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Evidence for cognitive subgroups in bipolar disorder and the influence of subclinical depression and sleep disturbances

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Abstract

Recent research in bipolar disorder (BD) points to the relevance and persistence of cognitive deficits even in euthymia. Up to now, the mechanisms behind why some bipolar patients (BP) do not reach their former level of cognitive performance and psychosocial functioning while others remit completely, are not understood. In this study we aimed to identify a “cognitive deficit” vs. “non-deficit” subgroup within BD by using an extensive neuropsychological test battery. The test performance of 70 euthymic outpatients (BD-I and II, recruited as a sample of convenience from our bipolar disorder programme) was compared to 70 matched, healthy controls (HC). Furthermore, we investigated the association between demographic/clinical variables and the cognitive performance of BP. As expected, our sample of euthymic BP performed significantly worse than HC in psychomotor speed, divided attention, working memory, verbal memory, word fluency and problem solving. However, 41.4% of the patients did not have any neurocognitive deficits at all, and whether or not a patient belonged to the non-deficit group was not influenced by disease severity. Instead, our results demonstrate that patients suffering from persistent sleep disturbances and sub-threshold depressive symptomatology show more severe cognitive dysfunctions. In addition, antipsychotic treatment and comorbid anxiety disorder were associated with cognitive deficits. In sum, these results suggest that a major part of cognitive impairment is due to current symptomatology, especially sleep disorder and sub-syndromal depression. Rigorous treatment of these symptoms thus might well improve cognitive deficits and, as a consequence, overall functioning in BD.

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1. Introduction

Cognitive deficits are common during acute episodes of depression and mania in bipolar disorder (BD). However, a great number of bipolar patients (BP) report persistent cognitive impairments even after remission of an acute episode. Recent studies verified cognitive impairments during euthymia with deficits up to one standard deviation below average in psychomotor speed, attention, working memory, long term memory and executive functioning (Ancin et al., 2013; Burdick et al., 2010; Hellvin et al., 2012; Martinez-Aran et al., 2004; Ryan et al., 2012; Thompson et al., 2005). With the exception of crystallised abilities (e.g. vocabulary, word reading) and premorbid IQ, these deficits seem to be global and comparable to schizophrenia, albeit less severe (Vohringer et al., 2013). However, current studies suggest that cognitive deficits in BD are not as severe as previously assumed. For instance, Bourne et al. (2013) conducted an “individual patient data meta-analysis” by including unpublished studies and considering more confounding factors. The authors confirmed cognitive impairments in euthymic BP, but found considerably lower effect sizes ($d=.26-.63$) than previous meta-analyses ($d=.5-1$). In line with this result, other studies reported that only a part of their bipolar sample had significant cognitive impairments (Altshuler et al., 2004; Aminoff et al., 2013; Iverson et al., 2011; Martino et al., 2008). Thus, there seem to be subgroups within the cluster of BD; however, the underlying reasons are unclear: Some patients do not reach their former level of cognitive performance and become more disabled with progressing illness, while others remit completely after each episode and maintain a high occupational and social functioning (Gilbert and Marwaha, 2013).

The disparity of cognitive impairments depend on the applied criteria of the normal range (Reichenberg et al., 2009). However, due to the heterogeneity in the cognitive profile of BP, group statistics are not sufficient. Mean values convey the risk to suggest a global deficit for all patients and can obscure inter-individual differences. Nevertheless, the accurate description of neurocognitive impairment is essential for the understanding of aetiopathogenesis. An accurate description of neuropsychology in BD allows conclusions about disease specific cortical or subcortical pathologies. More homogeneous subgroups are needed in the search for genetic underpinnings and mechanisms underlying treatment response. In the clinical setting, personalised interventions are needed to improve not only affective symptoms but also cognitive dysfunctioning. This is crucial because cognitive impairments are significant predictor of psychosocial and occupational outcomes in BD (Gilbert and Marwaha, 2013). Given that low psychosocial functioning in turn leads to a high risk of relapse, reduced life quality and rising economic costs (Morselli et al., 2004), the identification of moderator variables associated with cognition in BD has relevant implications for further research and the improvement of treatment strategies.

To address this issue, the relationship between cognition and demographic, clinical and treatment variables has been investigated in recent years. Numerous studies found an association between symptom severity and neurocognitive impairments. For instance, significant associations between

cognitive deficits and greater number of episodes have been found (Martinez-Aran et al., 2004; Thompson et al., 2005). Furthermore, higher incidences of manic episodes (Aminoff et al., 2013; Bourne et al., 2013; Lopez-Jaramillo et al., 2010), longer duration of disease (Ryan et al., 2012; Torrent et al., 2012), and more hospitalisations were described to be associated with cognitive impairment (Ancin et al., 2013; Ryan et al., 2012). Therefore, some authors suggested that severe mood symptoms could act like stress-induced neurotoxins (Lopez-Jaramillo et al., 2010). In contrast, other studies found that first-episode BP patients have the same neurocognitive performance as patients with multiple episodes in the past (Bombin et al., 2013; Hellvin et al., 2012). Torrent et al. (2012) described in a recently published longitudinal study that cognitive impairments (except for a worsening of executive functions) remained stable over nine years, irrespective of severe relapses in the meantime. Hence, several authors concluded that cognitive impairments are present in the early beginning of BD and that there is an evidence of a neurocognitive decline with illness progression. These results led to considerations of cognitive deficits as trait effect and the idea of them being a bipolar endophenotype (Bora et al., 2009).

Another often reported finding is that patients diagnosed with BD Type-I have more cognitive deficits than patients with BD Type-II (Aminoff et al., 2013; Ancin et al., 2013; Palsson et al., 2013; Torrent et al., 2006). However, Dittmann et al. (2008) and Chaves et al. (2011) found no neuropsychological differences between these two diagnosis types. The fact that patients with BD Type-I have full-blown manic episodes and more psychotic symptoms could contribute to these inconsistent findings (Palsson et al., 2013), as some authors found indeed a significant association between psychotic symptoms and cognition (Aminoff et al., 2013; Bora et al., 2011), while others did not (Brissos et al., 2011). Furthermore, individuals diagnosed with BD Type-I are more often treated with antipsychotics compared to BD-II, which could contribute to cognitive impairments in this patient group (Arts et al., 2011; Jamrozinski et al., 2009; Palsson et al., 2013; Torrent et al., 2011). If other drugs or polypharmacy have negative effects on cognition has not been fully understood yet. Mood stabilisers, especially lithium, interestingly only exerts a marginal (Dias et al., 2012) or no influence on cognition (Altshuler et al., 2004; Arts et al., 2011; Lopez-Jaramillo et al., 2010). Moreover, a longitudinal study pointed out neuroprotective effects of lithium (Diniz et al., 2013). Antidepressants seem to have no remarkable adverse cognitive effects, aside from the anticholinergic effects of tricyclics (Amado-Boccaro et al., 1995). In summary, only antipsychotics seem to have a negative effect on cognitive performance in BD. However, cognitive dysfunctions cannot fully be explained by drug side effects because even in medication-free euthymic BP, neurocognition is impaired (Bourne et al., 2013; Goswami et al., 2009).

In summary, cognitive deficits in remitted BP have been repeatedly demonstrated, but findings are inconsistent regarding the severity of impairments and associated disease characteristics. In the present study, we aim to scrutinise neuropsychological functioning in BD. Our hypotheses were that BP as a group show cognitive deficits compared to healthy controls (HC). Furthermore, we

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