



## Neurocognition and psychosocial functioning in adolescents with bipolar disorder



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### ABSTRACT

**Background:** Adults with bipolar disorder demonstrate significantly poorer psychosocial functioning and neurocognition compared to controls. In adult bipolar disorder neurocognition predicts a substantial portion of variance in functioning. Adolescents with bipolar disorder have reduced psychosocial functioning, but less is known about neurocognitive impairments, and no studies have examined the relationship between neurocognition and functioning in an adolescent sample.

**Methods:** 38 adolescents with bipolar disorder and 49 healthy controls under 20 years of age completed assessments of psychosocial functioning, neurocognitive ability, and psychiatric symptoms.

**Results:** Adolescents with bipolar disorder had significantly poorer psychosocial functioning in domains of daily activities, social functioning, and satisfaction with functioning,  $p < .006$ , compared to healthy controls. They also had poorer general neurocognitive functioning than controls,  $p = .004$ , with the greatest impairment on a test of sustained attention. Neurocognition was not a significant predictor of psychosocial functioning in this sample, but depressive symptoms significantly predicted functioning in all domains,  $p < .033$ .

**Limitations:** Limited sample size did not allow for complex statistical analyses. Differences in demographic characteristics of the clinical and control groups may limit generalization of these results.

**Conclusions:** This adolescent sample with bipolar disorder experiences significantly poorer neurocognitive and psychosocial functioning compared to controls; however, psychosocial functioning appears to be more strongly related to mood symptoms than to neurocognition. Future work is needed to delineate the time course of neurocognitive functioning and its relation to psychosocial functioning across the course of illness. Adolescence may provide an ideal time for cognitive enhancement and intensive psychosocial intervention.

### 1. Introduction

Bipolar disorder is associated with various measures of functional disability, including increased health care costs (Simon, 2003), higher unemployment rates (Coryell et al., 1993; Tse and Walsh, 2001), higher dependence on public assistance (Judd and Akiskal, 2003), lower annual income (Goetzl et al., 2003), decreased work productivity (Goetzl et al., 2003), poorer social functioning (Morriss et al., 2007), poorer overall functioning (Goldberg et al., 1995; Keck et al., 1998; Judd et al., 2005), and lower quality of life (Vojta et al., 2001). Given the degree of functional disability associated with the disorder, definitions of recovery now include improvement to normative levels of psychosocial functioning (Harvey, 2005; Grunze et al., 2013). Unfortunately, reviews estimate that up to 60% of individuals with bipolar disorder will not achieve full functional recovery (MacQueen

et al., 2001). Consequently, greater efforts are being made to understand the nature of functional disability in bipolar disorder, and these domains of functioning have become important treatment targets.

Psychosocial functioning appears to be significantly impaired in individuals who are in acute depressive or manic / hypomanic episodes (Rosa et al., 2010; Malhi et al., 2007); depressive symptoms induce the most enduring functional deficits (Simonsen et al., 2010). However, functional impairments persist even after significant mood symptoms have remitted (Andreou and Bozikas, 2013). In samples of euthymic adults, subclinical depressive symptoms continue to be associated with poorer functioning (Baş et al., 2015; Bonnin et al., 2010, 2012), however other factors must also be considered. In fact, although depressive symptoms relate to a person's psychosocial functioning, a better predictor of community function is neurocognitive ability (Wingo et al., 2009; Andreou and Bozikas, 2013). Generally, adults

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with bipolar disorder demonstrate significantly poorer cognitive functioning in most domains compared to healthy controls (Robinson et al., 2006), and it appears that neurocognition is the best predictor of community functioning (Bowie et al., 2010; Depp et al., 2012; Tse et al., 2014). When both mood symptoms and neurocognition are considered, mood only has a modest direct relationship with functioning (Bowie et al., 2010). Global neurocognition is often reported as predicting community functioning (Bowie et al., 2010; Depp et al., 2012; Andreou and Bozikas, 2013), however, relationships with specific cognitive domains have also been reported: verbal memory and executive functioning (Wingo et al., 2009; Tse et al., 2014), attention (Andreou and Bozikas, 2013; Wingo et al., 2009), and processing speed (Wingo et al., 2009). Similar to functional impairments, neurocognitive difficulties persist into periods of euthymia (Goswami et al., 2006), and appear to have only modest relationships with mood state (Kurtz and Gerraty, 2009), perhaps explaining why reduction in mood symptoms with psychopharmacological treatments often has little effect on everyday functioning.

Despite the robust literature in adults demonstrating the real-life importance of this topic, there is a paucity of data among youth with bipolar disorder. Geller et al. (2000) found that over half of the adolescents with bipolar disorder in their sample were functioning poorly, and that they were more impaired than adolescents with attention-deficit / hyperactivity disorder (ADHD) and healthy controls in social, family, and academic functioning. Global functional impairments are consistently reported across samples of adolescents with bipolar disorder (Lewinsohn et al., 1995; Biederman et al., 2005; Wilens et al., 2003) and tend to be paired with lower subjective quality of life (Rademacher et al., 2007). Specific impairments in social and interpersonal functioning are also reported (Wilens et al., 2003; Goldstein et al., 2006) and may be a primary reason why adolescents enter treatment initially. Similar to adult bipolar disorder (Fagioli et al., 2005; Morriss, 2002), these impairments in psychosocial functioning persist despite remission of significant mood symptoms, however, they tend to worsen during periods of symptom exacerbation (Goldstein et al., 2009).

Considering the serious functional consequences of adolescent-onset bipolar disorder and the relationship found between neurocognition and functioning in adults, research has begun to examine neurocognition in adolescents with the illness. Initial studies in this area suggest that the general profile of cognitive abilities in adolescents appears to be qualitatively different than that observed in adults with bipolar disorder where a generalized neurocognitive deficit appears to be present. In adolescents, neurocognitive difficulties appear to be more specific. Compared to healthy controls, studies have suggested that adolescents with bipolar disorder have significantly poorer neurocognitive abilities compared to controls in domains of attentional set-shifting and visuospatial memory (Dickstein et al., 2004); sustained attention, working memory, and processing speed (Doyle et al., 2005); and verbal declarative memory (Glahn et al., 2005); however, other studies have suggested that adolescents with bipolar disorder may have intact cognitive functions (DelBello et al., 2004). Meta-analyses have attempted to determine the nature of neurocognitive functioning in adolescent bipolar disorder, with similarly inconsistent results. Most studies find reduced verbal memory abilities (Joseph et al., 2008; Horn et al., 2011; Frías et al., 2014). Working memory and visuo-spatial memory also appear to be significantly poorer compared to healthy controls, but to a lesser extent than verbal memory (Joseph et al., 2008; Horn et al., 2011; Frías et al., 2014). It has also been suggested that adolescents with bipolar disorder have poorer executive functioning (Joseph et al., 2008), processing speed (Frías et al., 2014), and social cognition (Frías et al., 2014); however, these findings are not consistent across meta-analyses.

This report is the first, to our knowledge, to examine the relationships between neurocognition and psychosocial functioning in adolescents with bipolar disorder, and is part of a broader study examining

oxidative stress and vascular function as biomarkers of neurocognition in adolescent bipolar disorder, in which we have reported relationships between executive functioning and several traditional cardiovascular risk factors (Naiberg, 2014). The current report has three main aims: 1) to examine psychosocial functioning in adolescents with bipolar disorder compared to healthy controls; 2) to examine neurocognitive ability in adolescents with bipolar disorder relative to healthy controls; 3) to determine how neurocognition is related to psychosocial functioning in adolescent bipolar disorder.

## 2. Method

### 2.1. Participants

Thirty-eight adolescents with bipolar disorder (9 bipolar I, 17 bipolar II, 12 bipolar NOS) were recruited from the Youth Psychiatry Division of Sunnybrook Health Sciences Centre, and 49 psychiatrically healthy controls were recruited from the surrounding community. Bipolar disorder diagnoses were confirmed by experienced clinicians using the Schedule for Affective Disorders and Schizophrenia for School Age Children, Present and Life Version (K-SADS-PL; Kaufman et al., 1997). Healthy control participants were excluded if they had a history of mood or psychotic disorders, alcohol or drug dependence in the past 3 months, history of an anxiety disorder in the past 3 months, or first or second degree relatives with a bipolar or psychotic disorder. Demographic characteristics of the sample are presented in Table 1. There were equal numbers of males and females in the two groups; however, despite attempts to match based on age, the bipolar diagnosis group was significantly older on average than the healthy control group.

**Table 1**  
Demographic characteristics of adolescents with bipolar disorder and healthy controls. Bolded values indicate a significant difference between groups.

	Bipolar Disorder (n=38)	Healthy Controls (n=49)	Test Statistic	p value
Age (Mean <i>SD</i> )	17.42 (1.82)	15.96 (1.75)	<b><math>t=3.81</math></b>	<b><math>p &lt; .001</math></b>
Gender (n) (females:males)	23: 15	23: 26	$\chi^2=1.59$	$p=.208$
WASI IQ (Mean <i>SD</i> )	104.21 (13.19)	110.04 (12.97)	<b><math>t=2.06</math></b>	<b><math>p=.042</math></b>
K-SADS Mania Rating Scale – Current (Mean <i>SD</i> )	11.39 (12.28)	.41 (1.19)	<b><math>t=6.24</math></b>	<b><math>p &lt; .001</math></b>
K-SADS Depression Rating Scale – Current (Mean <i>SD</i> )	15.41 (10.33)	.46 (1.22)	<b><math>t=9.96</math></b>	<b><math>p &lt; .001</math></b>
K-SADS Mania Rating Scale – Past (Mean <i>SD</i> )	28.45 (11.75)	2.00 (10.07)	<b><math>t=11.30</math></b>	<b><math>p &lt; .001</math></b>
K-SADS Depression Rating Scale – Past (Mean <i>SD</i> )	30.11 (13.54)	2.73 (13.03)	<b><math>t=9.44</math></b>	<b><math>p &lt; .001</math></b>
Duration of Illness (years) (Mean <i>SD</i> )	6.55 (4.45)	–	–	–
Lifetime Anti-Depressant Medication(n)	8	1	<b><math>\chi^2=8.34</math></b>	<b><math>p=.004</math></b>
Lifetime Lithium(n)	9	0	<b><math>\chi^2=12.94</math></b>	<b><math>p &lt; .001</math></b>
Lifetime Antipsychotic Medication (n)	29	0	<b><math>\chi^2=56.09</math></b>	<b><math>p &lt; .001</math></b>
First Degree Relative with Mood Disorder (n)	21	3	<b><math>\chi^2=27.92</math></b>	<b><math>p &lt; .001</math></b>

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