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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).

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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; Bhatia 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 ($HAM\text{-}D_{31}$) (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