Attention and psychomotor functioning in bipolar depression

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Abstract

The objective of this study was to assess psychomotor functioning and attention in individuals with bipolar disorder during the depressed phase of illness. Measures of attention and psychomotor functioning were administered to a sample of 24 bipolar I and II patients and a matched sample of healthy controls. Relative to the healthy controls, the bipolar sample demonstrated evidence of psychomotor slowing and revealed deficits on measures of effortful attention, yet demonstrated comparable performance on measures of automatic attention. In the bipolar sample, we detected significant correlations among measures of psychomotor functioning and some aspects of attention and a strong relationship between the severity of depression and psychomotor functioning, but no direct relationship between attention deficits and depressive symptomatology. These results suggest an attentional impairment during the depressed phase of bipolar disorder that may be specific to effortful processing, while automatic processes remain relatively intact. Associations among indices of attention deficits and psychomotor slowing may be indicative of similarities in the underlying neurobiology of these frequently co-occurring symptom domains in depressed individuals with bipolar disorder.

Keywords: Neuropsychology; Bipolar disorder; Depressed; Attention deficits; Psychomotor impairment; Cognition; Dopamine

1. Introduction

Neurocognitive deficits are common in bipolar depression (Martinez-Aran et al., 2004), with psychomotor and attention deficits among the most frequently reported. Impairment in these domains may be of particular importance insofar as they represent basic components of other neurocognitive functions and may contribute to deficits seen in learning, memory, and executive functions. The characterization of neurocognitive impairment in the depressed phase of bipolar illness is particularly critical given the pervasiveness of depressive symptomatology during the course of bipolar illness (Judd et al., 2002), concomitant psychosocial disability (Martinez-Aran et al., 2004), and the morbidity of this phase of illness (Judd et al., 2002).
Psychomotor changes are common in depression and include a slowing of almost all aspects of behavior and emotion (i.e., decreased rate of speech, decreased energy, decreased libido and anhedonia) (Nelson and Charney, 1981). Psychomotor retardation is a more prominent feature in patients with bipolar depression than in patients with unipolar depression (Nelson and Charney, 1981; Wolff et al., 1985; Benazzi, 2000; Mitchell et al., 2001) and may represent a particularly important prognostic trait with regard to cognitive course, as data indicate that psychomotor retardation predicts the degree of attentional impairment better than the extent or severity of depression itself (Smith et al., 1995; Lemelin and Baruch, 1998).

Psychomotor functioning and attentional control have been linked symptomatically and share common underlying neuropathology. Psychomotor retardation in depression may indicate abnormalities in specific structures and pathways in the brain (i.e., disruption in the basal ganglia and frontal lobe connections) (Peyser and Folstein, 1990), as it shares many characteristics with disorders that are known to be, at least in part, due to damage to the striate and the depletion of dopamine [i.e. abulia, parkinsonism (Fisher, 1983; Cummings, 1992; Bharia and Marsden, 1994)]. Attention deficits and impairment in executive functioning have been consistent findings in bipolar patients (Martinez-Aran et al., 2000; Clark et al., 2002) and can be linked to the integrity of prefrontal cortical areas, which receive dopaminergic input from the basal ganglia (Posner and DiGirolamo, 1998). Specifically, tasks of effortful attention, such as measures that require selective attention, divided attention, and inhibition of a highly learned response in favor of a more novel response, are likely to engage prefrontal areas, whereas more automated attentional processes involve distinct neural networks.

Posner described visual attention as being composed of three components (each involving different neuroanatomical areas): orienting, detecting and vigilance (Posner and Petersen, 1990). The detecting network is consistent with Posner’s description of an anterior attention network and is commonly thought of as responsible for executive attention, or controlled processing. The orienting and vigilance networks combine to form the basis of Posner’s posterior attention network, which functions more automatically and is not otherwise involved in controlled processing (Posner and Petersen, 1990). The anterior attention system, described by Posner (1980), is involved in executive attention control (conscious attention) and overlaps anatomically with brain areas related to psychomotor functioning (Posner and DiGirolamo, 1998).

Therefore, we conducted a systematic investigation of psychomotor function in a group of bipolar depressed patients and examined visual attention disturbances using a specific, well-studied paradigm that distinguishes effortful from automatic attention as previously developed by Posner (1986). Severity of depression was also measured to determine its effect on attention and psychomotor disturbance. We hypothesized that: 1) bipolar depressed subjects would manifest psychomotor impairment as compared with healthy control subjects; 2) bipolar depressed subjects would demonstrate attentional impairment, specifically on measures of effortful attention, as compared with healthy control subjects; and 3) psychomotor retardation would be correlated with attentional deficits in bipolar subjects.

2. Methods

2.1. Subjects

Study participants comprised 24 patients who met DSM-IV criteria for bipolar I \( (n=16) \) or bipolar II \( (n=8) \) disorder and 24 age-matched healthy controls. All participants were between the ages of 18 and 55. The bipolar patients were in the depressed phase of illness and were derived from the Bipolar Disorders Research Center at the New York Presbyterian Hospital-Weill Medical College of Cornell University. Approval from the Institutional Review Board was granted from the New York Presbyterian Hospital–Weill Medical College of Cornell University as well as from the Queens College IRB and written informed consent was obtained prior to any study procedures being performed. Subjects were assessed using the Structured Clinical Interview of the DSM-IV (SCID-IV) (First et al., 1997) to establish research diagnoses of bipolar I or bipolar II disorder, with moderate to severe depression. All patients were on one of three mood stabilizer medications (lithium, valproate, or carbamazepine) at standard therapeutic doses, confirmed by blood levels. Subjects were not taking any antidepressant or antipsychotic medications at the time of testing.

Subjects were included if they scored at least 18 on the first 17 items of the 31-item Hamilton Depression Scale (HAM-D31) (Hamilton, 1960), indicating moderate to severe depression. Mania symptoms were measured by the Young Mania Rating Scale (YMRS) (Young et al., 1978) and psychosis was assessed using the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1989). Both the YMRS and the PANSS were used to screen out the presence of current psychosis and current
mania. Other exclusion criteria included the presence of (a) substance abuse or dependence in the 1-month or 6-month periods, respectively, preceding enrollment; (b) past year rapid cycling as per DSM-IV criteria; (c) concomitant active Axis I disorders; (d) current antipsychotic medications, due to their potential psychomotor effects (e) current antidepressant or stimulant use; and (f) the use of benzodiazepines within 6 h of testing.

Healthy control subjects were recruited from local advertisements and postings and underwent a SCID-IV interview to verify that there were no current or past psychiatric diagnoses. In addition, a HAM-D31, PANSS, and YMRS were completed for each control subject to rule out any current subsyndromal psychiatric symptoms, such as appetite or sleep disturbances. The cutoff for inclusion for healthy control subjects was set at HamD31<8.

2.2. Psychopathology assessment

Severity of depression was measured by the HAM-D31 (Hamilton, 1960). The HAM-D31 assesses both typical (i.e. loss of appetite, insomnia) and atypical (i.e. increased appetite, hypersomnia) symptoms of depression, while shorter versions of the same scale (HAM-D 17 item, and HAM-D 24 item) assess only typical symptoms.

2.3. Attentional tests

2.3.1. Automatic attention: Cued Target Detection

A computerized task (PRECUE) was administered that produces an orienting and alerting index of attention (Psych/Lab, 1999). This measure, a version of the “Posner” precuing paradigm known as the Cued Target Detection (CTD), is a target detection task in which patients were asked to detect the presence of an “x” inside of one of two boxes while keeping his/her eyes fixated on a central “+”. Just prior to the target appearing in one of the boxes, subjects were explicitly told to watch for a cue which would correspond with the side of the screen in which the target would appear 80% of the time. Subjects were then instructed to respond to the target as quickly as possible by pressing a key on the side of the keyboard corresponding with the side of the screen on which the target appeared.

Cues were of varying types, including valid (target appeared in the same location as the preceding cue), invalid (target appeared opposite the preceding cue), and neutral (cues appeared on both sides, giving no prior information regarding target location). The task included five blocks with 50 trials per block. The intertrial interval was set at 500 ms, with an interval between display onset and cue set at 1000 ms. The time between the onset of the cue and the onset of the target was variable, with various stimulus onset asynchronies (SOAs) of 100, 400, and 800 ms. The entire task lasted for approximately 8–10 min.

The three cue conditions (either valid, invalid, or neutral), the location of the stimuli, and the SOAs presented were determined randomly for each block. Reaction times of correct trials were analyzed and the maximum allowed reaction time was set at 3000 ms.

Variables derived included ‘Cost’, as measured by mean reaction time on neutral cue conditions minus the mean reaction time on invalid cue conditions and ‘Benefit’, measured by mean reaction time on neutral cues minus mean reaction time on valid cues. Additionally, differential performance across SOA and cue condition (Valid, Invalid, Neutral) was measured.

2.3.2. Effortful attention variables: Stroop and d2 Test of Attention

The Stroop Color–Word Test (SCWT) (Stroop, 1935) is a commonly used task measuring selective attention and inhibitory control (Spreen and Strauss, 1998). The Golden (Golden, 1976) version was used in the current study and consists of the following three conditions 1) Word series — 100 stimuli consisting of color names printed in black. Subjects were told to read the printed words as quickly as possible. 2) Color series — 100 stimuli of five colored Xs and subjects were told to name the color (red, blue, and green) of the Xs. 3) Color–word series — 100 stimuli consisting of color words printed in incongruent colored ink. For example, the word “green” was printed in red ink. Subjects were instructed to ignore the word that was printed and instead say the color of the ink that the word was printed in. For all conditions, subjects were instructed to read aloud as quickly as possible for a total of 45 s.

Variables of interest were word (W), color (C), color–word (CW), and interference scores. Word and color scores reflect the number of items read for the word only and color only conditions. Interference is designed to reflect “pure interference” by controlling for speed while providing an index of inhibitory control (Golden, 1976). Interference scores are calculated by the following formula: \((C \times W)/(C + W) = \text{Predicted CW score (CW')\}}\). Interference scores are the actual CW score minus the predicted CW'.

The d2 Test of Attention (Brickenkamp and Zillmer, 1998) is a paper–pencil cancellation test that is intended to measure selective attention and mental concentration. This task is a basic cancellation task where patients must cross out targets that include a “d” and two dashes, in
any distribution (dashes can be two above, two below, or one above and one below the d). The variable utilized as a measure of effortful attention for the current study was Concentration Performance (CPR), which is the number of correctly cancelled relevant items (hits) minus the errors of commission. Concentration Performance provides a reliable index of coordination of speed and the accuracy of performance, taking into account the total number of items processed.

2.4. Psychomotor assessment

2.4.1. Reaction Time testing

Motor speed was assessed using a Simple Reaction Time test (SRT), where each patient was administered five practice trials followed by fifteen test trials. Each trial consisted of the patient responding with his/her dominant hand to a red light presented in the center of the visual field. Choice Reaction Time testing (CRT) was obtained by the same method with the addition of green and blue lights that were presented as non-target stimuli, while the patient was instructed to respond to the red light only. The interstimulus intervals for both the SRT and the CRT were set at 500 ms.

2.4.2. Speed of finger oscillation

The Finger Tapping Test (FTT) (Reitan, 1969) was used to assess motor speed. Using a specially adapted tapper, subjects were instructed to tap as quickly as possible using the index finger, without moving the arm or hand. Each subject was administered ten trials (5 on each hand) of 10 s each, with mean number of taps per 10 s as the variable of interest.

2.4.3. Finger and hand dexterity

The Grooved Pegboard (GPB) (PAR Inc, 2002) was used to measure fine motor speed and dexterity on the dominant hand. Patients were asked to place key-like pegs into a board as quickly as possible, starting at the top left-hand corner of the board, using their dominant hand. Subjects were instructed to pick up one peg at a time and to complete the board in succession, without skipping over any of the holes. Total time to complete the board was used as the variable of interest.

2.4.4. Statistical analyses

A multivariate analysis of covariance (MANCOVA), with age and education as covariates, was used to detect mean group differences in performance on psychomotor and attention measures, including: d2 Concentration, Finger Tapping, and Grooved Pegboard. Simple Reaction Time and Choice Reaction Time measures were transformed to meet assumptions of homogeneity of variance, using log transformation, and then entered into the MANCOVA. Stroop Interference scores were analyzed separately so as to avoid double-correction for age and education, as these corrections are applied in the scoring of the tests; therefore, for Stroop Interference, we used a univariate ANOVA. Comparisons between groups on the automatic measures of Cost and Benefit from the CTD at varying SOAs included only 19 of the 24 bipolar patients, as this task was added after the first five patients had already completed the battery. CTD results were analyzed by two methods. First, we used standard multivariate analysis of covariance (MANCOVA) controlling for age and education. Reaction measures derived from the CTD at each SOA were entered into this analysis to provide for additional assessment of psychomotor speed. Second, we tested for Group×SOA interactions using repeated measures ANOVAs for costs and benefits with appropriate post-hoc tests.

The relationship between psychomotor functioning, depressive severity and attentional deficits was investigated using Spearman’s rho correlations and this was done in each group separately.

All statistical tests were two-tailed and utilized an alpha level of $P<0.05$. While multiple comparisons were made, these neurocognitive measures are not independent of one another; thus, a Bonferroni correction would be too conservative. For the purposes of controlling for multiple testing and non-independence of the variables, a multivariate analysis of variance was used as opposed to independent $t$-tests. With regard to multiple testing in conducting a large number of correlational analyses, we did not use a standard correction, rather we note that the strength of the relationship can be evidenced by the correlation coefficient in the context of $P$-values $<0.05$.

3. Results

Table 1 presents demographic data separately for the bipolar and control groups. No significant differences

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Healthy control $(N=24)$</th>
<th>Bipolar depressed $(N=24)$</th>
<th>Statistic ($P$-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (S.D.) age</td>
<td>33.96 (9.1)</td>
<td>39.96 (9.5)</td>
<td>$t_{-22} = -2.2 (0.03)^*$</td>
</tr>
<tr>
<td>Mean (S.D.) years of education</td>
<td>16.9 (2.1)</td>
<td>15.6 (2.4)</td>
<td>$t_{-22} = 1.2 (0.05)^*$</td>
</tr>
<tr>
<td>% female</td>
<td>58.3</td>
<td>37.5</td>
<td>Chi$^2 = 2.09 (0.15)$</td>
</tr>
<tr>
<td>% right-handed</td>
<td>87.5</td>
<td>100.0</td>
<td>Chi$^2 = 3.20 (0.07)$</td>
</tr>
<tr>
<td>Hamilton 31-item</td>
<td>2.54 (2.3)</td>
<td>33.7 (7.1)</td>
<td>$t_{-20.5} = -20.5 (P&lt;0.001)^*$</td>
</tr>
</tbody>
</table>
were observed between groups for sex or handedness; however there were significant differences between groups for age and education. These variables were thus utilized as covariates for all analyses, with the exception of the Stroop Interference score, as this measure controls for age and education in the initial calculation.

Results from the MANCOVA indicating d2 Concentration, Finger Tapping, Grooved Pegboard, SRT, and CRT indicate an overall significant effect of Subject Type ($F=4.04; df=3, 47; P=0.005$); Age ($F=4.32; df=3, 47; P=0.003$); and Education ($F=2.56; df=3, 47; P=0.042$). Tests of between-subject effects are depicted in Table 2, with patients with bipolar disorder demonstrating significantly slower performance on only a single measure of psychomotor function: Choice Reaction Time (CRT). On measures of gross motor speed patients seem to be somewhat less impaired with non-significant differences in performance on FTT, SRT, and GPB, suggesting a possible differential slowing on tasks that are associated with a cognitive component. In support of this hypothesis, when we analyze the SRT and CRT variables as two levels in a within-subjects factor (Condition), we find that the Group × Condition interaction term is also significant ($F=13.61; df=1, 46; P=0.001$).

Tests for group differences on attentional measures revealed a significant main effect for Subject Type ($F=4.02; df=3, 47; P=0.050$) and Age ($F=12.78; df=3, 47; P=0.001$) on d2 Concentration performance, with bipolar patients performing significantly worse than healthy controls on these attentional measures. Further, results from the univariate ANOVA indicate an overall significant effect of Subject Type ($F=4.48; df=1, 47; P=0.040$) on Stroop Interference. In contrast, there were no significant group differences for any of the key Cost and Benefit variables from the Cued Target Detection task (CTD), using two different methods of analysis.

First using a MANCOVA, we found that measures of Cost did not result in significant group differences at any of the SOAs. This suggests that bipolar patients do not incur greater costs than healthy controls, when their attention is directed to an incorrect location prior to target onset. Additionally, there were no significant differences found between groups on the Measure of Benefit. This suggests that BP subjects gained comparable advantage of detection, when correct cues were given prior to target presentation, across all SOAs. Results from the MANCOVA are presented in Table 3. Second, we analyzed the CTD variables with a repeated measures ANCOVA, as in Sweeney et al. (2000), considering the various SOAs as levels of difficulty and utilized the Huynh–Feldt correction method. There were no significant results for any main effects for the measure of Cost, with non-significant effects of SOA ($F=2.16; df=2, 78; P=0.122$) and of SOA × Subject Type interaction ($F=0.163; df=2, 78; P=0.850$). Consistent with this, there were no significant results for the measure of Cost, with non-significant effects of SOA ($F=0.509; df=2, 78; P=0.552$) and of SOA × Subject Type interaction ($F=0.018; df=2, 78; P=0.958$).

Spearman’s correlational analyses (Table 4) revealed that in the healthy controls, there was not a strong relationship between performance on the attentional measures and normal psychomotor function, with no significant correlations noted (data not shown). This finding was as expected, with healthy controls’ performance falling within the normal range on attentional measures in the absence of psychomotor deficits. In addition, as expected, HAM-D31 ratings were not correlated with attention or

### Table 2
Mean differences on psychomotor variables in bipolar (BP) patients versus healthy controls (HC).

<table>
<thead>
<tr>
<th>Measure</th>
<th>HC (N=24) Mean (S.D.)</th>
<th>BP (N=24) Mean (S.D.)</th>
<th>F-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FTT—total</td>
<td>54.4 (8.5)</td>
<td>52.5 (10.4)</td>
<td>0.08</td>
<td>0.78</td>
</tr>
<tr>
<td>GPB—time to complete</td>
<td>63.0 (5.7)</td>
<td>71.8 (16.1)</td>
<td>1.64</td>
<td>0.21</td>
</tr>
<tr>
<td>SRT—ms (log transformed)</td>
<td>431.4 (111.5)</td>
<td>525.0 (144.2)</td>
<td>1.25</td>
<td>0.27</td>
</tr>
<tr>
<td>CRT—ms (log transformed)</td>
<td>524.7 (110.2)</td>
<td>704.5 (189.4)</td>
<td>8.57</td>
<td>0.004*</td>
</tr>
</tbody>
</table>

*P ≤ 0.05.

### Table 3
Mean differences on attention variables (bipolar versus healthy control).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Healthy controls (N=24) Mean (S.D.)</th>
<th>Bipolar patients (N=19) Mean (S.D.)</th>
<th>F-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EFFORTFUL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop interference score</td>
<td>5.25 (8.6)</td>
<td>0.63 (6.5)</td>
<td>4.43</td>
<td>0.04*</td>
</tr>
<tr>
<td>d2-Concentration total</td>
<td>214.17 (39.1)</td>
<td>171.46 (49.5)</td>
<td>4.02</td>
<td>0.05*</td>
</tr>
<tr>
<td><strong>AUTOMATIC-CTD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cost 100 ms</td>
<td>−38.2 (51.6)</td>
<td>−75.4 (191.7)</td>
<td>0.47</td>
<td>0.50</td>
</tr>
<tr>
<td>Cost 400 ms</td>
<td>−42.3 (46.5)</td>
<td>−88.2 (152.3)</td>
<td>0.57</td>
<td>0.46</td>
</tr>
<tr>
<td>Cost 800 ms</td>
<td>2.4 (42.3)</td>
<td>−23.8 (75.8)</td>
<td>1.27</td>
<td>0.27</td>
</tr>
<tr>
<td>Benefit 100 ms</td>
<td>32.6 (29.7)</td>
<td>44.1 (36.4)</td>
<td>1.50</td>
<td>0.23</td>
</tr>
<tr>
<td>Benefit 400 ms</td>
<td>23.4 (26.7)</td>
<td>47.7 (59.8)</td>
<td>1.72</td>
<td>0.20</td>
</tr>
<tr>
<td>Benefit 800 ms</td>
<td>−9.0 (41.9)</td>
<td>8.9 (40.3)</td>
<td>3.38</td>
<td>0.07</td>
</tr>
</tbody>
</table>

*P ≤ 0.05.
psychomotor measures in the healthy controls (data not shown).

In contrast, in patients with bipolar disorder, impaired performance on the d2 Test of Attention was significantly correlated with several measures of psychomotor slowing, including Simple, and Choice Reaction Time, as well as grooved pegboard performance. The Stroop Interference measure did not correlate with the psychomotor measures, which is likely a reflection of the inherent control for motor speed on this variable.

Furthermore, in the bipolar sample, reaction time measures (both simple and choice) were significantly correlated with the HAM-D31, suggesting a significant impact of depressive severity on psychomotor performance. Interestingly, HAM-D31 was not strongly correlated with performance on the attentional measures in the bipolar group, with the exception of one CTD variable (Cost at SOA of 800 ms).

4. Discussion

The present study compared outpatients with bipolar depression with healthy controls in an attempt to identify and further classify psychomotor and attention deficits seen in bipolar depression. Bipolar patients’ performance was significantly slower than that of healthy controls on a single psychomotor measure, involving a cognitive speed component. On tasks that measure pure motor speed, the performance of bipolar patients was comparable to that of healthy controls. These data are consistent with those previously reported in a sample of bipolar depressed patients who performed within normal limits on tests of pure motor speed (Sweeney et al., 2000).

Attentional measures designed to assess both automatic and effortful processing were used to facilitate the characterization of attention abnormalities. Results from the Stroop Task and the d2 Test of Attention suggest that depressed patients with bipolar disorder demonstrate effortful attention deficits as compared with healthy controls. In contrast, automatic attention, as measured by the Cued Target Detection test, was unimpaired in the bipolar group. Patients with bipolar disorder, like healthy controls, responded more quickly when presented with a valid cue than with a neutral or invalid cue. These data indicate that bipolar depressed subjects’ basic ability to orient attention to a target is not substantially compromised.

This “Benefit” of valid cueing indicates an ability to maintain attention automatically drawn to a location, regardless of the interval following the cue and preceding the target. Because the longest SOA utilized in the current study was 800 ms, we are unable to state with confidence that this is indicative of intact vigilance, or sustained attention; however, we also cannot infer unimpaired phasic alertness or cortical arousal in our sample of patients with bipolar depression. The “Cost” or the disadvantage of orienting attention to an incorrect location prior to target onset was also comparable in our sample of patients with bipolar disorder as compared with non-psychiatric controls. This is consistent with our hypothesis and previous studies (Hartlage et al., 1993) demonstrating that automatic attention processing is intact in patients with depression.

Our data indicate a significant and robust relationship between measures of psychomotor retardation and specific aspects of attentional performance in the bipolar sample. This relationship was only noted on the effortful attention measure of d2 Concentration; as increased reaction time for simple and choice paradigms and slower performance on the Grooved Pegboard were associated with a lower Concentration Index on the d2 Test of Attention. This relationship was not found for the Stroop Interference scores; however, this was expected, as the calculation of the Interference score includes a control for speed of response, reducing its reliance on intact psychomotor function.

Although the bipolar patients’ performance on most of the psychomotor measures was not significantly different from that of healthy controls, they did differ significantly on a choice reaction time measure, which
required some cognitive speed (a decision to be made), as opposed to the other measures, which are more strictly motor speed tasks. The two psychomotor measures that were significantly correlated with depressive severity as measured by the HAM-D31 included the choice reaction time measure, as well as a simple reaction time measure. These results may reflect a slowing of speeded processing related to depression that may not reach a full “deficit” threshold (i.e. impaired versus healthy control subjects) on measures that require only simple motor speed but are related to the severity of depression nonetheless. The lack of significant correlations between attentional measures and depressive severity indicates the possibility that psychomotor performance may serve as a moderating variable between these domains. It is, of course, also possible that our study was not adequately powered to detect more direct relationships among these measures. Further, the differential sensitivity of these tests undoubtedly impacts upon the potential to identify relationships between these domains.

These data are consistent with work by Lemelin and Baruch (1998), in which clinician-rated psychomotor assessments predicted attentional impairment in a group of depressed patients. We found, using objective measures of psychomotor function, that performance on a range of motor tasks was strongly correlated to deficits in specific aspects of effortful attention. These data expand upon the study by Lemelin and Baruch (1998), in which a clinician-rated scale was used to measure both the clinician’s impression and the patient’s subjective experience of cognitive impairment and motor slowing, but did not objectively characterize the sample using standardized motor and neurocognitive tasks.

The specificity of these findings with regard to diagnosis (bipolar versus unipolar depression) is not yet known. Results comparing neurocognitive performance between bipolar and unipolar depressed samples have varied, with some studies reporting more significant deficits in memory and executive functions in bipolar depressed patients as compared to unipolar depressed patients (Borkowska and Rybakowski, 2001); while other studies have reported similar cognitive profiles in unipolar and bipolar depressed patients, consisting of deficits in episodic memory in both groups relative to healthy controls (Sweeney et al., 2000).

In addition, the considerable effects of mood state on neurocognitive performance may confound data indicating neurocognitive impairment in acutely ill patients; however, impaired performance on attentional measures has been described in patients with bipolar disorder across mood states, suggestive of a trait-like impairment. Basso and colleagues (2002) have demonstrated similar cognitive impairment in three subgroups of bipolar disorder (depressed vs. manic vs. mixed), with results indicating significant deficits in each group versus healthy controls, but with no differences noted between patient groups. Furthermore, specific attention deficits have been demonstrated in euthymic bipolar patients (Ferrier et al., 1999; Liu et al., 2002; Clark et al., 2002; Thompson et al., 2005), when patients are free of acute symptomatology. Finally, a recent study by Rubinsztein et al. (2006) evaluated 24 patients with bipolar depression on a range of neurocognitive measures and found widespread impairment including attentional deficits, with no evidence of a commonly described state-dependent affective attentional bias, further supporting the notion of a stable deficit in basic attentional processes. Of particular importance are recent data from a sample of 78 patients with bipolar disorder that indicate that the inter-episode persistence of attentional impairment is highly predictive of functional outcome in patients with bipolar disorder (Jaeger et al., 2007). The fact that the cognition-functional status relationship was present even after a one-year interval in this study underscores the robustness of the association, as well as increases the likelihood that it reflects trait related cognitive impairment that is independent of fluctuating symptoms.

There are a number of limitations to this study. First, we were not able to fully address a number of clinical factors which may bear adversely on the cognitive profile of bipolar patients, including the number of episodes, duration of illness, possible medication effects, and history of psychosis. Denicoff et al. (1999) found that neuropsychological deficits in bipolar patients were associated with a longer duration and more severe prior course of illness, as reflected by number of episodes of mania and depression and number of hospitalizations. Specifically, increased number of episodes was significantly correlated with impairment in memory and abstraction tasks, while increased number of hospitalizations and longer duration of illness was associated with select deficits in attention and concentration measures. Second, the current sample was medicated at the time of testing and we were unable to stratify by medication due to a limited sample size. We did limit the heterogeneity of treatment however, by allowing only one of the three most common treatments (lithium, valproate, or carbamazepine), with no concomitant antipsychotics or antidepressants allowed.

With regard to diagnostic subtype, the majority of patients carried a diagnosis of bipolar I disorder (65%) which did not allow for splitting patients by bipolar type (BPI vs. BPII). This may have different implications on
clinical and cognitive functioning. Previous studies in a sample of depressed bipolar patients with a history of suicide attempts have reported impaired performance on attention and reaction time tasks in bipolar II patients relative to bipolar I patients (Harkavy-Friedman et al., 2006). However, in a study of euthymic bipolar patients, bipolar I patients exhibited more impairment on tasks of attention and memory as compared to bipolar II patients (Torrent et al., 2006), suggesting that performance differences may be sensitive to clinical state. Finally, we are limited by the psychometric properties of the measures chosen. Specifically, we cannot rule out the possibility that the differences noted on the effortful attention measures (but not the automatic measures) simply reflect differential sensitivity of the tasks themselves.

In summary, these data provide preliminary evidence of dysfunction in the effortful (anterior) attention network in depressed subjects with BP disorder, with a relative sparing of automatic (posterior) attention. Attentional performance on specific measures of effortful attention was strongly correlated with psychomotor function. Depressive severity was correlated with psychomotor performance but was not significantly related to attentional performance in our sample. Results from the current study add to existing evidence of a noteworthy deficit in psychomotor function that may be more commonly found on tasks requiring some higher-order processing, as opposed to pure motor speed measures. Further, these data are consistent with a shared neurobiological basis for psychomotor and attentional impairment in bipolar depression.

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