicity | | | |
| Native Hawaiian/Pacific Islander | | 2 | (3.4%) |
| Asian | | 6 | (10.2%) |
| African American or Black | | 2 | (3.4) |
| White | | 45 | (76.3%) |
| Other/More than Once Race | | 4 | (6.8%) |
| Ethnicity | | | |
| Hispanic or Latino/a | | 7 | (12.1%) |
| Marital Status | | | |
| Single | | 37 | (62.7%) |
| Married or Cohabitating | 12 | (20.3) | |
| Divorced/Separated | | 10 | (16.9) |
| Occupational Status | | | |
| Currently Employed | | 25 | (43.1%) |
| Highest Level of Education | | | |
| High School/GED or less | | 4 | (6.8%) |
| Some college/Associates/Trade School | 23 | (39.0%) | |
| 4-year college graduate | 21 | (35.6%) | |
| Post-college education | 11 | (18.6%) | |
| **SCID-IV Diagnoses** | | ***n*** | **(%)** |
| Generalized Anxiety Disorder (Lifetime) | | 9 | (15.3%) |
| Social Anxiety Disorder (Lifetime) | | 13 | (22%) |
| Specific Phobia (Lifetime) | | 8 | (13.8%) |
| Obsessive-Compulsive Disorder (Lifetime) | | 9 | (15.3%) |
| Posttraumatic Stress Disorder (Lifetime) | 11 | (18.6%) | |
| Panic Disorder (Lifetime) | | 18 | (30.5%) |
| Alcohol Dependence (Lifetime) | 22 | (37.9%) | |
| Other Substance Dependence (Lifetime) | 23 | (40.4%) | |
| Anorexia (Lifetime) | | 2 | (3.4%) |
| Bulimia (Lifetime) | | 4 | (6.8%) |
| **Current Medications** | | | |
| No Medications | 13 | (22%) | |
| Lithium | 16 | (27.1%) | |
| Antidepressant Medication | | 16 | (27.1%) |
| Atypical Antipsychotic Medication | | 21 | (35.6%) |
| Benzodiazepine | | 12 | (20.3%) |

| Demographic and Clinical Characteristics | | *n* | (%) |
|---|---|---|---|
| Missing | 6 | (10.2%) | |

*Note*. Due to comorbidity and multiple medications, percentages sum to more than 100 for the Diagnosis and Medication sections.

**Table 2.**

Correlations between Variables at Baseline

| | | | | | | Mean (SD) | Range | *n* |
|---|---|---|---|---|---|---|---|---|
| **Scale** | **1** | **2** | **3** | **4** | **5** | | | |
| 1. Total Score-Reverse Digit Span | --- | | | | | 7.68 (2.11) | 4 - 13 | 59 |
| 2. RRS-Brooding | .095 | --- | | | | 13.05 (4.01) | 5 - 20 | 58 |
| 3. ERQ-Suppression | .09 | .071 | --- | | | 12.40 (5.08) | 4 - 24 | 58 |
| 4. ERQ-Reappraisal | .111 | −.260† | .107 | --- | | 28.81 (7.54) | 13 - 42 | 58 |
| 5. HRSD | .031 | .278† | .299† | .012 | --- | 2.93 (2.22) | 0 – 9 | 58 |
| 6. YMRS | −.088 | .05 | .220 | .051 | .437* | 1.59 (1.6) | 0-7 | 52 |

Note.

†$p < .05$

*$p < .003$. RRS = Ruminative Responses Scale; ERQ = Emotion Regulation Questionnaire; HRSD = Hamilton Rating Scale for Depression; YMRS = Young Mania Rating Scale. No tests were significant at the corrected Bonferroni level (alpha = .003), with the exception of the correlation between HRSD and YMRS scores.

**Table 3.**

Hierarchical Regression Analyses.

**A.** ***Hierarchical Regression Analyses with Brooding as Emotion Regulation Variable (Final Model)***

| | Depression – 6 Months *n* = 41 | | | Depression – 12 Months *n* = 36 | | | Mania – 6 Months *n* = 37 | | | Mania – 12 Months *n* = 32 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Block/Variable** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** |
| 1. Symptoms-Baseline | .26 | .09 | .08 | .38 | 0.01 | .24* | .35 | 0.03 | .05† | .46 | < .01 | .18* |
| 2. Gender | .25 | .13 | .06 | .38 | 0.01 | .09* | .55 | < .01 | .22* | .49 | < .01 | .20* |
| 3. Brooding | .15 | .34 | .03 | −.15 | 0.31 | .00 | −.07 | .65 | .00 | −.11 | 0.56 | .01 |
| 4. Digit Span | .21 | .18 | .04 | .41 | <.01 | .15* | .21 | .18 | .04 | .06 | 0.73 | .00 |
| *Model F (final adj. $R^2$)* | | 2.43 (.13) | | | 6.89 (.40)* | | | 3.72 (.23)* | | | 4.24 (.30)* | |

**B.** ***Hierarchical Regression Analyses with Reappraisal as Emotion Regulation Variable (Final Model)***

| | Depression – 6 Months *n* = 41 | | | Depression – 12 Months *n* = 36 | | | Mania – 6 Months *n* = 37 | | | Mania – 12 Months *n* = 32 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Variable** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** |
| 1. Symptoms-Baseline | .29 | .07 | .08 | .42 | < 0.1 | .24* | .23 | 0.19 | .05 | .43 | .02 | .18† |
| 2. Reappraisal | .00 | .99 | .00 | −.11 | 0.45 | < .01 | .14 | 0.43 | .02 | .10 | .57 | < .01 |
| 3. Digit Span | .18 | .26 | .03 | .35 | 0.03 | .11† | .07 | 0.66 | .01 | −.08 | .64 | < .01 |
| *Model F (final adj. $R^2$)* | | 1.52 (.04) | | | 5.82 (.29)* | | | 0.91 (.00) | | | 2.18 (.10) | |

**C.** ***Hierarchical Regression Analyses with Suppression as Emotion Regulation Variable*** **(Final Model)**

| | Depression – 6 Months *n* = 41 | | | Depression – 12 Months *n* = 36 | | | Mania – 6 Months *n* = 37 | | | Mania – 12 Months *n* = 32 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Variable** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** | ***β*** | ***P*** | **Δ*R*²** |
| 1. Symptoms-Baseline | .25 | .14 | .08 | .47 | < .01 | .24* | .19 | 0.27 | .05 | .41 | < .01 | .18* |
| 2. Suppression | .09 | .58 | .01 | −.11 | .47 | .02 | .19 | 0.27 | .04 | −.21 | 0.16 | .05 |
| 3. Digit Span | .17 | .29 | .03 | .31 | .04 | .09† | .06 | 0.71 | < .01 | .07 | 0.65 | < .01 |
| 4. SuppressionXDi gits | -- | -- | -- | -- | -- | -- | -- | -- | -- | .48 | < .01 | .21* |
| *Model F (final adj. $R^2$)* | | 1.64 (.05) | | | 5.80 (.29)* | | | 1.13 (.01) | | | 5.17 (.35)* | |

*Note:* Each variable equals one block. The ReappraisalXDigits and BroodingXDigits interaction was not significant for any model. In Depression Models, “Symptoms-Baseline”=Baseline HRSD score. In Mania Models, “Symptoms-Baseline”=Baseline YMRS score. Brooding and Digit Span were centered at their means.

† $p < .05$; not significant after Bonferroni correction.

* $p < .0125$ (4 predictors) or *$p < .0167$ (3 predictors).